Program for Friday, November 5, 1999

Third Pacific Vascular Symposium On Venous Disease November 2-6, 1999

Mauna Lani Bay Hotel & Bungalows • Kohala Coast, Big Island of Hawaii, USA

FRIDAY, NOVEMBER 5, 1999 (MAUNA LANI BALLROOM)

7:00 a.m. CONTINENTAL BREAKFAST/EXHIBITS OPEN

VENOUS ULCER
PART THREE - MANAGEMENT OF THE DECOMPENSATED LEG

7:30 a.m. Moderator: Thomas F. O'Donnell, Jr., MD
Panel: Michael C. Dalsing, MD
Ralph G. DePalma, MD, FACS
Peter Gloviczki, MD
Robert L. Kistner, MD
Michel Perrin, MD
Seshadri Raju, MD

7:45 a.m. VENOUS ULCER AND CEAP
Nicos Labropoulos, PhD, DIC, RVT

The management will be demonstrated by presentation of cases that will illustrate increasing severity of chronic venous disease. Each case will be handled by a group of specialists that will propose diagnostic measures and treatment. Their suggestions will be discussed by the panel and opened up to the audience for wider participation.

8:00 a.m. CASE OF SUPERFICIAL INCOMPETENCE

A 65-year old lady from the Big Island with 5 children developed large varicose veins during her pregnancies. She has had recurrent large ulcerations on her right leg for 30 years and within the last 6 years has developed a painful, circumferential ulceration on the right leg.

Specialists: Gianni Belcaro, MD, PhD (Save the Saphenous Vein)
G. Mark Malouf, MBBS, FRACS, FRC (High Ligation and Stripping)
J. Leonel Villavicencio, MD, FACS (Sclerotherapy)

9:00 a.m. CASE OF PERFORATOR INCOMPETENCE

65-year old woman with large recurrent venous ulcerations on left lower leg. Past history is significant for left GSV ligation and stripping, 1979 and left popliteal and superficial femoral vein DVT, 1989. Laboratory evaluation now shows the following: no significant deep venous obstruction and mild reflux (VFI 3.2 ml/sec) by APG; Duplex scan shows mild recanalization changes of superficial femoral and popliteal veins, no deep reflux and 3 large incompetent perforated veins in the medial aspect of the lower leg.

Specialists: Ralph G. DePalma, MD, FACS (SEPS)
Ermenegildo A. Enrici, MD (Open Perforator Ligation)
Jean-Jerome Guex, MD (Ultra-Sound Guided Sclerotherapy)

10:00 a.m. BREAK/EXHIBITS OPEN

10:30 a.m. CASE OF PRIMARY DEEP VENOUS REFLUX

A 36-year old gentleman from Western Samoa has had recurrent venous ulcerations of both legs that first began when he was 15 years old. He has not been able to work for the past 4 years because of painful, large ulcerations of the right leg.

Specialists: Robert L. Kistner, MD (External Valvuloplasty)
Thomas F. O'Donnell, Jr., MD (Video-Assisted Valvuloplasty)
Michel Perrin, MD (Internal Valvuloplasty)

11:30 a.m. CASE OF SECONDARY DEEP VENOUS DISEASE

47-year old male with disability of the left lower extremity due to post-thrombotic disease. Symptoms and findings of venous claudication, swelling, skin pigmentation, thickening and ulceration of 4 years duration.

Specialists: Seshadri Raju, MD (Something Always Can Be Done)
Michael C. Dalsing, MD (Valve Transplantation)
Peter N. Neglen, MD, PhD (Angioplasty and Stenting)

12:30 p.m. LUNCH (CANOE HOUSE)

1:30 p.m. REVAS
Michel Perrin, MD

2:00 p.m. OUTCOMES EVALUATION
Robert B. Rutherford, MD, FACS, FRCS

2:30 p.m. BREAK/EXHIBITS OPEN

VENOUS ULCER
PART FOUR - QUO VADIS?

3:00 p.m. RESULT OF QUESTIONNAIRE REGARDING DEEP VENOUS RECONSTRUCTION
Tomohiro Ogawa, MD, PhD

3:10 p.m. VALVULOPLASTY AND PRIMARY VENOUS INSUFFICIENCY
Fedor Lurie, MD, PhD

Moderator:	Robert L. Kistner, MD	Thomas F. O'Donnell, Jr., MD
Panel:	Michael C. Dalsing, MD	Michel Perrin, MD
	Ralph G. DePalma, MD, FACS	Seshadri Raju, MD
	Peter Gloviczki, MD	Robert B. Rutherford, MD, FACS, FRCS
	Kenneth A. Myers, MS, FACS,FRCS	D. Eugene Strandness, Jr., MD, DMed(Hon)

5:00 p.m. SCIENTIFIC SESSION ENDS

Faculty

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CHRONIC VENOUS ULCERS

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A.K. Tassiopoulos, MD

The prevalence of lower extremity ulceration secondary to chronic venous disease (CVD) in European and Western populations is estimated to be 0.5% to 1%.[1,2] Approximately 12% to 14% of patients with CVD in recent series[3,4] have venous ulcers (clinical classes C_5 and C_6). Despite the high prevalence and the significant morbidity of this problem, it has always been relatively neglected. Even currently, many patients are offered conservative management only. Duplex ultrasound examination of the lower extremity veins was introduced in recent years and significantly improved our understanding of the etiology of CVD. Moreover, this non-invasive test has allowed precise identification of the malfunctioning vein segments and has contributed to a more rational therapeutic approach that targets the affected venous segment.

The main mechanisms responsible for venous ulcers are reflux, venous outflow obstruction, or a combination of the two. Reflux is the most common cause of CVD and is seen in the vast majority of limbs with ulcers. Combined reflux and obstruction is more often seen in limbs belonging to classes C_4 to C_6.[3] A recent prospective study demonstrated that a combination of reflux and obstruction had worst prognosis for developing limb ulceration compared to reflux or obstruction alone (odds ratio 3.5, 95% CI 1.4-8.6).[5]

In contrast to what was previously thought, a documented episode of DVT is only seen in 33%-50%[6-10] of patients with ulceration and this prevalence is higher than in any other CVD class.[11] This prevalence is probably underestimated because many thrombi remain undetected and may resolve without leaving any evidence of luminal damage other than reflux due to destruction of the valves.

The efficiency of calf muscle pump and amount of reflux are also associated with the development of ulceration. Nicolaides, et al, in a study of 220 unselected patients with CVD demonstrated that the prevalence of ulceration increased with higher ambulatory venous pressures.[12] The calf muscle function in terms of strength (peak torque/body weight) and endurance (total work) of patients with recently healed venous ulcers were significantly reduced compared to age- and sex-matched healthy subjects (p=0.049, 95%CI 0.3-18.4% and p=0.05, 95%CI 6.01-97.6 Nm respectively).[13] It has also been shown that patients with ulceration had worst ejection fraction compared to patients with varicose veins only.[14] However, in another study where the patients were matched for age and duration of disease, the amount of reflux was the most significant factor for the severity of CVD.[15]

Several studies have shown that the site and extent of reflux are important determinants for the severity of CVD and the development of ulceration. The greater saphenous vein is most often involved from the superficial veins. In fact, reflux involving both the above and below knee segments of this veins is the most common pattern of reflux in patients with ulceration.[7,9,16] Reflux in the lesser saphenous vein alone rarely causes ulcers unless it is combined with reflux in the greater saphenous and/or the deep veins.[16,17] The more deep veins involved the higher the prevalence of ulceration. However, among all deep veins, reflux in the popliteal vein has been shown to be very significant for ulcer development and it is also a predictor for poor healing.[17-20]

Most recent studies have shown that reflux in the superficial system is seen in 79%-93% of limbs with ulceration.[3,7-10] Reflux confined to the superficial veins alone is responsible for 17% to 54% of venous ulcers[3,6-9] whereas deep venous reflux alone accounts for 2.1% to 15%.[4,6,9,15] The prevalence of deep venous reflux in patients with venous ulcers ranges between 50% and 70% in various reports.[3,7-9,11] Most patients with ulcers (52%-70%) have incompetence in more than one system[7-9,21] and reflux in all three venous systems is seen in 16%-50%.[3,7-9,15,21]

When venous ulceration is due to superficial and perforator incompetence, surgical treatment may heal up to 90% of the ulcers with very good medium to long term results.[22,23] About 30-50% of patients with ulcers belong to this category.[6-9] Superficial vein ligation and/or stripping has worst results, with very high recurrence rates at 5 years, when there is reflux in the deep venous system.[23] In these patients additional procedures that are designed to improve the underlying abnormality may be required, and several studies have shown encouraging results.[24-27]

The significance of incompetent perforating veins remains controversial. Some investigators reported that incompetent perforators do not contribute to venous hypertension,[28-30] whereas others suggest that they are important.[9,10,31-33] Some recent reports have shown that subfascial ligation of these veins, combined with ligation and/or stripping of the superficial incompetent veins, is associated with high rates of ulcer healing and improved disease free intervals.[32,33]

A detailed study of the ulcerated lower extremity that will identify the etiology and outline the anatomic distribution of CVD is necessary prior to planning a surgical intervention in order to achieve the best outcome.

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I. PRIMARY VENOUS DISEASE: SUPERFICIAL INCOMPETENCE

SAVING THE SAPHENOUS VEIN

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The extent of the problem. Pure, superficial, primary venous incompetence[15] may be present (without large varicosities) for several years as a preliminary, subclinical stage. The incompetence of the SFJ and of the LSF (documented by color duplex in the San Valentino epidemiological study, 20,000 patients, 10-year follow-up) may be present in some 9% of subjects ages 45 to 65[16] and simple, uncomplicated varicose veins varicose in some 6% of asymptomatic subjects of an European population. However, only a part of these subjects will be referred for diagnosis and treatment. The total absence of valves in the superficial venous system is a rare occurrence (i.e. never verified in the 20,000 subjects of the San Valentino study). Therefore most subjects (70%) with simple incompetence of the SFJ could theoretically be treated with selective correction of incompetence.

Surgery. Repair of incompetent femoral vein valves in subjects with primary valve incompetence has been developed by Kistner in 1968.[1] The original method involved a venotomy in the femoral vein with placement of sutures to shorten the vein cusps under direct vision. Variations of open valve repair have been reported by others.[2,3,4,5] More recently closed valvuloplasty has been developed by Kistner.[4] Variations of external valvuloplasty including the use of intraoperative angioscopy to visualise the effects of the external valvuloplasty has been developed by Gloviczki at the Mayo Clinic.[5] All these techniques were aimed to treat deep venous system problems. The surgical method involves complete dissection of the femoral vein for 4 to 10 cm in order to place sutures on both sides of the vein wall at the level of the valves commissures. A progressive dilatation of the femoral vein after months has been observed in some subjects and this has led to the placement of several types of nets or vein cuffs in order to contain such dilatation and keep the valve competent.[2,6] The progressive dilatation of the vein – causing recurrence of incompetence - may be possibly due to the dissection of the vein wall with destruction of vasa vasorum supply and innervation. The superficial venous system, when initially incompetent, has been also treated with external valvuloplasty with initial (6-8 months)[7,8] and long term (> 10 years) satisfactory results. The first randomized study on superficial veins valvuloplasty (long saphenous vein) was initiated in 1986 by our group and appeared for the first time in the medical literature 1989. Therefore the idea of treating in a conservative way the LSV and the relative surgical method are relatively new. In the same period a conservative way of treating superficial venous incompetence based on information given by color duplex (which became available for venous evaluation in that period) were developed[9,10] and documented by long-term (5 years) randomized, controlled studies. The randomized, controlled study on the correction of superficial venous incompetence using SFJ valvuloplasty and selective LSV repair indicated a very important potential application of non-destructive superficial venous surgery.[11] In this period external valvuloplasty was also attempted in a limited number of patients with superficial venous incompetence, dilated but not varicose veins.[12] A randomized pilot study was also conducted using an external Gore-tex patch applied in tubular shape at the proximal part of the SFJ.[12] The control treatment was simple, proximal ligation of the SFJ. No complications were observed in all 28 cases treated. Complete correction of venous incompetence was achieved in 21 cases and partial correction in 5. Competence persisting was still present after 6 years. Also no significant reaction to the Gore-tex patch or infection were observed in the following 6 years.

In the following years a few reports have indicated that external valvuloplasty of the deep venous system too are effective in reducing and controlling both superficial and venous incompetence[13,14] even in protracted follow-up (>3 years).

Data on file from our group (the study is still in progress with the aim to achieve 20-year follow-up before review of data) indicate that long-lasting, effective competence of the superficial and deep venous systems may be achieved both after external valvuloplasty or by placing an external vein support (i.e. Gore-tex patch or tubular graft cut and sutured according to the vein shape) systems. However, longer studies are needed to confirm the possibilities, limits and potential clinical applications of non-destructive venous surgery.

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Table: WHY TO SAVE THE LONG SAPHENOUS VEIN?

1. In case of DVT less complications (edema, ulcerations, etc.)
2. No obstruction (outflow is better saving the vein)
3. The vein may be useful as a graft
4. Lower costs
5. Noninvasive tests tell you precise sites of incompetence. You do not need to destroy the whole vein.
6. It is physiologically better to correct incompetence more than system destruction.
7. To save the integration between the deep and superficial system (which is a single, composite system) is important for long term outcome.

AMBULATORY VENOUS SURGERY VERSUS SCLEROTHERAPY

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Great advances have occurred in recent years in venous surgery that have allowed us to perform a walk-in walk-out type of surgery now called ambulatory vein surgery. The development of this type of surgery is at different levels throughout the world and has doctors of different specialties involved in it, including physicians, surgeons and sclerotherapists.

Sclerotherapy over the last few years has also made tremendous advances principally more and/or better solutions or sclerosants available in various countries, more research into the scientific basis for sclerotherapy, better or more convenient methods of compression, and better placement of needles into veins using duplex ultrasound.

When comparing and contrasting these two methods of treating varicose veins I think we have to compare ideal ambulatory venous surgery done with good anaesthesia, minimal access incisions, hook phlebectomy, and compression, with ideal sclerotherapy using what we think is the best sclerosant, of course using the best techniques and achieving good compression for an adequate period of time with minimal patient discomfort. Both of these techniques therefore will produce good results and hopefully the best possible result.

In years past, surgeons performed traditional major varicose vein surgery in hospital and this was quite an ordeal for the patient. Surgeons soon learned that less radical, better tailored operations using new techniques of minimal access would enable the patient to be out of hospital quicker and back to work earlier with far less morbidity. This progressed to the use of local anaesthesia and hook phlebectomy, so that a lot of this surgery is now done in the rooms.

Traditional European sclerotherapists held a strong view that all types of varicose veins could simply be treated by repeated and extensive courses of sclerotherapy. The dedicated sclerotherapists are divided in their opinion as to the wisdom of this approach. Many have seen the wisdom of surgical removal of large varicose veins, which produced a more effective and long lasting result and with the advent of hook phlebectomy followed by compression sclerotherapy, better and more long lasting results were produced. Some sclerotherapists, however, armed with duplex scanning for placement of needles in bigger and bigger veins, have been seduced into believing that injection of large volumes of high concentration sclerosants into the saphenous trunks would produce just as effective a result as surgical removal of the vein. This has prompted a push in some sections for routine ultrasound guided placement of needles and sclerotherapy for even the most major of varicose veins cases.

Ambulatory venous surgery can be done under general anaesthesia, in a day surgery centre or licensed operating room. The patient is in "hospital" usually for about four hours, there is no restriction on the extent of vein surgery performed, including high ligation of the long saphenous or short saphenous veins, stripping, ligation of perforators and multiple phlebectomy. This is still the recommended treatment by surgeons, particularly vein surgeons, in patients suffering from major upstream incompetence. Ambulatory venous surgery under local anaesthesia can be performed in an office setting, either in your consulting room or procedures room. This would basically involve multiple small stabs along the leg and hook phlebectomy of segments of the saphenous trunks, large saphenous tributaries or large reticular veins. It would not involve saphenofemoral or saphenopopliteal ligation or stripping. This is the major form of ambulatory venous surgery that I wish to compare with sclerotherapy.

If the good quality venous incompetence duplex scan that you have ordered or performed on your varicose veins patient shows major upstream incompetence I still believe that the four hours in hospital, general anaesthesia, doing as much as you can to remove those veins surgically, with the patient going home later on that day, but perhaps having some days off work is the best way to proceed in our Australian medical environment. If a patient presents, however, with segmental saphenous vein incompetence, saphenous tributary disease, or with early residual or recurrent varicose veins then the office setting under local anaesthesia is ideal for removing the raised palpable veins and then following this up with sclerotherapy for what is left. This procedure under local anaesthetic is ideal for raised tributaries over bony prominences, such as over the patella or the front of the shin or ankle, or across flexures such as in the popliteal fossa, or for more proximal veins high on the thigh.

Let us now turn to ideal sclerotherapy. There is no doubt that

compression sclerotherapy is suitable for tributaries of the saphenous vein, reticular veins, venules and telangiectasias. When it is performed on large saphenous tributaries and even incompetent saphenous trunks, it is effective at reducing the size and the symptoms of these veins. By itself it is unlikely to control major trunkal incompetence long-term. Sclerotherapists have used improved diagnostic imaging with duplex scanning to improve the placement of their needles under ultrasound control. In so doing, they are canulating bigger and bigger vessels, but they still need large volumes of high concentration sclerosant to effectively do any damage, mostly thrombosis possibly sclerosis, to these large vessels. I am speaking specifically here of the long saphenous and short saphenous trunks. This technique of ultrasound guided sclerotherapy, particularly to the saphenofemoral junction manages to temporarily occlude or cork this junction, taking the pressure off the varicosities distally. Subsequent sclerotherapy to large veins distally when the pressure is reduced will have a beneficial effect. On some available data, the recanalisation rate of the long saphenous vein at two years is over 40% and so the veins begin to re-open even in the best of hands and the process has to be repeated. It is my strong contention that these major trunks are best dealt with surgically, with high ligation and limited stripping, and at the same time surgically removing the larger varicosities, and thus saving the sclerotherapy for residual and recurrent veins.

Ideal sclerotherapy must use what you regard as the ideal solution. In Australia from 1991 to 1995 we performed an open clinical trial in an effort to introduce aethoxysklerol into the country, comparing it with the two established solutions of sodium tetradecyl sulphate and hypertonic saline (20%). The trial included 120 doctors who were experienced injectors, and a series of 34,878 legs that were injected, 40% principally for varicose veins, and 60% for surface or spider veins. The results of that trial showed most injectors to believe that aethoxysklerol had a better efficacy than the established solutions, was much less painful for the patient on injection, produced less frequent and less severe injection ulcers, pigmentation and phlebitis. The clinical occurrence of deep vein thrombosis in that trial was one leg in 7,000 injected. This has lead a large proportion of Australian doctors performing sclerotherapy to chose aethoxysklerol as their preferred or best sclerosant.

We must of course compare potential complications of the two forms of treatment that we are discussing. With minimal access technique the scarring with hook phlebectomy is indeed minimal. Trauma to cutaneous nerves and subcutaneous lymphatics is certainly possible but rare and with experience of this technique can be avoided. When looking at sclerotherapy, even using one's ideal solution, the problem of retained blood in the vein, thromboembolism, toxicity of the sclerosant, telangiectatic matting and injection ulceration remain possible, but again with a low incidence.

As individual doctors practising on their patients daily, we encounter many variables that go into our decision to advise the patient to have one treatment or the other. As far as the treating doctor is concerned, he may have very little time available and it is obvious that the surgical removal of veins takes longer, but he is rewarded proportionally to his time expended and so the cost is higher. If he is a sclerotherapist or physician who is not used to actually making incisions and pulling things out then this may influence him towards sclerotherapy, but if he is a surgeon who finds hook phlebectomy quick, easy and satisfying then this direction may be followed. The doctor's access to facilities and equipment is also a vital factor. The patient also presents variables in the eventual decision and sometimes they demand one way and one way only to be treated. The level of financial remuneration governed by their level of medical insurance is also going to be a factor, but what is a very important clinical factor is the site of the veins on the legs and over any difficult site surgical excision of raised palpable veins in my opinion does a lot better than sclerotherapy.

And so ambulatory venous surgery can be used on saphenous trunks, major saphenous tributaries and large reticular veins. It is more time consuming than sclerotherapy, but one treatment is all that is required to remove the particular vein. Compression following ambulatory venous surgery need only be for 48 hours and there is a very low medium term recurrence rate with very good patient satisfaction. Pigmentation, ulceration and phlebitis do not occur after this technique. Sclerotherapy, looking at the same parameters, can be used on large and small veins alike from saphenous trunks all the way down to telangiectasias. It is a much quicker technique to perform than ambulatory venous surgery, but often requires multiple treatments and longer compression, depending on the size of the vein, up to six weeks. There is a variable medium term recurrence rate and variable patient satisfaction. When high volumes of high concentration sclerosant are used this has more chance of leading to pigmentation, ulceration or phlebitis.

In summary it is my contention that using the best available minimally invasive ambulatory venous surgery for the larger vessels that are raised and easily removed should then be followed by sclerotherapy, using the best sclerosant available to you, and I consider that to be aethoxysklerol. This will yield your best results. Ambulatory venous surgery versus sclerotherapy therefore should now read ambulatory venous surgery followed by sclerotherapy for best results.

NEW TRENDS AND OPERATIVE TECHNIQUES IN THE MANAGEMENT OF VARICOSE VEINS

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The advances in noninvasive diagnostic vascular technology have allowed us to study with accuracy the anatomy and pathophysiology of the venous system. With the use of the bi-directional Doppler, color-flow duplex scanner, and air plethysmography, we can investigate the venous pathology in a very precise manner and plan the appropriate treatment. As a result of these technological advances, we have learned that the main trunk of the saphenous vein may be quite healthy despite the presence of large clusters of varicose veins in one or more of its tributaries. Sparing a healthy saphenous trunk has become of importance for two main reasons: the first reason is that it makes no sense to excise a healthy organ that is performing its assigned function in a satisfactory manner.[1] The second reason is that the saphenous vein is the most important autogenous conduit utilized as bypass, patch, or substitute in the arterial system.

Despite these reasons, surgery for primary varicose veins with preservation of the saphenous trunk is controversial. There are several published reports that support sparing the saphenous vein. Large[1] studied 202 patients in whom he performed stab avulsion and proximal ligation only in cases of sapheno-femoral incompetence (51.5%). There was a 10.5% recurrence rate at three years. Hanrahan, et al,[2] performed duplex examination of 54 lower extremities with varicose veins. Seventy-two percent of these patients manifested branch incompetence alone. The relationship of incompetence of the sapheno-femoral junction to the presence of superficial varices was inconsistent. Deep venous incompetence was present in 41% of the extremities. These data support the position that the preoperative assessment of reflux in vein segments is more appropriate than routine stripping of the main trunk or ligation of the sapheno-femoral junction. Koyano and Sakaguchi[3] studied 337 legs of 208 patients using Doppler and calf compression-release. They found that 66% of these patients had incompetence of the long saphenous vein. They were able to demonstrate reflux in segments of the saphenous vein. Based on Doppler findings, they performed selective stripping in 80 limbs in which only the segments with venous reverse flow were selectively removed. Their follow-up studies showed that selective stripping operations were as satisfactory as those in which the standard stripping operation was carried out (189 limbs). Additionally, the incidence of saphenous neuritis was 4.8% in patients with selective stripping versus 27% in patients submitted to full stripping.

Other studies have challenged the preservation of the saphenous vein. Munn, et al,[4] reported that routine greater saphenous vein stripping had a lower incidence of recurrent varicosities (18%) than was seen in patients in whom the saphenous vein was preserved (29% recurrence). Of interest, however, was the finding that the patients were more satisfied with the results of the leg that was not stripped. This was the result of a high incidence of nerve injuries in the legs which had full stripping.

Sutton and Darke[5] performed peri-operative retrograde saphenography to study 80 incompetent long saphenous veins in 60 patients with primary varicose veins. They found that there were varicose changes in 65% of the greater saphenous veins and stated that the incompetent vein did not appear to be a suitable arterial replacement. They concluded that insufficiency of the saphenous valves may contribute to the development of venous ulceration. In these studies, Hunterian perforators of variable size were seen on 70 saphenograms (87%) and these investigators concluded that stripping of the incompetent greater saphenous vein would certainly disconnect such perforators and reduce the incidence of both recurrent varicose veins and persistent ulceration. The diagnosis of saphenous incompetence was performed with Doppler and Valsalva maneuver and confirmed by intra-operative phlebography.

MacFarlane, et al[6] performed phlebography in 35 extremities that were going to be operated on for varicose veins. They demonstrated that the segment of saphenous vein below the knee does not undergo varicose dilatation. In seven of nine patients examined post-operatively, the segment remained patent after the vein above had been stripped. Additionally, 75% of the greater saphenous veins above the knee were not dilated or tortuous. These authors concluded that varicose veins resulting from sapheno-femoral incompetence can be treated surgically by stripping the long saphenous vein only to the knee and yet still leave a non-varicose segment for possible future utilization. In 75% of their cases, the greater saphenous vein above the knee was incompetent, but not grossly dilated or tortuous. In the remaining 25%, the above knee portion was severely diseased and would have been unsuitable for any form of bypass grafting.

McMullin, et al,[7] studied 54 limbs with duplex scanning before high ligation and multiple avulsion of primary varicose veins. Duplex scanning confirmed sapheno-femoral incompetence and excluded short saphenous incompetence. Of interest is that in 24 of the 52 limbs in which the junction had been ligated, there was persistent reflux down the long saphenous vein. In only two limbs was this reflux attributable to mid-thigh perforating veins. Photoplethysmography was performed before and after surgery and the venous refilling time measured. Using this method to evaluate results, the authors concluded that high ligation without stripping fails to control significant reflux within the long saphenous vein in a high proportion of cases. However, van Bemmelen and Strandness,[8,9] by applying graded pressure in a cuff placed proximal to the valve segment investigated, have shown that a velocity exceeding 30 cm/sec is necessary to prompt the normal valve to close. With velocities lower than this, the valves will not regularly close and reflux can persist, giving a false impression of the status of the valves in question. After ligation of the sapheno-femoral junction, there is not enough pressure or sufficient blood velocity to close the valves below the junction. For this reason, trunk reflux may be detected by color duplex scanning after compression-release at the calf.

At our institution, patients who are going to be submitted to varicose vein surgery for primary venous insufficiency have examination of their saphenous system by duplex scanning in the upright position before surgery. The saphenous diameter is measured at the femoral junction, and upper, middle, and distal thirds of the thigh. Depending on the height and weight of the individual, saphenous veins which were found dilated, tortuous, and irregular, and which demonstrate reflux, are routinely stripped from groin to just below the knee (Fig. 25.1). However, a saphenous vein trunk of normal size, which tapers down uniformly without tortuosity or aneurysmal dilatations, is preserved.

Even though there is evidence in the literature and in our clinical experience that the saphenous vein can be spared in those cases of patients with varicose veins in whom the saphenous trunk is found to be healthy, the definitive answer to the controversy of stripping the saphenous trunk or preserving it in a selective manner must await prospective randomized studies using duplex scanning and air plethysmography to evaluate long-term results. The diameter of the vein at different levels and the soundness of the trunk are considered in the randomization to strip or to preserve.

Another method of achieving competence of the sapheno-femoral junction in patients with varicose veins has been the application of venous cuffs at the first centimeter of the sapheno-femoral junction. The cuff is tightened until competence is achieved. The cuffs are made of Dacron or other prosthetic materials. Again, long-term results in a sizable sample of patients with hemodynamic information are lacking. Besides, the insertion of prosthetic material in young individuals, which form the majority of patients with primary varicose veins, is costly and, in our opinion, not justifiable.

Varicose Vein Surgery in a Bloodless Field

During the last three decades, we have learned to appreciate the benefits of a bloodless field as practiced by the orthopaedic, plastic and cardiac surgeons. In vascular surgery, the pneumatic tourniquet has been utilized to avoid clamping calcified vessels during the performance of distal anastomosis in bypass surgery. We have used the pneumatic tourniquet extensively during the resection of hemangiomas and other vascular malformations in patients with congenital vascular anomalies. More recently, the pneumatic tourniquet has been utilized to render a bloodless field during the endoscopic subfascial ligation of perforators. At our institution, we have used the pneumatic tourniquet in patients with extensive and grossly dilated varicose veins. In the past, patients with extensive varicose veins occupied several hours of our operating time. The introduction of the tourniquet has dramatically reduced the operating time and has allowed us to perform a complete removal of all varicosities without blood loss. The technique consists of exposure of the sapheno-femoral junction before applying the tourniquet. After division of the saphenous vein, in those cases in which stripping is to be performed, a flexible stripper is introduced through a small incision below the knee and retrieved at the groin. The vein is prepared for invaginated stripping, and the inguinal wound is closed in layers. A pneumatic tourniquet is applied at the upper thigh after wrapping the leg tightly with an Esmarch rubber bandage. The tourniquet is inflated at 300 mmHg and the operation can then be performed in a bloodless field. In 78 patients with extensive varicose veins and 97 congenital vascular anomalies operated upon, the average operating time has been 55 minutes (range: 45-80 minutes). When necessary, stripping is performed with the tourniquet inflated. The vein stripper slides easily under the tourniquet. The wounds are usually closed with steri strips, the extremity is carefully padded and a compression bandage applied. The patient remains in the hospital for a few hours and goes home during the late afternoon.

Ambulatory Venous Surgery

Ambulatory venous surgery has been practiced for many years. In 1930, De Takats[10] published his experiences with this technique. Nabatoff,[11] in 1972, described his method of complete stripping of the varicose veins as an outpatient procedure. The competition for hospital beds, the long waiting lists for varicose vein surgery and financial considerations have stimulated surgeons to seek options to operate as many cases as possible without compromising the hospital capacity. There are numerous reports in the literature dealing with this subject.[12-16] In a recent publication, Baccaglini, et al,[17] conducted a multicenter trial in 2568 extremities with varicose veins. Different anesthesia techniques were utilized (local, spinal, general, and local plus sedation). Post-operative hospitalization was required in only two cases. Post-operative complications included headache after spinal anesthesia and residual paraesthesia after femoral nerve block. The consensus of surgeons participating in this protocol confirms that surgery of varicose veins can be carried out on an outpatient basis with similar complications as observed in hospitalized patients. The authors conclude that outpatient surgery is cost-effective and diminishes the demand for hospital beds, while assuring that the quality of patient care is maintained.

Crochet-hook Vein Excision

In 1957, Thomas T. Myers, of the Mayo Clinic, reported that extensive removal of all varicosities, followed by ligation and stripping of the greater and lesser saphenous veins, was accompanied by 85% good-to-excellent results at 10 years. Since then, many thousands of patients throughout the world have been submitted to this procedure. The extensive scarring of the operation and the length and tediousness of the procedure have prompted the surgical community to seek new avenues of treatment for varicose veins. In 1975, Rivlin[18] reported the use of mini-incisions and fine pointed forceps for the extraction of the varicose tributaries of the saphenous vein. Since then, the method of stab avulsion through mini-incisions has been extensively practiced throughout the world. Special hooks have been developed by Müller,[12,13] Oesch,[14] Varady,[19] and Villavicencio.[20] The technique of stab avulsion phlebectomy has been used extensively over the last 20 years by Villavicencio. A set of specially designed hooks has been routinely utilized in every case of varicose vein surgery during the last 14 years.

Technique

With the patient in the upright position and the extremity shaved, the varicose veins are marked with indelible ink. The operating surgeon should mark the patient. The extremity is placed in the Trendelenburg position, and the crochet hook phlebectomy is performed through 2-3 mm incisions done with a number 11 scalpel blade. Incisions must be made parallel to the axis of the vein to prevent piercing it. The hook is introduced with its flat side under the vein. Once the tip of the hook has passed on the other side of the vein, it is rotated so its tip is pointing toward the skin. At this point, it is pulled back, lifting the hooked vein while counter-pressure is applied over the skin. A loop of vein is exteriorized by pulling gently. The maneuver is repeated through several incisions placed along the course of the vein until the complete venous segment has been excised. Incisions are placed approximately 1-2 inches apart, and at the end of the procedure are closed with steri-strips. Compression pads are placed over the wound and the extremity is wrapped with a thick roll of gauze and a firm elastic bandage. The small incisions heal uneventfully and are practically invisible after several months.

In summary, great advances have occurred in the management of venous disorders. The surgical management of varicose veins is more objective and based on sound hemodynamic and anatomic principles. However, the most important aspect of this progress is its continuous evolution. Research protocols are in progress to solve some of the most important problems in the management of these diseases. This is an exciting time in which technological advances have provided the necessary tools for sound investigation.

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CASE OF SUPERFICIAL INCOMPETENCE

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Large Recurrent Circumferential Ulcer Due to Primary Incompetence of Long Saphenous Vein (Possibly Perforators) with Competent Deep Venous System

A 65-year old lady from the Big Island of Hawaii, who works in a coffee plantation and has 5 children, developed increasing varicose veins during the pregnancies. She has no previous history of DVT. Thirty years ago, in the end of the 60's she developed the first ulceration of the right leg. It developed gradually into a circumferential ulceration and amputation of the leg was recommended. In 1972, Dr. Yee in Honolulu operated on the patient with skin grafting and the ulcer remained healed for several years. Since the beginning of the 80's she has had several recurrences but never sought medical advice due to the previous threat of amputation. Since the beginning of the 90's the ulcer never healed and has developed into a large painful circumferential ulceration that she was treating with local herbs without compression. Because of the pain she again consulted Dr. Yee in November 1995 and she was referred to the vascular service at Straub in Honolulu.

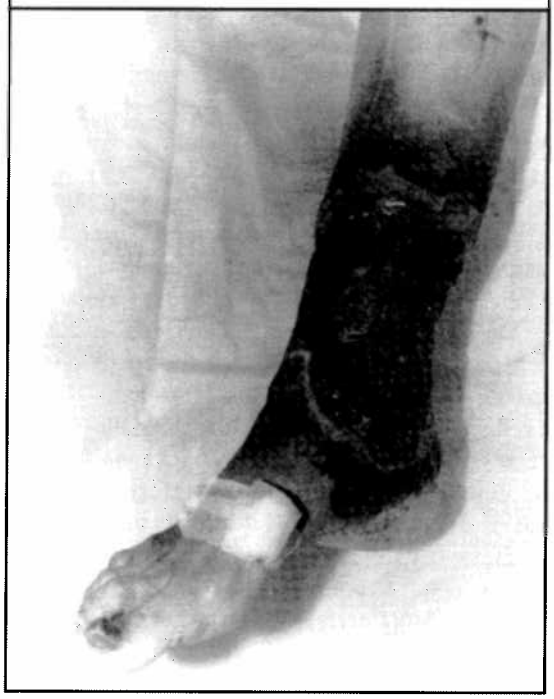
At admission she had a circumferential, granulating ulceration at the lower part of the right leg with a length of 12 cm (Fig. 1). Cultures showed growth of staphylococcus and pseudomonas. She had normal pulses in the posterior tibial and dorsalis pedis arteries. Hand-held Doppler examination showed severe reflux of the long saphenous vein with competent deep veins.

Duplex scanning of the veins showed patent and competent deep venous system. There was severe reflux of the long saphenous vein which communicated with the short saphenous vein through a Giacomini communicator. SSV was competent. There were no incompetent perforators, but the area under the large ulceration could not be studied. Ascending venography showed no obstruction and no signs of previous DVT; the deep veins as well as the long saphenous vein were widely patent; there were several perforators between the posterior tibial vein and the long saphenous vein (incompetent?)

Descending venography of the right leg through catheterization of the left femoral vein showed grade 4 reflux of the long saphenous vein. The deep femoral vein was competent and there was a slight grade 2 reflux of the proximal superficial femoral valve (Fig. 2).

CEAP CLASSIFICATION: C2,3,4,5,6s; Ep; As; Pr2,3

Figure 1.– 65 year old woman at admission with a circumferential granulating ulceration of the right leg with a length of 12 cm.



(Figures 2 & 3 on the next page)

DISCUSSION

DR. O'DONNELL: I think everybody agrees that ligation and stripping is most appropriate. What isn't settled is whether the perforators should be treated. I found it fascinating that we have a new anatomical finding that the perforators are now related to the long saphenous vein! The questions are: 1) do you treat the perforators at this time? And (2) how do you treat the basic lesion, the skin ulcer? Dr. Raju, would you place a skin graft on this ulcer at the same time or leave it alone?

DR. RAJU: I think I would just strip the saphenous vein and skin graft the ulcer at the same time. However, our practice has been altered a little bit in recent years simply because the gatekeepers don't allow us to pre-admit these patients. There is no luxury of admitting these patients for a week and give them antibiotics even though you can do it with a great deal of effort. We just admit them in the morning unless the patient is septic. If the wound is reasonably clean, we proceed and have even done deep venous reconstruction under those circumstances. Even though you would think that the incidence of infection would be high, it is not very different from

Figure 2.– Descending venogram of the right leg through catheterization of the left femoral vein showed severe reflux (Kistner grade 4) of the long saphenous vein (upper right arrow), competence of the deep femoral vein (left arrow), and slight reflux (Kistner grade 2) of the proximal superficial vein (lower right arrow).

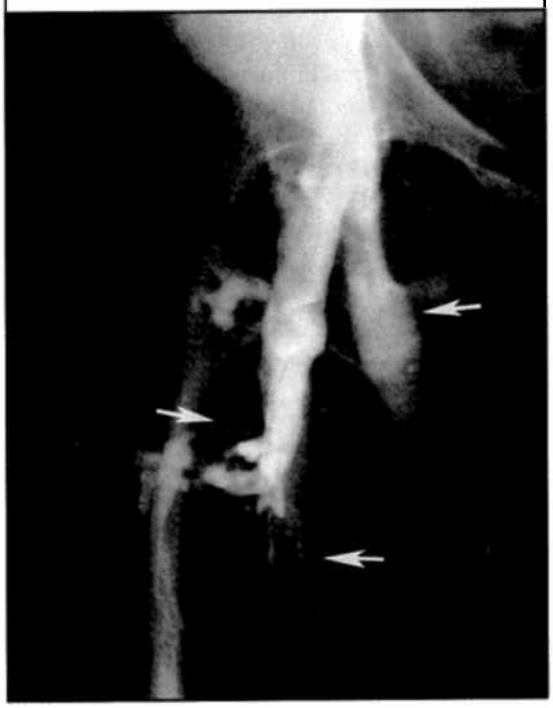
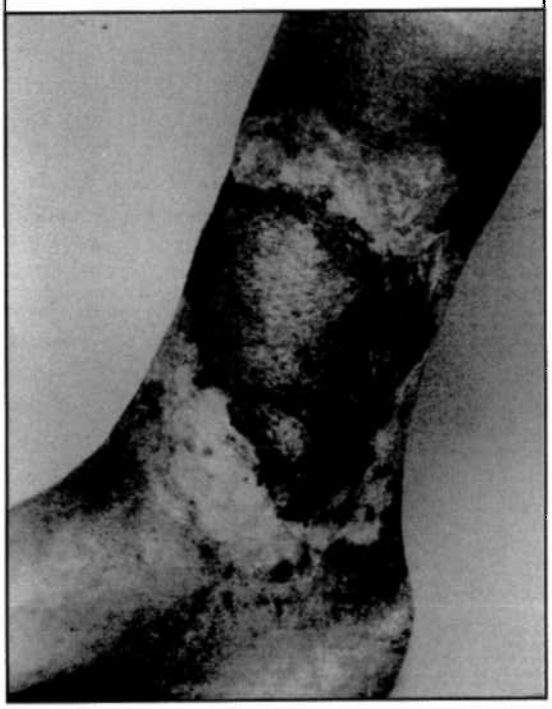


Figure 3.– Healing of the circumferential ulcer after high ligation and stripping of the long saphenous vein with skin grafting. The patient has remained healed for more than 3 years.



clean contaminated cases. So I think there should be less preparatory time before you do what you need to do.

DR. O'DONNELL: Ralph, what would you do? Are you going to use a venous pump for these patients?

DR. DEPALMA: No! I would operate on this patient, strip the long saphenous probably from above downward and all the way to the ankle.

DR. O'DONNELL: Why are you going all the way to the ankle?

DR. DEPALMA: Well, I'd get as close as I could to the ulcer. I think about 20 percent of saphenous vein have deep perforators associated with them by other communicators at the same level. Dr. Cockett has shown pictures of this anatomy. I've seen it frequently in my own dissections. So I think in this case the saphenous vein is the main point; I wouldn't worry separately about the perforators. I am concerned, however, about the combination of organisms present here. They reported staphylococcus and pseudomonas in this case. I would be very worried about these. I would clear that patient for a 24-48 hours hospitalization with IV vancomycin and an effective anti-pseudomonas drug. I would probably treat the ulcer locally with sulfa-mylon after this treatment. I'd go ahead and strip. I would not attack the deep system at this point. Then I'd graft, all at one operation. I would mesh the graft and keep that patient in the hospital for three to four days on IV antibiotics with the leg elevated.

DR. PERRIN: I agree with Ralph DePalma. I will try first to heal the ulcer by compression, lateral compression, but I will operate on him before I complete healing. I will prescribe antibiotics, systemic antibiotics, the day before or four or five days before the operation, but I will do high ligation and stripping.

DR. O'DONNELL: You strip down to the ankle, and I think it's nice to say that, but as I remember, this ulcer is circumferential. So what happens to the saphenous vein across this ulcer? Dr. Perrin, where would you strip to?

DR. PERRIN: I would strip to the ankle in this case.

DR. O'DONNELL: It would be pretty tough to get anterior to the medial malleolus in this patient, but maybe you know better.

DR. DEPALMA: I have an opinion. I'd make an incision over the saphenous at the malleolus, and pass a lighted probe proximally. If the vein is there, I would probe it upwards with the lighted probe to see if it went to the knee. If it were in continuity under the ulcer, I would strip it. I'd remove the long saphenous completely to the ankle. I probably would make a malleolus incision -- there's room there. When you can see the anterior malleolus the vein is probably under it. So I'd pick it up here first.

DR. KISTNER: I agree with cleaning it up, probably including intravenous antibiotics, and then going ahead with surgery. Surgery would be similar to what the others have suggested. We begin with ambulatory treatment with unna boot on these cases to see how they respond. The response may be dramatic and helps make a quicker, safer treatment phase. If this is not successful, I'd go ahead with surgery. The question I'd like to ask is whether those who would treat with stripping and skin grafting would use prophylactic anticoagulants? This is a patient who has venous disease and will be on bed rest for a period of time. I'd ask Ralph about that.

DR. DEPALMA: If I were going to put the patient to bed, I would probably use Lovenox in prophylactic dose, 30 milligrams BID, Sub Q.

DR. O'DONNELL: The rest of the panel pretty much concludes

they would use prophylaxis here.

DR. DALSING: I have one question. Was that a swab or did you actually take some tissue and send it for culture, because I think it makes a difference. A swab culture can grow almost anything if it's an open wound without protective skin. So I'm not so sure I'd be that worried about systemic or even significant local infection in the wound. This looks like a real clean ulcer to me. So I'm not so sure I'd be as aggressive with antibiotics. I'd probably administer perioperative antibiotics. In terms of therapy, I agree with the stripping operation probably being just above the ulcer and ignore the perforators for now.

DR. O'DONNELL: Peter, would you add a SEPS procedure because there are perforating veins here. It's hard to believe that a patient with an ulcer like this doesn't have incompetent perforating veins in addition to her greater saphenous incompetence. Would you go ahead and do a SEPS?

DR. GLOVICZKI: This is a patient I would not necessarily insist to perform SEPS on. I would operate on the superficial system. I would strip the saphenous vein all the way down as far as I could above the ulcer. It makes no sense to leave in an incompetent calf saphenous vein in this patient. This patient does not have proven significant perforator incompetence, and with that amount of circumferential ulceration, it may be technically difficult to gain good access to the subfascial space.

DR. O'DONNELL: Just to get the panel's opinion, would anybody use Apligraf?

DR. GLOVICZKI: I have recently used Apligraf several times in this situation. If the ulcer is infected however, it is not going to work. So you really want to clean it up thoroughly before you put the Apligraf on it.

DR. BELCARO: This is a very good example that surgery should be performed by a group of people with different competences. When you come here and talk about saphenous vein stripping and perforators, it's only part of the problem. Surgery is evolving. You have to consider many aspects. You have to be a physician expert in medical therapy. You have to have different competences. I know we say that for hammers everything is a nail, but the point is that surgery is only part of the solution of the problem because you can do anything and after three months you have the patient with the same problem. So the focus of a faculty like this is to address that surgery is changing. Surgical treatment is only part of a complex treatment, which includes medical treatment, psychology, tutorials, and several other aspects. If we focus only on stripping, and interrupting perforators, we really miss the point because the evolution is not in simple superficial competence but in integrating different levels of competence.

DR. O'DONNELL: Well, I appreciate those remarks, and I guess that defines who a vascular surgeon is, in the United States at least. Vascular surgeons do medical as well as surgical therapy. I agree with you that it needs to be a team approach. To think that vascular surgeons in the United States, however only focus on surgery I think is a miscalculation.

DR. CAPRINI: I just wanted to bring up a couple of things because we have such an outstanding panel here just to see what they might say. We have been taking a look at the saphenous with the duplex. In many cases just below the knee the saphenous turns normal so we usually don't strip the calf part of the saphenous. However, in cases like this case report we almost always see that the saphenous is big all the way down to the ankle with duplex. When it is, we try to strip all the way to the ankle. That would be what I would use to determine whether or not to fully take out the vein. Another thing is that I notice this patient has pretty bad toes, and one of the things that we found in taking care of some of these patients is to make absolutely sure to eradicate any elements of fungal infection in those toes before we do a definitive operation. We've also used pneumatic compression, but with foot compression, or foot and calf compression, we've gotten pretty good results, especially if like Raj said, there was some problem getting that patient right into the hospital when they didn't want to come. The other thing is that I think as far as prophylaxis is concerned this patient needs a risk assessment. If that patient has a lot of risk factors, then that patient should have prophylaxis. With surgery in age over 65, something like low molecular weight Heparin would be indicated. Then finally, a comment about after care. As was mentioned, a lot of these patients wouldn't wear their Class II appropriate stocking, but we found sometimes we can get them to wear the circaid device.

DR. THORPE: It concerns me that two of the speakers said they didn't think venography was of any value, and I want to say that I think it's very important as part of the workup. In fact, I've seen a patient who had this situation, stripping and valvuloplasty, and later underwent an amputation. Now, when ulcers start to happen on the opposite leg, we look at the IVC. It's possible that in some of these patients there's a high caval stenosis or a caval occlusion that can be treated. We didn't know how to do this ten years ago but now we can look in the iliac or cava for an obstructive problem that could account for venous hypertension in one leg or both legs.

DR. O'DONNELL: Well, that's interesting. Certainly in our unit we probably would not get a phlebogram. We'd rely on duplex scan, but I'd be curious what the panel would do. How many would opt for obtaining a phlebogram. Three out of the panelists agree with you, Patricia. So that's interesting, probably in an advanced case.

DR. MYERS: Can I propose a totally different approach to this patient than that given by the panel? I think Mark Malouf made the most important comment, and that is that removal of the long saphenous vein is not required to heal this ulcer, but is required to prevent it from recurring. Therefore, I don't personally believe there is any hurry whatsoever to remove the long saphenous vein. Now, if you look at the ulcer, a lot of it is over quite mobile parts of the leg, and I consider that ambulatory treatment has the potential to markedly reduce its size and possibly even get it to heal without the need to admit the patient to the hospital. I would certainly start on a regime of local dressings with simple gauze, and an extremely tight compression bandage to get rid of the venous hypertension that's causing it with the expectation that the combination of contraction and epithelial growth into the ulcer will markedly reduce its size and the extent of the skin graft that you may or may not require at the end. I think this is an eminently healable ulcer. Once it's healed, then at some time at your convenience, perhaps even on an ambulatory basis, the long saphenous vein can be removed purely to prevent recurrence and not to help the healing of the ulcer. This would result in the least time in hospital and the greatest praise from your medical insurance group.

DR. O'DONNELL: Do the rest of the panelists agree that they would not strip the vein, that they would try a course of compression

and special wound mendicants rather than stripping? Do you all agree with that?

DR. DEPALMA: No, I don't. I think it would take a long time to get this ulcer to heal, and I think it would be sort of what I describe to my residents as "diddlefritz." I don't think I'd get that to heal in any reasonable time. I couldn't, maybe Kenneth could. I would just get right to surgical treatment and do it.

DR. KISTNER: While external treatment may get it to heal, I think it heals quicker if you take care of the venous pathology and this pathology is easy to take care of. I think it should be repaired surgically.

DR. O'DONNELL: Maybe you could comment because there's a thread of Kevin Burnand's thesis of "predestination" to venous ulcer. In New England a long time ago there was a religious movement that held everybody was predestined, either to heaven or hell. As Burnand espoused yesterday, ulcers are similarly predetermined in to which ones are going to heal and which are not. Can you predict whether this ulcer is going to heal or not depending on whatever you give, Bob? Do you subscribe to that predestination theory?

DR. KISTNER: No, I don't.

DR. GLOVICZKI: I concur with Bob that although you could reach complete healing without treatment, why would you want to leave a largely incompetent greater saphenous vein and have a persistent, otherwise treatable cause of venous hypertension even during the healing period? Once the infection is treated, I would treat the underlying cause and operate.

DR. GOREN: It is a pity that additional data such as VFI and the size (by duplex) of the long saphenous vein is not available. I would like to congratulate Dr. Villavicencio for his excellent presentation of sclerotherapy approach. May I quote the late Dr. Tim Myers from the Mayo Clinic who said many years ago that "sclerotherapy (for truncal varicose veins) is a return to the Dark Ages of medicine." This statement is also true today in spite of the introduction of the guided injection method. Superficial reflux was shown by Bjordal in the early seventies to be the cause of elevated ambulatory venous pressure in truncal varicose veins, and its elimination is a must for a long lasting result. But I take issue with the arbitrary removal of the entire length of the long saphenous vein to achieve that goal. Prof. Hach has classified long saphenous varicosities according to the location (in the saphenous trunk) of the end reflux point into four groups. Only in group IV (the "straight through" incompetence of Tibbs of Oxford) will the ankle to groin stripping be indicated. In 1082 consecutive operated long saphenous varicose limbs in my practice, only in 7% (66 limbs) was there need for a total ankle to groin stripping. In the majority of cases (74% or 704 limbs) the end reflux point was found located just below the knee (Hach group III), thus a stripping to this level will was performed. In this particular case, since the end reflux point was not assessed, any blind approach would be a questionable approach. I fully subscribe to Dr. Belcaro's suggestion that maximum possible preservation of the long saphenous vein trunk is imperative in all cases. The Hach classification based on the location of the end reflux point will aide in this noble quest.

DR. O'DONNELL: Yes, and to your point, I think the organizers did provide the information. They did a descending phlebogram which was interesting. It was a Grade IV reflux.

DR. DEPEDRO: May I have the lateral projection slide of the ulcer. Although I am a vascular surgeon, I think that the point should be at first what we do in our country in a case like this. We think that unless we mobilize the fixed tibial tissue via physiotherapy and take this to a lesser stage of severity, we cannot treat the rest of the pathology of the venous reflux. We first utilize physiotherapy to liberate the joint and improve the muscular pump function, and afterwards we do surgery. If this is not done, the wound is predestined to poor healing no matter what kind of surgical technique you use.

DR. O'DONNELL: That's a very good comment, and it goes along with what Dr. Belcaro said, that this should not just be a bunch of surgeons. It should be a multi-modality team. In addition to the psychiatrist and the social worker, now we've added the physiotherapist. So I think that's a very good point.

DR. ABU-BAKER: I'd like to congratulate all the participants about their good workshops. So about my thinking, we have here two problems to treat. The first one is the reflux of the venous saphena and the perforants, and the other thing is the big varicose ulcer. To treat the reflux, we have many methods. We can do invasive surgery by stripping, or noninvasive surgery, which means micro phlebectomy. The second is sclerotherapy, or echo-guided sclerotherapy. Third, we can make a compression bandage. Other, treatments include drugs such as Daflon, Detralex and Glivenol. So about the surgery, we have two things to do. First of all, the reflux of the saphenous vein. We can strip it and make a skin graft at the same time. As the ulcer margin I think, we must go with a knife below it because just to take it away. The case is very simple, but you must know how to do it. Within three months I can close the ulcer.

DR. ALLEN: I'd like to ask the panel if they would consider using growth factors in the treatment of this ulcer.

DR. RAJU: I have a brief comment. I think this patient will not come back to you if you delay definitive treatment too much. She has been living with this ulcer for 20 or 30 years, and has been totally noncompliant. I think these modalities that take a long time, will lose this patient. I think you should approach this patient quickly, do what you have to do, and if you want to fiddle around with long term modalities, do that after you have done the basic stuff.

DR. O'DONNELL: The rest of the panel, do you use growth factors?

DR. DEPALMA: Very controversial. I don't, no. I really don't understand their rationale. I don't think I'd use them here. You just need to fix this thing: i.e., the reflux and graft the ulcer.

DR. DALSING: Growth factors are very expensive. I think the only time you should consider their use is when you've tried everything else and have a chronic wound that won't heal. This lady really hasn't been treated appropriately with compression and standard dressings. So I probably would not use growth factor agents on this wound.

DR. MALOUF: I have a very small comment about the antibiotics. There is some difference of opinion about the use of antibiotics, but this is a relatively clean ulcer. The only antibiotics I would entertain using is the prophylactic antibiotics to preserve the skin grafts as the others suggested.

DR. VILLAVICENCIO: We use antibiotics in-patients with infected ulcers. The ulcers are always colonized, like in this particular case. I'm sure that when you take a swab you will grow

something. But when the ulcer is infected, it's painful, it's red, it's angry looking. In those individuals we have found that a course of IV antibiotics will quickly control the infection, and then you can go to further measures.

DR. BELCARO: Just an assumption. Every ulcer is infected. According to cultures, you will always find something there. So almost by definition as long as you have the skin open, there is an infection. So I think you should use antibiotic treatment any time you have an open ulceration.

UNIDENTIFIED SPEAKER: Can I come back on that? That's absolutely right. Even if you just deal with antibiotics, you're not going to get rid of all these parts. What you need to do, once you actually aim to heal it with skin graft, is just to actually protect your skin graft for the appropriate organism. It doesn't matter if you've got pseudomonas, staph, whatever. Skin grafts will take providing there is a good bed. What you need is to protect it from a hemolytic strep.

DR. PERRIN: I would propose that the patients would be assessed one month after surgery by a duplex scan in order to identify any perforating vein which was missed, or a collateral branch, in order to treat them because it is easy to miss something in this kind of patient. I think duplex is very helpful.

DR. TRIPATHI: Based on the experience with diabetic ulcers, what we in our center do with the venous ulcers is to take a superficial scrape and also use aspiration culture from the deeper layers of the venous ulcer. If the deeper layer culture is negative, then we go ahead and we scrape the superficial layer and put skin grafts in the OR with prophylactic antibiotics. I have never had a problem. I don't know if other people have experienced the same.

DR. PARSI: I'm interested in the role of microthrombi in the pathophysiology of this sort of ulcer. As you know, a lot of these patients have thrombophilia. Up to 26 percent of patients with ulcers have Activated Protein. Up to 40 percent have anticardiolipin C Resistance antibodies. Were these patients screened at all? Some of the thrombophilias like MTHR mutation with high homocysteine levels, can actually be treated with folic acid.

DR. O'DONNELL: That's a very good point. Certainly with deep venous reconstruction in our unit or in patients undergoing SEPS we would do a screening. What about this lady? Would most of you do a hematologic coagulation screen?

DR. KISTNER: No. This is pure primary reflux disease. I don't think she has any sign of thrombotic disease.

DR. DEPEDRO: A small comment. Permanent venous hypertension in this patient, which of the members of the panel think that this kind of ulceration is due to the insufficiency alone of the long saphenous vein and which think that this is due to the atrophy of the muscular pump?

DEPALMA: I think it's due to both factors, and I think she's absolutely right. There is wasting of the calf muscle. My idea in operating on it rapidly is to get it covered so that the patient can move the ankle around.

DR. RAJU: I think these massive ulcers are larger than what you think would be appropriate for isolate saphenous reflux. That seems to be somewhat more common in the older age group. We have seen a number of seventies and eighties with this kind of presentation that you don't see in the younger age group. So sclerosis of the deep veins and compliance changes might have something to do with it. I wonder whether anybody in the panel has seen the younger patient with this kind of isolated saphenous reflux with massive ulceration.

DR. KISTNER: The patient was 65 years old according to the history and had her first ulcer at 30 years. It brings up the question of where this patient has been in the 30 years. Has she been under any therapy at all or just totally neglected? The reason to raise this point is that there exists a mass of medical practitioners who have no understanding of this whole problem, and they would treat with a salve but never provide any compression. I wonder if this patient fits into that category.

SURGICAL MANAGEMENT

DR. EKLOF: This is an unusual case to show for saphenous vein incompetence. I took this case because I think it's a badly treated case for 30 years. The only alternative she was offered before was amputation of the leg, indicating how far we have to go to get widespread understanding of how to deal with sick patients like this. She had poor treatment until the most senior of surgeons in Hawaii, the first surgeon who got his American Board in Surgery sent her over to me, Lester Yee. He is now in his eighties and still practicing. I think he saved her at least from amputation. We admitted the patient, treated the infection, and cleaned up the ulcer. The reason we did the venograms was that I thought that this was more than just saphenous vein incompetence. It was such a longstanding ulceration. We couldn't find any perforators with a scanner under the ulcer. She had no perforators above the ulcer. We did high ligation and stripping of the GSV to just above the ulceration, and skin grafted the ulcer, which healed in about a week (Fig. 3). This was about three years ago. I tried many times to scan her, and I offered her to come to the hotel tomorrow to be part of the workshop, and scan her leg, but she could not make it. So I don't know more about the perforators in this patient. Pathology of the vein that we removed showed an arterialized vein due to the long standing turbulent reflux for many years.

II. PERFORATOR INCOMPETENCE

CASE OF PERFORATOR INCOMPETENCE - MAKING SEPS BETTER

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Perforator vein incompetence contributes to ulceration when abnormally elevated pressure is transmitted to the skin usually at the ankle medially. To correct this, a variety of surgical techniques have evolved; I suggest technical modifications of SubFascial Endoscopic Surgery (SEPS) to include extrafascial submalleolar perforator division and combinations of other interventions.

In 1966, Linton's approach to perforators was modified by eliminating longitudinal incisions, creating a series of bipedicle flaps in natural skin lines and avoiding areas of severe skin involvement. This procedure was performed by remote subcutaneous access

obliterating perforating veins from the crest of the tibia to the submalleolar region. Long-term results with this approach were reported in 1974.[1] No major wound infection or necrosis of flaps occurred. The ligation of inframalleolar and foot perforators as well as those in the calf was emphasized. The ulcer was dissected subfascially and perforators in the ulcer bed ligated directly and skin grafts applied in one operation. Using this approach, the recurrence rate for venous ulcers ranged from 9 to 10% in 168 limbs amongst 141 patients with observations extending 5 to 10 years in three series.[3,4,5] We later used a phlebotome passed subcutaneously in a line to interrupt the posterior venous arcade and Cockett's perforators.[6]

With the availability of Duplex scanning, and the realization that a more precisely focused approach could alter transmission of venous hypertension to the skin, the scene was set for less invasive procedures. At the same time, endoscopic techniques were developed resulting in specialized equipment. In 1985, Hauer reported an experience using endoscopic techniques to divide perforating veins in the subfascial space in the lower extremity.[7] Two retrospective series from Europe, Jugenheimer, et al 1992[8] and Pierik, et al 1995,[9] described their experience with SEPS. Variations of the technique were subsequently described in the literature.[10,11,12,13] In 1997 the North American SEPS Registry results were presented at the Society for Vascular Surgery, reporting on 148 patients from 17 different centers. Although the results as reported in the Registry, the largest study to date, continued to be encouraging, a disappointing 22% ulcer recurrence rate at 30 months,[15] suggested a need to re-evaluate not only indications for SEPS but technical modifications which might improve results.

Indications

This procedure is used for patients with severe CVI clinical disease, CEAP Class 4 to 6.[16] It is used most frequently in our practice for Class 5 or 6 patients, who, by definition, have active or immediately past ulceration. The procedure is employed for patients with previous deep venous thrombosis, valvular incompetence, or combined abnormalities, which may be corrected whenever practical. Patients with reflux tend to have better outcomes. Unless correctable, we consider caval occlusion to be a contraindication to SEPS as a sole procedure.

Preoperative evaluation

Duplex scanning includes examination of the greater saphenous veins and deep veins for obstruction and valvular incompetence, as well as identification of incompetent perforator veins. It is important that this be done in a standing position. We now recommend ascending and descending phlebography for all patients, in addition to duplex scanning. These procedures are best for finding areas of recanalized thrombosis or to locate incompetent perforators.[17] Phlebography is needed to plan combined operations such as valvular repair, inframalleolar perforator interruption, and iliac bypass for occlusive disease.

Surgical Technique

SEPS is a relatively new procedure and techniques vary somewhat. We no longer use a tourniquet or exsanguination and we use two port systems placed just below the knee. When two ports are used, incision placement is crucial to prevent dueling instruments. We use a balloon dissector[11] inserted through an initial transverse incision in the fascia with saline inflation to expand the balloon's width and length, thus creating an elongated bloodless working space. The balloon is then removed and replaced by an endoscopic probe which provides a constant flow of carbon dioxide to maintain expansion of the subfascial space. This port also has an endoscope and light source, which are introduced into the created space at this point. A working port is then placed under direct visualization. Perforating veins ranging in number from 6 to 8, involving the Cockett 2 and 3 complexes are divided through this working port.

When inframalleolar perforators are present, i.e. Cockett 1, which is located on average 7 cm above the calcaneous, we have used two approaches. A 14 gauge mini-port has been inserted and the subfascial ligation of this perforator is carried out. On the other hand, when an ulcer is present which requires skin grafting, the ulcer is excised and extrafascial technique is applied in the lower third of the leg and inferior to the malleolus to include the foot perforators.

Results

Twenty-six Class 5-6 patients have received operative interventions. All patients received Duplex scanning along with ascending and descending phlebography. Depending on the patterns of reflux or obstruction, differing interventions were used, including: superficial stripping 18; perforator interruptions 26: 12 extrafascial, 14 SEPS; valveplasty 2; and Palma cross over 2. Twenty three extremities remain healed at follow up, ranging from 15 to 50 months; three ulcers recurred, two healed promptly after a second operation either using SEPS or extrafascial interruption of the lowest Cockett or foot perforators. One recurrent ulceration persists; this patient declined further intervention.

Discussion

The largest study to date, the North American Subfascial Endoscopic Perforator Surgery Registry Report,[14] provided data from 17 medical centers including ours, between June 1993 and February 1996. The preliminary report looked at safety, feasibility and early efficacy of the procedure and was a retrospective analysis of 146 patients undergoing SEPS with different surgeons, medical centers, techniques and instrumentation. Mean follow up was 5.4 months and an ulcer healing rate of 88% was reported. There was evidence of more rapid ulcer healing as compared to non-operative treatment. No postoperative deaths or early thromboembolism occurred. Since that time, at our center, we have experienced a single episode of postoperative thromboembolism which was effectively treated with anticoagulation.

Clinical scores using CEAP grading have shown improvements ranging from 9.4 to 2.9 after surgery. A further study of intermediate results was published in 1999.[15] Although early results were encouraging, the 22% ulcer recurrence rate at 30 months was not. Recurrence is most common in CVI due to obstruction rather than valvular incompetence. I believe that in order to improve the results of SEPS, increased attention to submalleolar and foot perforators will be required. The subfascial space at the ankle at the malleolus is quite tight and difficult to access. While we have used SEPS approach with a separate 14 gauge port in the lower leg. I believe it is best to deal with these perforators using an extra-fascial approach.

These can be documented by ascending phlebography and can also be located immediately pre-operatively using a Doppler technique. As Kistner[17] recommended, one should not hesitate to use combinations of procedures beginning with the simplest as determined by preoperative duplex scanning, and ascending and descending phlebography. Varying interventions in the deep system might be needed; particularly caval or iliac obstructions which can be missed by limb duplex scanning. These respond poorly to perforator interruption. SEPS is a useful and elegant procedure. Results can probably be improved by additional interventions. Among these, extra-fascial ablation of the submalleolar perforators is advisable when these contribute to skin changes.

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THE VENOUS ULCER

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The venous ulcer is the most important epiphenomena occurring in the course of the Chronic Venous Insufficiency (C.V.I.) Syndrome. When present, its onset strongly affects the patient, who is unable to continue his normal activity. In our experience, from 444 cases followed up for 10 years, 50.67% presented an ulcer during this lapse. This percentage rose in posthrombotic syndromes (65.51%) and decreased in those patients who did not present deep venous pathology (46.25%).

As aforementioned, the venous ulcer is a further episode in the course of indurative hypodermitis of the C.V.I. Syndrome, and therefore its pathophysiology is closely related. This disease is a product of the permanent reflux towards the superficial system during muscular contractions, through the insufficient Direct Perforant Venous System.

In our series, patients were evaluated with phlebography and phlebomanometry. Among them, 56,53% had a history of previous DVT and 43.46% showed an intact DVS.

In 1876, Le Dentú described 2 types of perforant veins: direct & indirect. The latter connect the Superficial System with a muscular vein of the leg and thence, through it, run into the Deep Venous System. They do not participate much in the disease, as during muscular contraction, the point of maximal reflux, with pressures ranging from 200 to 300 mm Hg provoke the total collapse of this muscular vein, so avoiding the reflux or buffering its magnitude.

On the other hand, direct perforants are those that directly connect the Superficial System with the deep principal or axial veins, which latter are submitted to lower pressures - 100 to 15° mmHg - during muscular contraction; they do not collapse totally but partially, in the middle. Direct perforants emerge from the lateral partly open edge and, due to the Venturi effect, tend to suction the blood towards the interior of the axial vein.

Direct perforants of the thigh and upper third of the calf run into larger cross-section veins like the Long Saphenous Vein or collaterals of similar caliber. Because of this and once perforant valvular insufficiency is established, the reflux is rapidly neutralized. On the other hand, the direct perforants in the lower part of the leg open up in a fine superficial vein, the Leonardo's vein which is usually of smaller caliber than them. This provokes the important dilatation of these perforants, and the impossibility of the superficial vein to neutralize the reflux, which progressively will reach the capillary bed and finally cause the trophic changes at skin level. We have observed that this situation takes place within what we called "Venous Buffer Circuit", formed by the Long Saphenous Vein to the front, Leonardo's vein to the rear and the LAA at bottom, which links both closing the "superficial circuit" at the submalleolous level. The superior, medial, inferior Cockett perforants and the fourth, submalleolar, which we had described, constitute the participating perforant system. At the deep level, the Posterior Tibial and Internal Plantar Vein complete the circuit. This Venous Buffer Circuit, which in the first stages is able to compensate the reflux by its superficial constituents; when global insufficiency ensues, the circuit fails to compensate the reflux thus creating permanent stasis and retrograde hypertension which transmits to the skin and underlying tissues originating the cutaneous lesions.

When the Deep Venous System participates in the insufficiency either caused by obstructive, essential, posthrombotic reflux, or a combination of these expressions, does not contribute to modify the magnitude of the reflux but its duration, possibly limited by the caliber of the insufficient perforant at the peak of the reflux.

Conversely, and determined by the destruction of the valves, the duration of the reflux is prolonged, as the reflowing blood amount increases and the size of the intervalvular compartments augments. This fact is responsible for the high incidence of venous ulcer.

Microscopically, the zone is severely affected; capillaries look elongated, dilated and tortuous mainly near insufficient direct perforants. Thrombosis of the capillary vessels interferes in skin nutrition process thus predisposing to ulceration. This phenomenon also compromises the initial lymphatics, which play a role in this pathophysiological process.

Norman Browse has demonstrated abnormalities of interstitial fibrinogen in venous hypertension. Plasma fibrinogen level is elevated, and is related to an increase in the inhibitor PAI 1 from the endothelium of the vessels and the smooth muscle of the venous wall. After some time it is possible to observe a fibrin cuff within the papillary plexus of the skin, along with an inflammatory infiltrate basically composed of macrophages and T lymphocytes.

Another very important factor in the reduction of venous hypertension is the calf muscular-venous pump. When hypertrophied, it reduces the intra-muscular pressure in the standing position to less than its half, thus causing functional insufficiency. Ineffective contraction of the pump insufficient to evacuate the DVS, generates permanent hypertension which can start by itself the disease onset. Its maximal expression is the Phlebo-arthrosic Syndrome, characterized by fibrous sclerosis, which advances over tendons and joints, fixes the tibiae-tarsal joint, and impairs the function exerted by the muscle-venous pump thus obstructing the evacuation of the DVS. The result is gigantic ulcers, which tend to heal when the patient is put to rest, but reopen with prolonged standing position.

Not all venous ulcers have a similar behavior. Some of them are due to such a severe deep venous hypertension that collapses arterial capillary flow and originates necrotic foci in the meta-arteriole. These ulcers have an ischemic behavior, and are characteristically small, submalleolar, extremely painful and do not develop the consecutive stages of hypodermitis due to its sudden onset.

Physical Examination of the Ulcers

Debridement should precede physical examination so as to fully assess the characteristics of the ulcers. Localization, borders, bottom, depth, type of secretion, and persistence despite treatment are important points. The latter obliges to rule out malignization which although infrequent, may be present.

Lesions may be unique or multiple, and present variations in shape and size. Progressive growth without treatment may attain after several years the whole circumference of the leg. In the beginning, they present as superficial lesions, but as they increase in diameter, they also gain in depth. Examination of the bed of the wound is essential. When chronic, its appearance is indurative and rough, sometimes with sanious secretion and even presenting satellite adenopathies. During progression, involvement of deep bone and nervous structures may lead to short or long saphenous nerves compromise depending on the localization of the lesion. This produces acute pain which is usually less intense than that of ischemic origin. Superimposed infection may cause reticular or trunk lymphangitis which worsen the outcome.

- **Peri-Ulcer** tissue is primarily affected with capillarytis, and/or streptococcal dermytis. During its evolution, different modalities may be present:
- **Eczematous** with inflammatory features and scabby surface.
- **Paracheratosic** with fine or gross desquamation adopts a psori asiform aspect.
- **Purulent** exudates.
- **Combinated Forms**.

Depth and extension of the ulcer are very important characteristics to assess the response to medical treatment. Healing is fast when only epidermis and superficial dermis layers are affected, while it is slower when the lesion compromises glandular culs-de-sac: if the ulcer extends to the deep cellular tissues the destruction of generative layers conditions the growth only from the lesion border. In these cases, evolution is relented in proportion to the ulcer size.

Treatment

Venous Ulcer treatment must complete two phases:
1. Ulcer Healing
2. Cure of the disease.

It is essential to treat infection as well as measures to control edema to achieve ulcer healing. Systemic Antibiotic therapy preceded by culture and sensitivity studies, especially when long term duration of treatment is considered very important. Skin care is of major importance to the end of avoidance of superimposed erysipelas or other infections, which may compromise the local lymphatic system.

Edema will be controlled mainly by means of Manual Lymphatic Drainage and adequate elastic compression hosiery, which will in turn increase venous return velocity and provide an external support to the Superficial System. Locally, within the ulcer, repeated saline or non-ionic fluid wash is recommended. When necrotic areas are present, surgical debridement must be considered.

A good granulation tissue to facilitate the ulcer healing can be obtained by applying the most various types of dressings in the shape of gel, colloids, films, tissues or foams according to determined needs (debridement, epitelization, granulation, etc.). When the ulcer size is important, healing must be eased by free skin grafts, collagen implants polyurethane dressings or biosynthetic membranes. Sclerotherapy in the preoperative period has seldom or no indication. Conversely, postoperatively it may help to maintain a good result with time.

Surgical Treatment

Once venous ulcer has been healed, it is necessary to try to cure the CHRONIC VENOUS INSUFFICIENCY disease, by means of restoring normal physiology. This is achieved by surgery, which will be permanently successful if radical and complete. It must be directed to treat all affected territories (superficial, perforant & deep if possible). Long term postoperative follow up is also essential, as future decompensations can be detected and recurrence avoided with minimal post-surgical complements. Before deciding the operation, one must keep in mind the assessment of the calf venous-muscular pump. In case of atrophy, it should be recovered to reduce edema to its minimal expression. If persistent, edema will allow clinical evaluation of the role of deep venous hypertension and its role in hydro-electrolytic retention. Quantitative Duplex Scan, APG, Phlebomanometry, and Ascending Phlebography will determine the magnitude of the lesion of the Deep Venous System and the potential repair possibilities.

Superficial Venous System

When a Long or Short Saphenous Vein is incompetent, total or partial stripping according to the magnitude of the insufficiency is the most adequate surgical measure over all the conservative techniques and compression sclerotherapy given the high incidence of

recurrence of these two latter procedures. Second and Third order collaterals will be excised by phlebectomy or may be complemented by sclerotherapy.

The Perforant Venous System

When the DVS is normal, direct perforants responsible for the decompensation of the Venous Buffer Circuit must be ligated. Whether the DVS is also affected, complete interruption of perforants is mandatory with ligation of normal perforants as well. With time, these vessels are prone to decompensation and the disease will recur. Nowadays the perforant system can be approached by:

1. Through Open Surgery (modified Linton or Cockett operations).
2. Subfascial Videoscopic Surgery.
3. Echo-assisted Surgery.

1. Open Surgery

Despite a traumatic operation, with a large incision, this technique provides radical benefits when an abnormal DVS Syndrome is present, as it is the only one that permits the ligation of normal perforants thus avoiding long term recurrence. Videoscopic subfascial surgery and echo-assisted procedures have a higher percentage of post-operative recurrence in these cases.

Linton & Cockett operations must be complemented by the sub-malleolous resection of the Lower Anastomotic Arc, the retro-malleolar perforants described by Van Limborg, and the fourth perforant. We do not perform the Superficial Femoris vein ligation (Linton), and avoid the incision on diseased tissues. Subcutaneous tissues section should be done in block so as to facilitate healing. Finally, suture of the fascia must be done to preserve the function of the calf muscular-venous pump.

When this modification is carried out, practically as high as a 14.6% of postoperative recurrence in this localization is eliminated.

2. Videoscopic Surgery

As aforementioned its main indication remains the interruption of the perforant system with normal DVS. Access to gastrocnemious and solear sector is good, as it is to the upper direct & indirect perforants of the leg when not localized within the muscular zone of insertion in the tibial medial border or the superior and medial Cockett perforants. This technique definitely does not allow the approach to the 3rd, la 4th, & the Lower Anastomotic Arc and thus this operation must be completed by open surgery at this level.

Its main advantage is the short size of the incision (3 cm.), which carries a better cosmetic result and a swifter postoperative recovery.

3. Echo-assisted Surgery

This operation consists of perforants ligation performed through 0.5 to 1 cm. incisions, previously marked by duplex scan. If necessary, this diagnostic method may be also used intraoperatively. Similarly to the former, it is not useful in the post-thrombotic syndrome, given that the Duplex does not detect normal perforants. Its main advantage upon Videoscopic Surgery is that the subfascial compartment remains unopened, thus leaving it untouched for a future open operation. Besides, it allows a more complete dissection of superficial vessels collateral to the perforants. It is advisable to close the fascial perforant ring with a stitch after it has been sectioned, to avoid further diagnostic confusion in the postoperative period. Thumb fingertip palpation through the incision of the fascia allows also the detection of smaller caliber perforants. The operation must be completed if necessary with the sub-malleolous approach. The cosmetic result is very good, if intradermal suture of the incisions is carried out.

The Deep Venous System

There is a group of patients with lesion of the DVS, in which venous hypertension continues to act upon the interstitial tissues of the leg, despite restoration of functional independence between the DVS and the Superficial System. In these cases, normal pressure levels must be achieved, to allow the patient to lead a normal lifestyle.

We must keep in mind the following items:

- It is essential to the evolution to count with a fully developed muscular-venous pump.
- Clinical examination is crucial for decision making, especially in the young patient with severe manifestations.
- These features are important:
  a. Venous Claudication, which is similar to that of the arterial patient.
  b. Venous ulcer with an ischemic behavior.

Operation must be considered as soon as possible, mostly if the patient is young, to achieve the best results in terms of prompt recovery without previously damaged connective tissues and skin. Duplex scanning, either qualitative or quantitative, and APG will be able to measure the magnitude of reflux and presume surgical chances. Once intervention is decided, ascending and retrograde phlebography are indispensable not only to assess the reflux or the obstruction quantification, but also to appreciate the potential resources for the reparation. Despite the fact that surgical procedures per-formed on the DVS exceptionally bring a physiologically complete restitution, partial results are usually compatible enough for the patient to lead a normal life.

Valvular insufficiency has an excellent surgical solution by means of valvuloplasties, original techniques developed by Dr. Kistner, which can be carried out either externally or internally; when not feasible, transpositions to the vertical branch of the Profunda Femoris from the Superficial Femoral constitute a very good alternative.

Venous valvulated bypasses with preserved veins allografts are in a developmental stage, with encouraging possibilities.

Palma Operation (1958) is indicated in obstruction or subocclusion of the Iliac and/or high Common Femoral segments, when the organism mechanisms are not enough to compensate venous hypertension, thus expressing by severe symptoms and signs at the level of the leg. Despite this compensatory mechanisms are directed towards the support of other natural bridges developed to overcome the obstructive problem, they are usually not enough. In these cases, patency maintains permanently and the patient experiences a substantial progress. Many other operations on the DVS have been relegated due to their scarce success in the long-term follow-up. In the Phlebo-Arthrosic Syndrome, the surgical consideration remains depending to the recovery of the tibio-tarsal joint mobilization, which rigidity is a characteristic of this Syndrome. Once mobilization is achieved through cinesitherapy, the venous affected systems will be then considered to be treated according to what was previously expressed.

ULTRASOUND GUIDED SCLEROTHERAPY (USGS) FOR PERFORATING VEINS (PV)

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The rationale for treating varices and perforating veins is aimed at the three main pathophysiological abnormalities: to reduce venous hypertension, to suppress leak points from the deep to the superficial venous system, and to reduce the varicose volume/length (reservoir).

When to treat specifically perforating veins?

To suppress a varicose pattern that is mainly/solely fed by the PV, to avoid recurrent (persistent) varices after surgery, to heal an ulcer which is not cured by (appropriate) compression alone.

Why not to treat PV (with USGS)?

As an isolated procedure, USGS (like other techniques used for treating PV), does not seem appropriate in case of deep venous obstruction.[1] It has been observed recently that thrombophilia is often associated with thrombotic complications of sclerotherapy,[2] therefore a precise clinical and biologic screening of patients must be defined prior to any treatment of varicose veins. A history of venous thrombosis must be searched for.

When not to treat perforating veins (with USGS)?

The first obvious reason is that when they are competent or too small (<3mm diameter)[3] there is no need for suppressing them.

When they drain a varicose cluster (sometimes called "reentry"), there is a theoretical risk of worsening of the venous hypertension.

For medial leg perforators, when they are associated to an incompetent greater saphenous vein (GSV), 75% of them will become competent after removal of GSV.[4,5]

In all cases, a complete duplex assessment of the venous networks must be carried out before treatment, with the patient standing or sitting for this examination. Reflux duration of > 0.5 sec. indicates incompetence.[6]

Indications for sclerotherapy of PV:

As indicated above, most PV will become competent after stripping of the GSV. Therefore, when not all PV are removed at the operation time, additional sclerotherapy will take care of residual incompetent PV.

Sclerotherapy as a primary treatment of PV is feasible for example to deal with the Dodd & Hunterian perforators, non saphenous networks (for example on the lateral aspect of thigh; Albanese network), incompetent medial leg PV without GSV incompetence, Recurrent Varices After Surgery (REVAS) related to certain cases of PV incompetence (femoral canal for example), recurrent varices after prior USGS or Sclerotherapy. Sclerotherapy of PV in patients with history of DVT is not a routine treatment, but can help to heal venous ulcers.

Criteria for the choice of treatment (USGS vs. SEPS or vs. stab avulsion):

Diameter and duration of reflux should be considered as criteria for decision although there are no data to support this opinion. Veins with a diameter of more than 8 mm are more likely to be resistant to sclerotherapy. Patients should be more enthusiastic for sclerotherapy since the method is ambulatory, cheap and simple.

Techniques of injections:

The usual "blind" sclerotherapy is sometimes possible if the vein has been marked by duplex imaging (if the duplex is not easily available). However, ultrasound guided sclerotherapy provides more safety and accuracy.[7] Duplex will also provide information on the good evolution of the sclerosing reaction on the further examinations (1 week or more).

The sclerosing agents which can be used are Sotradecol 3% or Polidocanol 3%, lower concentrations are advised at the first session if the PV are smaller than 4 mm in diameter, an initial volume of 1 cc at the first session is recommended. Compression of the leg with bandages or medical stockings is mandatory.

There is a lack of evidence regarding the results of the technique, further studies must be carried out.

Based on one short-term study[8] and on our own experience, we estimate that a primary occlusion is obtained in about 90 % of cases with 3 or less sessions. Regarding long-term results, no controlled study and no data on recurrence rate are available.

Criteria of assessment for future studies should include: duplex-visible sclerosis of the vein, pre and post therapeutic diameter and reflux duration, and plethysmographic evaluations as well.

The convenience of the technique and its overall price (calculated on a long-term follow-up, estimated with a life-long treatment) must be taken in account.[9]

Some of the potential complications of the techniques are non-specific to sclerotherapy of PV, such as thrombosis, necrosis and allergy. Edema and bulge of lipodermatosclerosis are more specific. These latter complications are more frequently observed in patients graded C3 and higher. They are decreased by an adequate compression.

Pros & Cons of sclerotherapy of PV represent a good summary of the method:

Pros: Cheap, repeatable, painless and versatile.
Cons: Technically challenging, possible complications, no data on long term results.

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CASE OF PERFORATOR INCOMPETENCE

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This is a 65-year old woman with severe recurrent venous ulcerations of the left and right legs since May 1989. She has a history of bilateral DVT 20 years ago. The patient underwent left greater saphenous vein stripping in 1978 and 1985. She has normal pedal pulses.

Level 1: There is a 10x4 cm superficial ulceration above the left medial malleolus with moderate surrounding lipodermatosclerosis. No remarkable edema.

Level 2: Duplex scanning of the left leg (May 1989) showed absence of the greater saphenous vein. The common femoral and profunda femoris veins were patent and competent. There was partial recanalization of the superficial femoral and popliteal veins. The posterior tibial vein was also recanalized. No perforating veins were identified. Duplex scan Sep 1997: left superficial femoral and popliteal veins patent but partially compressible, posterior tibial vein incompetent, lesser saphenous vein incompetent, two incompetent perforators medial calf. APG Sep 1997: OF 16%, VV 68 ml, VFI 5.1 ml/sec, EF 65%, RVF 44.1%.

Level 3: Descending venography reveals valvular incompetence in the common femoral and proximal superficial femoral veins; contrast flows retrograde to the level of mid-superficial femoral vein. Lymphoscintigraphy: no abnormality left lower extremity.

Duplex Mar 1998: no change from Sep 1997. Three incompetent perforating veins medial calf.

CEAP Classification: C6s; Es; As,p,d; Po

Treatment?
(see figures 1-2, on p. 264)

DISCUSSION

DR. O'DONNELL: I do have a little problem with the use of the eponym "Cockett" for these operations. Cockett, as you know, originally described an extrafascial approach to perforators and reserved the subfascial approach for severe dense lipodermatosclerosis with ulcer. Actually, Dodd, Cockett's co-author of their classic text, abandoned the extrafascial approach very early on his experience because of wound complications. In addition he moved the incision postero-laterally. So what you call Cockett is not what Cockett himself described.

DR. O'DONNELL: This is a very interesting case, certainly not one of straightforward perforator incompetence in that there seems to be an element of deep venous obstruction. Our panel had very interesting responses. I question you, gentlemen and ladies, can you provide any evidence that doing something to the perforating veins is going to make this patient better? I would submit that no one in the audience can show in a case like this that the hemodynamics improve. Indeed, most of the data in the literature shows no hemodynamic improvement in patients with post-thrombotic syndrome following interruption of the perforators. Going back to some of the early studies by our Scandinavian colleagues - occlusion of a perforating vein and measurements with electric magnetic flow meters and venous pressures showed no improvement in hemodynamics. And our own work confirms the same. Therefore, I find it very interesting in this case that we're going to treat the perforators alone, but I don't know to what end. Let me open it up to the panel after these "prejudicial" statements. Peter, from your North American SEPS Registry study you have a one in two chance at least with a short-term follow-up of having a satisfactory result i.e., -no ulcer recurrence, in this case if you interrupt the perforators; right?

DR. GLOVICZKI: Well, this is a difficult case, and I seldom perform perforator ligation in a patient with deep vein obstruction or with an element of deep vein obstruction. In this patient obstruction has been confirmed by APG studies. Unfortunately, we do not have an adequate evaluation of this patient. Ultimately, I think that I am going to suggest SEPS, but I would probably make another attempt of an ascending venography. I think an ascending venography in this patient would be quite critical.

DR. O'DONNELL: Why don't you show the ascending phlebogram that you did do, Paul - after the procedure?

DR. GLOVICZKI: You should have done the ascending venogram before the procedure.

DR. DEPALMA: One question that I missed completely is the status of the lesser saphenous.

DR. CORDTS: The lesser saphenous vein was incompetent

DR. DEPALMA: It was incompetent. Okay. That's important because the lesser saphenous gives an Achillean perforator as it crosses the tendon initially to Cockett 1. That is what Dr. Enrici's arcade shows as he dissects. I think that it's very important to deal with that inflow problem as well as interrupting perforators from above.

DR. O'DONNELL: Ralph, how do you deal with the incompetent lesser saphenous? Do you strip it out? Do you ligate it? What do you do?

DR. DEPALMA: Well, I think all of the action is down at the lower end, and I would divide it. I would ligate it and then just put the small skin incision out of the area of involvement and then come down directly on the Cockett 1, ligate that, remove the Achillean communication. Then I elevate the skin around it and then dress the dissected area firmly and keep the limb elevated.

DR. GLOVICZKI: I like invagination stripping of the lesser saphenous vein. I think it is nontraumatic and it preserves the sural nerve. These are frequently perforator veins connecting the lesser saphenous vein to the deep veins, so stripping is a better operation than ligation only.

DR. NEGLEN: I would like to turn this case around. If I understood it right, you had axial reflux in the superficial femoral vein that was patent and partially recanalized. So if we forget the perforators and then look at axial reflux in the deep system in a limb with stripping of the saphenous vein already performed, reflux flow

on the APG as high as 5.1 ml/s is sort of high for perforator incompetence, even with the popliteal vein incompetence. That's the first point. The other point is that I would also be very interested in looking at the iliac vein and agree with Dr. DePalma, since the outflow fraction is only 16 percent. Although I don't trust the measurement of outflow fraction per se, 16 percent is very low and probably positive for obstruction. So I would say, isn't this a case of axial deep vein reflux with outflow obstruction, although I don't know where the obstruction is?

DR. CORDTS: The obstructive changes were in the superficial femoral vein and popliteal veins. The iliac veins looked normal by venography.

DR. NEGLEN: Completely normal by venography? Then I would suggest you perform a valvuloplasty of some sort of the superficial femoral vein, which would probably lead to a better result than a SEPS.

DR. O'DONNELL: What would the panel say to that? That would be my conclusion, but I'm glad you stated it. Treating the perforators alone at least hemodynamically does nothing.

DR. DALSING: In this case, I'm more concerned that the obstruction is important. Even if you couldn't obtain an ascending venogram, I think something like a magnetic resonance venogram, may be possible. This would allow you to look at the anatomy in some way. I am worried about the obstruction. I'm not so sure that putting a valve in this system to prevent reflux is going to take care of an APG of 16 percent. I don't think I've even seen a false positive APG to that degree. Yes, I have seen false negatives, but not a false positive. These are the things that I'm concerned about when considering this case.

DR. KISTNER: I thought you said that the reflux only went to the lower thigh and not down into the popliteal. Am I wrong?

DR. CORDTS: Reflux to the lower thigh on the descending venogram but then on duplex scan subsequent to that it went down more distal than that into the popliteal vein and the lesser saphenous vein.

DR. KISTNER: And how did it get there?

DR. CORDTS: It got there through the superficial femoral veins. Those studies were done at different times.

DR. DALSING: Was that an obstruction or a valve present there?

DR. CORDTS: An obstruction in the superficial femoral vein.

DR. KISTNER: Did you consider this a problem of reflux or a problem of obstruction? Could you separate those, or was it both?

DR. CORDTS: Initially obstruction and later reflux, later both.

DR. KISTNER: I don't see repairing the reflux unless you could demonstrate that it has significant volume.

DR. NEGLEN: Bob, is it occlusion and is it recanalization and to what degree do we have the lumen? I can understand the hesitancy of doing a valvuloplasty above an occlusion, but we'd really like to see those venograms.

DR. KISTNER: If there's significant reflux it should be eliminated. It could either be by putting a valve or by ligating the SFV.

DR. DEPALMA: If I can make a comment here, 30 cc's of dye is worth three opinions. I'm used to looking at arteriograms and venograms. I'm not so smart in guessing at ultrasound or physical examination. We've had this discussion about the use of duplex scans to do operations, and in the recalcitrant group of patients the venous system is pretty complicated. Here's Sherman's depiction of the saphenous and branches published in the 1950's. We tend to forget how complicated the superficial system is and how many branches there are. If you can figure that out on duplex, especially when the skin is thick and a big ulcer exists, I don't know how do it. I think this is the usual end result of surgery rather than conservative therapy. I have had a problem correlating our duplex scans, which are wrong about 30 percent of the time, in making operative decisions. This case is a perfect example of that. In this case the surgeon has left the saphenous behind along with missed perforators. I repeat again that 30 cc's of dye is always worth three opinions.

DR. O'DONNELL: I would agree that when you get a complex venous case that duplex alone is insufficient. It's our routine to get ascending and descending phlebograms, particularly in a patient with an ulcer.

DR. KISTNER: Certainly I'd have ascending and descending venography to map out everything in the leg. It looks like there's a good chance that the superficial femoral vein is contributing reflux, and I'd eliminate that, probably repair it, and if I couldn't repair it I'd probably ligate it. I'm not clear what's going on in the profunda femoral vein, and I think you need a descending venogram to find out what the flow patterns are. So often you find a different flow pattern with descending venography than you do with ascending venography, and if you add the two together and throw in the duplex, you get a picture. So I would analyze this case more completely, and fix what I could fix. I guess that reflux is the key more than obstruction.

DR. RAJU: I would totally agree with what Bob said. I mean, it's clear that the superficial femoral vein is ten times as large as any perforator we saw, and I think it would be a mistake to focus on the perforators. Dr. Gloviczki has been presenting data in the last few days in this very meeting saying that one-year recurrence is about 35 or 30 percent, in post-thrombotic syndrome is it not, Peter?

DR. GLOVICZKI: The two-year data was 46 percent, but that had a large percent of standard error because we didn't follow too many patients up to 2 years. Post-thrombotic patients do the worst.

DR. RAJU : So you have already done SEPS.

DR. GLOVICZKI: You have not. You did a sham operation.

DR. RAJU: It has already failed, and you have shown persistence, or new formation, of perforators. You saw some other unnamed saphenous branch taking in a perforator up in the thigh. I think it has been shown in the venous system that you cannot disconnect and isolate the superficial or some part of the venous system from the other permanently. This has been amply shown in the portal circulation. Warren operation is based on that. It works for four or five years. After that you get reconnection. That should be the time for SEPS. I think all the disconnecting operations are going to be temporary. Preliminary SEPS data shows the recurrence to be high in post-thrombotic cases. The superficial femoral vein is large in this case. There is massive reflux, and I do not understand the hesitancy to go and fix that reflux.

DR. O'DONNELL: Peter, would you comment? You said a sham operation. Is that because the lamina profunda was not incised in the posterior compartment?

DR. GLOVICZKI: I was joking. It was not a sham operation. It was just not a complete operation and that was obvious. In such a short time those large perforators don't just show up. I mean, obviously if the deep posterior compartment was not entered,

several important perforators were missed. So it was an incomplete operation. This patient has severe post-thrombotic reflux and obstruction. I would start out with a redo SEPS, but I don't debate that this patient will ultimately really benefit from a good operation to correct the deep reflux. The debate could be whether it's femoral ligation as Bob suggests, or something else. The question is if the valve is not reparable would you do an axillary vein valve transplant or would you, in a patient like this, put in a cryopreserved vein valve that has a 60 percent thrombosis at six months.

DR. PERRIN: I think the ulcers recurred after SEPS. I would propose ultra-sound guided technique for the perforators. That's the first thing. If that does not work, I would probably redo SEPS.

DR. O'DONNELL: You wouldn't treat the deep system?

DR. PERRIN: No.

DR. DALSING: I guess I would approach the deep system and try to repair it, like Bob says. The actual six-month patency rate for cryopreserved vein valves is probably in the 60 percent range, not the 40 percent patency rate suggested by Peter.

DR. KISTNER: If that profunda system is patent and competent, it's a different ballgame than if it's diseased or absent. So you've got to find that out because that tells you whether it's worthwhile to fix the superficial femoral system, I think.

DR. NEGLEN: I think this is very important what Bob said. We still don't know enough about the axial reflux. Seeing the films I got a feeling there is a sort of profunda transformation and maybe it wasn't the main superficial femoral vein we saw. The second point I want to return to is the low outflow fraction and the suspicion of outflow obstruction. I think this patient needs a trans femoral venogram which clearly shows the iliac segment. This segment can't be assessed in this film although it appears normal. I agree with Dr. Dalsing that 16 percent outflow fraction is very low and it's rare to have false positive findings of that magnitude. There is something cooking up there.

DR. DALSING: When you did your descending venogram did they look at the iliac when they went down?

DR. CORDTS: Yes.

DR. DALSING: And was it normal then?

DR. CORDTS: Yes.

DR. O'DONNELL: I personally would get a complete venogram and do an arm-foot vein pressure study to determine the elements of obstruction.

SURGICAL MANAGEMENT

DR. CORDTS: Let me show you what we did first. In March of '98 we did a SEPS. We did a SEPS using the standard techniques that have been described. We identified three incompetent perforating veins by duplex scan and marked them pre-operatively. Then we identified those, clipped them and divided them. We did not open the deep posterior space. Then we exposed the lesser saphenous vein at the saphenopopliteal junction and tried to but couldn't pass the PIN stripper. So we treated the lesser saphenous vein by ligation and division since we could not strip the lesser saphenous vein. Over the next few months she showed improvement but three small venous ulcers remained. In August 1999, a year and a half later, the ulcers had never healed. We did a duplex scan which showed the common femoral vein and popliteal vein were incompetent. The tibial veins

Figure 1.– Ascending venogram left calf, oblique view. Venogram demonstrates two residual incompetent perforating veins medial calf (white arrows). Clips from prior SEPS procedures are noted in more distal calf.

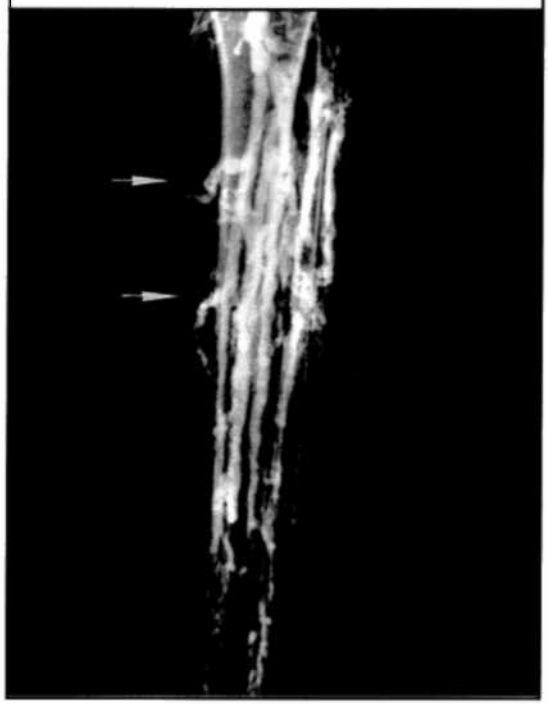
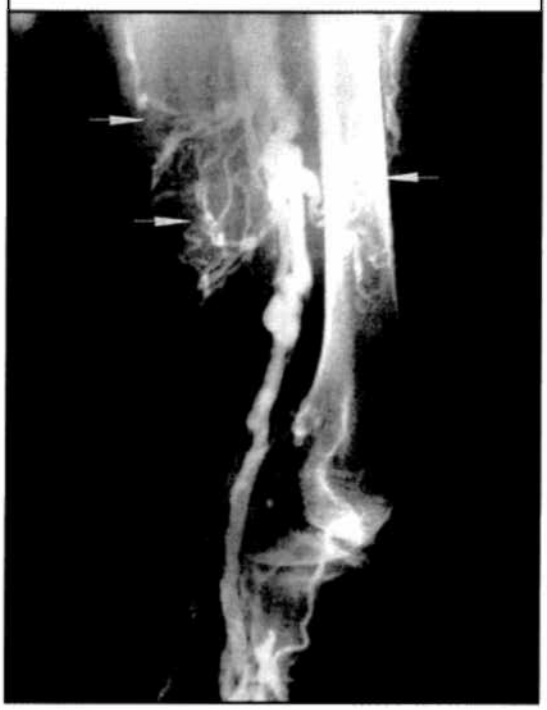


Figure 2.– Ascending venogram left leg, lateral view. Venogram demonstrates multiple incompetent thigh perforating veins (white arrows) filling superficial varicosities. Post-thrombotic changes of distal superficial femoral vein are noted.



appeared competent, and we identified three incompetent perforating veins. This is a year and a half after SEPS. We then did an ascending venogram. We were able to identify three incompetent perforators (Fig. 1). You can see the clips from the SEPS, and you can see that one of those perforators communicates with an incompetent segment of the greater saphenous vein below the knee. So that's not good. There were recanalization changes of the superficial femoral vein (Fig. 2), and an incompetent perforator in the thigh are seen. Then as you come up in the groin here we identify some portion of the profunda, and the remainder of the iliac veins appear to be normal. So we identified three incompetent perforators in the calf, at least one incompetent perforator in the thigh, and recanalization changes of the superficial femoral vein. We haven't done anything further at this point.

III. PRIMARY VENOUS DISEASE: DEEP AND SUPERFICIAL REFLUX

TREATMENT OF PRIMARY VENOUS INSUFFICIENCY

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Recognition that a chronic venous problem is due solely to primary venous insufficiency (PVI) defines that the entire problem is due to reflux in the veins, and that obstruction is absent. PVI cases can present with exactly the same clinical appearance as post-thrombotic cases, but the treatment implications are quite different because the venous system is entirely patent and the luminal surfaces of the veins are normal. Successful treatment is possible by surgical means in all segments of the lower extremity veins afflicted with PVI, including superficial (saphenous), perforator, and deep veins, and the results in all of these divisions of the venous tree are highly favorable.

PVI has distinctive histologic findings that have been published in the past but are not frequently appreciated. The findings in the endothelial layer consist of hyperplasia, which produces the white streaks often seen on the luminal surface of the opened vein. The real pathology is in the subendothelial and medial layers where the most striking change is an increase of the collagen which appears to become aggressive by wrapping around muscle bundles and actually fragmenting the syncitial continuity of the muscles in PVI. This is accompanied by fragmentation of elastic layers and areas of either hyper- or hypoplasia of the muscular layers. This process logically results in dilation of the venous wall, and dilation of the wall leads to valve incompetence.

The striking difference between primary and secondary disease is that the signs of prior acute thrombosis and inflammation or hemorrhage seen in post-thrombotic disease are absent in primary disease. These signs are hemosiderin deposition, neovascularization in the old thrombus and in the vein wall, and leukocyte infiltration of the wall.

The gross changes of PVI are strikingly different than those found in post-thrombotic disease (PTD). In PVI, the lumen is smooth and the wall is pliant and of relatively normal thickness. Valve sites are fewer in the saphenous vein of PVI than in the normal state, probably due to atrophy and ultimate disappearance. All stages of atrophy of valve cusps can be seen in these veins. In the deep veins, the valve cusps are normal in appearance, but are stretched and elongated. These findings are strikingly different than in post-thrombotic veins where the luminal surface is irregular, contains synechiae and random webs, and sometimes endoluminal masses are present. The valves are disfigured, scarred, and often entirely destroyed. The wall is thickened, non-pliant, and there is usually a peri-phlebitis with adhesions to surrounding tissues.

Given these differences, it is not surprising that there are excellent opportunities for surgical repair in PVI and little reason for limiting treatment to external support and change of life-style in the otherwise healthy person. With care in diagnosis, and adherence to the CEAP requirements for definition of the etiologic basis for the clinical problem between primary, secondary, and congenital causes, and the pathogenetic mechanisms of reflux and obstruction segment by segment, opportunities for correction of the abnormal physiology abound in primary disease.

Treatment of superficial primary disease in the saphenous system, and of the perforator veins, is widely practiced and is all that is needed in 30-50% of the cases of ulceration, and in the vast majority of non-ulcer cases. The deep system is implicated in 60%+ of primary ulcer cases, and requires surgical repair in a so-far unknown percentage of these to provide long-term relief of the Class 4, 5, and 6 problems. The long-term success of valve repair, coupled with control of saphenous and perforator incompetence in PVI cases of classes 4, 5, and 6 up to 4 years and beyond, is well-demonstrated in the literature to fall in the range of 65-80% in every published series of significant size.

Given the present ability to diagnose primary venous disease accurately and by non-invasive affordable tests, the appropriate management of all primary disease should be by surgical correction in the active patient. This includes saphenous, perforator, and deep vein correction. The major question is to define which patients do, and which patients do not, require correction in the deep veins to provide a long-lasting favorable result. The answer to this will require comparative prospective treatment groups.

(Scientific Articles continue on next page)

THE ROLE OF ANGIOSCOPIC VALVE REPAIR FOR PRIMARY VALVE INCOMPETENCE (PVI)

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The treatment of deep venous valvular reflux for advanced chronic venous insufficiency (CVI) has been cyclical in nature. During the late 1960s and early 1970s there was enthusiasm for ablation of the superficial venous system – saphenous and its branches as well as perforating veins, as the sole management for venous ulcer with deep venous reflux.[1] Both the ulcer recurrence rate and wound morbidity associated with this approach dampened interest in the treatment of venous ulcer by superficial venous system ablation alone.[2] Direct deep venous reconstruction pioneered by Kistner[3] sparked interest in the later approach. Recently, minimally invasive surgery for treatment of incompetent perforating veins (SEPS) however, has refocused the treatment strategy for grade IV through VI CVI back on superficial ablation rather than deep venous reconstruction due to the low wound morbidity rate of the former. The important work of Gloviczki and his North American SEPS Registry has shown that superficial venous ablation works better in patients with primary valvular incompetence than those with a post-thrombotic etiology for their deep venous reflux.[4] This data suggests that a staged approach to deep venous reflux due to primary valvular incompetence is the appropriate strategy. Although primary valvular incompetence has traditionally been related to a fibro-elastic degeneration of the valve with resultant valvular insufficiency,[3] there is increasing evidence that volume overload from a capacious varicose superficial system can cause dilation of the deep venous system and secondary deep venous valvular incompetence.[5] The valves in this situation are intact, but the dilation of the venous annulus prevents them from appropriately coapting. Superficial system surgery which removes the volume overload has shown to restore both normal venous diameter and valve competence. It has been our approach to treat the incompetent superficial venous system first in patients with primary valvular incompetence and restrict direct deep venous reconstruction to those patients who fail this therapy.

One of the hosts of this conference, Dr. Robert Kistner, was the first surgeon to describe a direct approach to rendering a valve competent rather than replacing the incompetent segment with competent valve "borrowed" from either a local or distant venous segment.[3] His initial report in 1968 stimulated interest in the surgical repair of primary valvular incompetence.[6] Our case report will detail the diagnosis, surgical technique and postoperative results of surgery for primary valvular incompetence.

Diagnostic Methods

Clinical Examination: Patients with deep venous valvular reflux characteristically have pain, which is described as a heaviness rather than the intense bursting pain experienced by patients with deep venous obstruction.[7] The pain or heaviness develops upon rising from bed and worsens after prolonged standing. Calf heaviness occurs irrespective of whether the patient is walking which is in distinct contrast to the situation with obstructive venous disease and venous claudication. Edema is a constant finding in patients with deep venous reflux and is of a mild to moderate degree. Cutaneous sequelae, lipodermatosclerosis and pigmentary changes occur frequently in these patients in association with incompetent perforating veins. In our experience, the skin changes may not be as severe as those encountered in patients with post-thrombotic syndrome.

Noninvasive Assessment of Venous Reflux: Duplex scanning is our preferred diagnostic study, because it answers several questions: 1) What is the pathologic process in the deep venous system, 2) what levels are involved, and 3) who are surgical candidates based on the degree of reflux? Quantitative evaluation of venous valvular reflux is performed by the technique described by van Bemmelen. While color flow analysis provides qualitative determination of deep venous valvular reflux, spectral analysis is recorded to quantify the degree of reflux by duration.

Air plethysmography provides hemodynamic information on deep venous valvular incompetence. The venous filling index (VFI) relates directly to the degree of venous reflux and is independent of the venous volume reservoir. Christopolous[8] as well as our vascular laboratory[9] have shown that patients with popliteal vein reflux in stage IV – CVI have VFIs in the range from 7 – 28 ml/seconds versus the normal 2 ml/seconds. We per-form both of these noninvasive studies prior to consideration for valve reconstruction as well as employing it as a method for documenting hemodynamic results postoperatively. In a prospective trial comparing quantitative duplex scanning and air plethysmography to the gold standard of descending phlebography in patients with stage V/VI chronic venous insufficiency, we demonstrated that the combination of valve closure times at the superficial femoral and popliteal vein levels accurately discriminated mild from severe reflux with a sensitivity of 90% and a specificity of 94%.[9]

Phlebography: In all surgical candidates, ascending phlebography is performed with multiple tourniquets to maximize visualization of the deep system.[7] With the superficial system occluded the contrast material is injected by hand forcefully into a foot vein. In addition to indicating the presence and level of incompetent perforating vein, valve sites may be seen in the superficial femoral vein. The diameter of the veins helps distinguish relative deep venous valvular insufficiency from true PVI.

Descending phlebography is then performed under fluoroscopy with the patient on a 75 degree tilt table. The contrast material is hand injected while the patient performs a Valsalva maneuver. Reflux of contrast material is followed by fluoroscopy and cut films are taken. As it slips past, the contrast material usually will outline the valve structure much as frost on a windowpane.

Surgical Procedure: The common femoral, superficial femoral, profunda femoris, and greater saphenous veins (if the latter is present) are approached through a longitudinal incision placed over the common femoral vein. Raju and Fredericks[10] prefer to perform the dissection with a scalpel rather than with scissors in order to avoid venospasm. As opposed to veins that have been involved by a previous episode of thrombophlebitis, the veins of a patient with

PVI usually do not have the intense perivenous scarring unique to post-thrombotic veins. The various branches of the major veins are ligated so that approximately 4 cm of superficial femoral vein is isolated. The proximal valve is identified by its characteristic bulge in the upper superficial femoral vein. The vein is then milked of blood to test its competence. An incompetent valve will permit blood flow down to the clamp placed distal to the valve. Following heparinization, soft, noncrushing clamps are placed on the common femoral, profunda femoris, and superficial femoral veins below the valve.

There are three open approaches to exposure of the valve commissure. Kistner originally described a longitudinal venotomy,[3] while Raju advocated a transverse venotomy placed above the valve.[10] Finally, Sottiurai used a combination of a transverse and longitudinal venotomy.[11] We, however, prefer the closed angioscopic technique first described by Gloviczki.[12]

Angioscopic Technique: Fourteen patients have undergone angioscopic evaluation of the valve before and after repair. The scope is inserted through a large tributary of the proximal greater saphenous vein which was invariably absent or via a branch of the femoral vein down into the superficial femoral vein.[13] Saline solution is infused through the angioscope, and the valve leaflets are observed for incompetence, which, when present, is both obvious and dramatic. The pathology of PVI usually demonstrates a wispy gossamer-like valve with redundant valve margins.

The Closed Angioscopic Valve Repair Technique: After placing two or three 7-0 monofilamant sutures on each side of the valve from outside the vein under angioscopic guidance, the repair was tested for competence by infusing saline solution through the scope. Common to the patients who have undergone angioscopy is the use of the angioscope to judge the competence of the repair rather than the strip test. If the valves are incompetent, additional sutures are added. By contrast, if the repair is narrowed sutures may be removed.

Results: Table I demonstrates the preoperative demographics for seven series in the literature which total 254 limbs. All series except ours were carried out by the open technique. The indication for surgery varied but averaged 75% for stage V/VI disease. Kistner's[14] series has the longest follow-up period. In general patients do quite well regarding ulcer recurrence with rates varying from 35% to 19%. Several series such as Kistner's and the recent one of Perrin[15] show that valvular incompetence is related to the duration of ulcer-free survival. The value of angioscopic repair of primary valvular incompetence lies in the determination while in the operating room that the repair is competent. In the absence of further valvular degeneration in the postoperative period, this finding should be correlated with a good outcome.

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Table 1.– Summary of Preoperative Demographics for the Surgical Treatment of Primary Valvular Incompetence by Valvuloplasty

Preoperative Demographics

Series	Country	Institution	Year	#Limbs	Indicat (% ulcers)	Technique
Kistner[14]	USA	Straub Clinic	1992	51 (48 pts)	57%	Open ± Perfs
Raju[10]	USA	Mississippi	1987	107 (42 pts)	71%	Open ± SFJ lig
Perrin[15]	France	Clinique Grand Large	1999	52 33 (28 pts)	100%	Open ± Perfs
Eriksson[16]	Sweden	Uppsala Univ Hosp	1989	22 (20 pts)	?? %	Open ± Perfs
Sottiurai[11]	USA	L.S.U.	1987	20 (12 pts)	100%	Open
Simkin[17]	Argentina	Clinica Quintana	1988	7 (7 pts)	100%	Open (3) Plication (4)
O'Donnell	USA	NEMC	1992	14 (14 pts)	100%	14 open

Table 2.– Summary of Postoperative Results for the Surgical Treatment of Primary Valvular Incompetence by Valvuloplasty

		Postoperative Results			
Series	Follow-up(mos)		Imaging	Hemodynam	Clinical Results
Kistner	48-252 (108)	86%	67% (PVI)	60%	35% ulcer
Raju	24-96			85% 1 year 75% 2 years 63% 3 years	7% DVT 5% bleed 5% infec
Perrin	24-96 (58 months)	85%	68%		19.2% ulcer recurrence 77.8% ulcer free survival
Eriksson	6-84		100%	64% 6 mos 62% 84 mos	30% ulcer
Sottiurai	10-73			80%	?
Simkin	??			50%	
O'Donnell	12-62			100%	85% remain healed

Range of follow, as well as (mean follow-up). Imaging refers to the percent of patients who were free of reflux on phlebography or duplex.
Hemodynamics refers to the percent of patients who had normalization of their APG or VRT#.
*Angioscopically guided valvuloplasty

INTERNAL VALVULOPLASTY

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In case of primary deep venous reflux and when deep venous reconstructive surgery is planned internal valvuloplasty looks to be in our experience the recommended surgical procedure.

The rationale for recommending Internal Valvuloplasty (IV): First of all because other techniques have not yet provided long term results as good as IV.

-Valve transfer (transposition, transplantation) has been mostly used to treat secondary deep vein reflux and generally their results are not as satisfactory as those obtained by IV (Perrin, Raju, Sottiurai).

-Psathakis operation II had given excellent results to his promoter but disappointing in small series reported by others (Perrin, Scurr).

-Several authors (Belcaro, Lane, Raju, and Schanzer) had performed external wrapping (Veno-cuff, banding with Gore-Tex or Dacron sleeve). Results are difficult to assess as various materials and techniques had been used, indications were different according to authors and long-term results are not available. Furthermore, I cannot clearly understand how shrinking of the vein diameter may work to correct reflux when the free borders of the valve are elongated and already in contact.

-Plagnol and Raju had used neovalve. The former had reported only mid-term results (average 18 month) in 44 extremities including 32-graded C6. Ulcer had recurred in 3/32(9.4%) and hemodynamic failure in 6 /44(13.6%).

-Hoshino, Kistner, Gloviczki, O'Donnell and Raju had used external valve repair, but again we have only short- or mid-term results. The advantages of the external valvuloplasty (EV) compared to IV are: EV is quicker than IV, allowing multivalve repair and avoids phlebotomy. In our unit we have only performed EV in addition to IV at the popliteal level without using angioscopy. Angioscopy is certainly very helpful as recommended by Gloviczki, Hoshino, and O'Donnell. I would add that in EV, the vein needs to be peeled off, and that might be detrimental to the vein wall vascularization.

-Internal Valvuloplasty: Kistner, Raju, and Sottiurai have described three techniques. We used the latter with minor modifications because it seems easier to perform valve repair through the T-shaped phlebotomy.

The ideal site for performing valvuloplasty is still under discussion: Sottiurai recommends popliteal level and Raju termination of the superficial femoral vein. In our series the latter has been chosen.

One of the potential hazards in IV is postoperative thrombosis. All our patients have had a postoperative ascending phlebography (24 to 36 hr. after surgery) to assess this complication. In IV (#65) for primary vein reflux we have recorded 5 (7.6%) limited thrombosis in situ or distal to the valve repair. Our results are summarized in Tables I, II, III, and IV. Table V displays results gathered through the published literature. Until updated data on others' techniques with long follow-up results assessment will be presented, IV seems the more reliable surgical technique to correct deep venous reflux.

Table 1

MATERIAL AND METHODS

1988 - 1997 — 85 extremities treated by Valvuloplasty

65 for PVI — Group I
19 for PVI (?) + PTS (distal) — Group II
1 for KT

Table 2

MATERIAL AND METHODS

1988 – 1997 — 85 lower limbs treated by valvuloplasty

35 for C5 - C6 (41.2 %)
Follow-up : 12 - 96 m
(average 64)

Table 3

VALVULOPLASTY for ULCER (C5 - C6)

Clinical Results

(Nb of extremities)	Ulcer Recurrence (%)	Non Healed (%)
Group I (24)	3/24 (12)	1/24 (4.1)
Group II (10)	5/10 (50)	1/10 (10)

P=0.03 (exact Fisher Test)

Table 4

VALVULOPLASTY for ULCER (C5 - C6)

Hemodynamic Results

(Nb of extremities)	No or minor Reflux	Major reflux (%)
Group I (24)	18	6 (25)
Group II (10)	4	6 (60)

P=0.05 (exact Fisher Test)

Table 5.– Internal Valvuloplasty Results

	No Limbs (# Valve Repaired)	Etiol. PVI	Follow-Up in months (average)	Clinical Results Ulcer Recurrence (%)	Hemodynamic Competent Valve (%)	Results ■ AVP, ▲ RT
KISTNER	32	/	60-252 (127)	(50)	24/31 (77)*	■ ↗ 81 % (m) ▲ ↗ 56 % (m)
RAJU	68 (71)	/	12-144	16/68 (26)	30/71	/
SOTTIURAI	118	/	8-146 (71)	9/42 (21)	89/118 (75)	/
ERIKSSON	27	27/27	(49)		19/27 (70)	■ ↗ 81 % (m) ▲ ↗ 50 % (m)
PERRIN	85 (94)	65/85	12-96 (58)	10/35(28.6)†	51/83 (62) 64/83 (77)*	■ Normalized 63.2 %

ABBREVIATIONS

I = Internal Valvuloplasty
E = External Valvuloplasty
W =Wrapping
↗=Improved
(m) =Mean

AVP = Ambulatory Venous Pressure
RT = Refilling Time with Tourniquet
* No or mild reflux
† Ulcer recurrence or non-healed ulcer

CASE OF PRIMARY DEEP VENOUS REFLUX

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A 36-year old gentleman from Western Samoa has had recurrent venous ulcerations of both legs that first began when he was 15 years old. He has had at least 15 recurrent ulcers mainly on the anterior and lateral aspect of the right lower leg. He has not been able to work for the past 4 years because of painful, large ulcerations of the right leg. His weight is 340 pounds. He has normal pulses in his foot arteries.

Level 1: There is moderate swelling of both legs with extensive lipodermatosclerosis of both lower legs. Large varicose veins are visible. Large ulcer on the outside of the right leg above the lateral malleolus, smaller ulcer anteriorly (Fig. 1). Hand-held Doppler examination showed significant reflux of the GSV and popliteal vein.

Level 2: Duplex scanning of the right leg showed significant axial reflux of the superficial femoral and popliteal vein, as well as the GSV. There were at least 2 medial and 2 lateral incompetent perforators. APG could not be performed because of the size of his calves.

Level 3: Ascending venography did not show any signs of postthrombotic changes. Descending venography showed cascading axial reflux through the common and superficial femoral and popliteal vein into the calf veins - Kistner 4 (Fig. 2). The deep femoral vein was competent (Fig. 3). The reflux of the GSV was confirmed. No incompetent perforators were seen. Lymphoscintigraphy showed irregular channels in the calf with localized dermal backflow with otherwise good lymphatic flow from the foot to the pelvis.

CEAP classification: C2,3,4,5,6,s; Ep; As,p,d; Pr2,3,11,13,14,15,18

Treatment?

(See figures on next page)

DISCUSSION

DR. O'DONNELL: I think we have two votes for a staged approach-treating the superficial system first and then reserving deep venous reconstruction for recurrence of ulceration. I'd be curious since we have many experts on the stage, Raj, what would you do in this case? Would you wait for the ulcer to heal on conservative therapy and then do the surgery or would you do the surgery right away?

DR. RAJU: The presence of active ulcer has not affected the outcome in any significant fashion. So I think it's a waste of time to wait for the ulcer to heal. Whether you do it in stages or do a complete hemodynamic correction I think depends on the patient. If it's a patient whom you think would tolerate staged procedures, i.e., do a little bit and wait for it to recur after six or seven years and do something else, that's okay. Generally speaking, very young patients and very old patients, those two extremes, you need to do the radical procedure because -- they may not come back if you do something less than total correction and the disease recurs. So I think it's a matter of clinical judgment.

DR. DEPALMA: I'd be happy if the patient didn't come back. I think this is a terribly high-risk patient, and I have dealt with some of these Samoans actually out in the West Coast. I would bet he's diabetic. I don't know if that test has been done. He's not. Okay. The weight, the driving weight of the abdomen, is the factor causing the venous insufficiency, not some magical thing with the valves. I would almost be tempted to send him to a member of my department for gastric partition first. I would probably recommend that because his life expectancy is shortened with this obesity. I would treat the ulcer locally probably with Circaid devices because stockings would never fit. I would also consider doing a lateral SEPS. I have done lateral SEPS in a very obese patient, and you can come down just posteriorly and get into a compartment and find perforators. I've got some pictures of such a procedure. I would do some local thing there or just put them in the stockings. The other thing I would do is to try to heal the ulcer. Medical therapy wasn't mentioned, doxycycline or tetracycline twice a day. A metalloprotease inhibiter tends to inhibit this kind of inflammation and skin change. I would prescribe these chronically. I would stay away from the deep system in this man. I personally would be afraid of getting a pulmonary embolus.

DR. DALSING: I think this patient's weight is a major problem and I think it would be very difficult to gain access to the deep system. I also would have liked to have seen the first portion of the descending venogram to know if I really had valves to work on. I may have missed it on the second part of the venogram, I couldn't tell if there was one there or not. I think if I had to do something and if we took care of some of his weight, I'd do the superficial stripping first with the perforators and then see what happened before approaching the deep system.

DR. GLOVICZKI: I agree with Bob that these types of patients, who come from a long distance, they come and they would like to have a complete operation. They have one chance. You want to fix all problems. Not very long ago I had a patient from Turkey where I did the same thing. I did SEPS, stripping and deep venous valve reconstruction. I am hesitant to do it all at one stage same day, and the reason I am is because I anticoagulate my deep venous reconstruction patients, but I would be hesitant to do that to patients who have stripping and SEPS and avulsion of varicose veins. So I would do the SEPS and the superficial ablation, and then probably a few days later I would do the deep vein reconstruction and full anticoagulation and then send the patient back to Samoa or Bora Bora.

DR. O'DONNELL: It's not bleeding with SEPS that you fear, but rather bleeding with avulsion of the greater saphenous. Many of us combine perforator interruption with deep venous reconstruction and use peri-operative anti-coagulation.

DR. GOREN: I applaud the panel's conclusion that a staged approach is best in these types of cases. I also fully agree with Dr. O'Donnell's statement regarding the existence of overload incompetence of the deep venous system caused by a hemodynamically significant superficial reflux existing in certain cases of varicose veins. I will illustrate this with three slides from a case study

Figure 1.– Large clover-shaped ulcer of the right leg which is swollen with extensive lipodermatosclerosis.

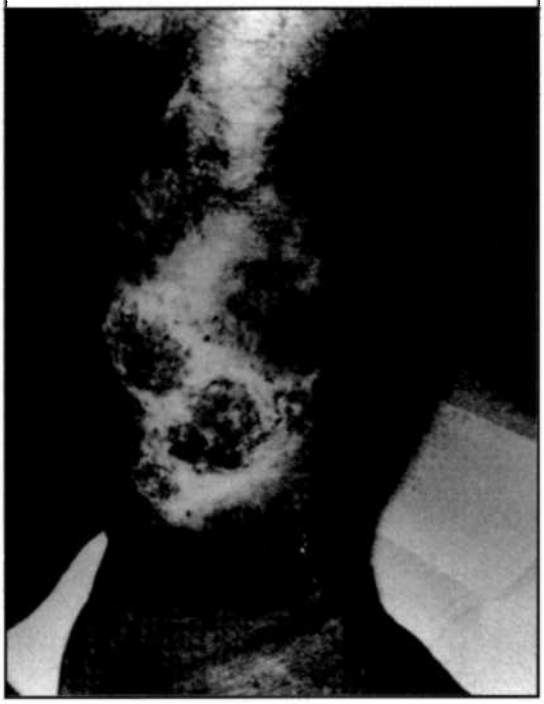

Figure 2.– Descending venogram of the right leg through catheterization of the left femoral vein showed cascading axial reflux (Kistner grade 4) through the common and superficial femoral, into the popliteal and tibial veins.

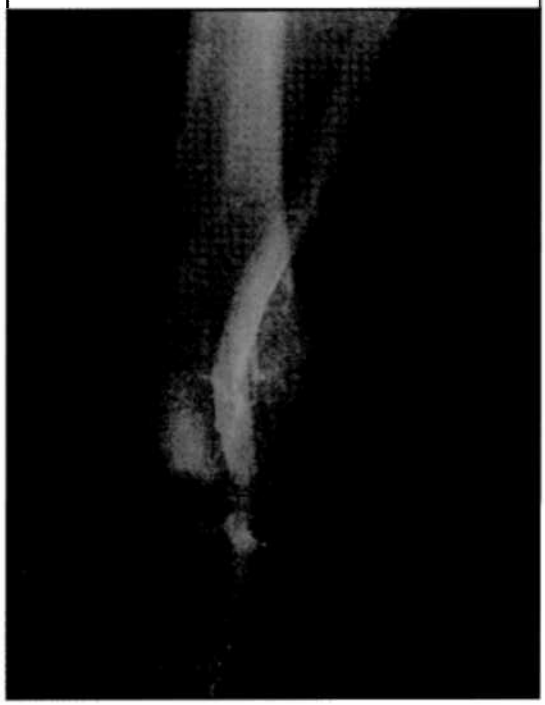

Figure 3.– Descending venogram showed that the deep femoral vein was competent.

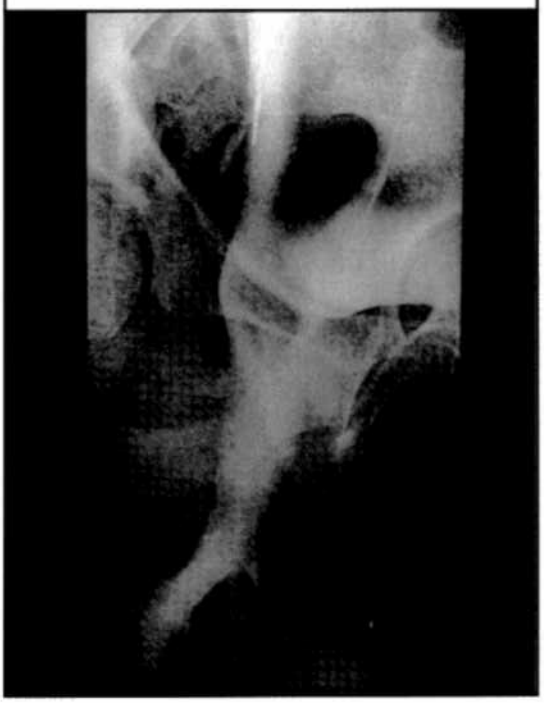

Figure 4.– Healing of the ulcerations after high ligation and stripping of the long saphenous vein, multiple perforator ligations and transposition of the incompetent superficial femoral vein into the competent deep femoral vein.

encountered in my practice. A middle-aged gentleman, college professor, consulted me for huge and neglected left long saphenous varicosities with obvious signs of chronic venous insufficiency (C5-6). On Doppler ultrasound examination he was found to have both superficial and deep (popliteal and superficial femoral) vein incompetence. An APG examination revealed a VFI of 9.1 ml/sec. The test was immediately repeated with finger occlusion of the long saphenous vein against the tibial condyle. The VFI dropped to only 5.3 ml/sec validating the existence of a significant deep reflux. Both his EF as well as RVF worsened, probably due to retrograde ejection through incompetent perforators. Two years after complete surgery for his long saphenous varicosities (with no perforator surgery) his VFI returned to normal at 1.48 ml/sec and his EF and RVF were normalized as well. In the case presented here before us, there are additional aggravating factors to reckon with. One is the excess weight and the other is a possible diminished calf pump function due to a limitation of movement in the ankle joint. Prof. Hach of Germany has described this condition namely the ankle joint failure as the "artherogenic congestion syndrome". I would like also to emphasize that some of these CVI patients have secondary gains from the condition. The whole family is catering to the patient who is the center of attention. Possibly too, these people are getting financial assistance for their disability so that the incentive to get well is simply not there. In my practice if I encounter a non-cooperating patient, he/she will be released and advised to seek help from someone else.

DR. KRYLOV: I have a question to our distinguished moderator. When you're doing these endoscopic repairs do you use anticoagulation?

DR. O'DONNELL: Do I use anticoagulation? Yes. Postoperatively these patients receive anticoagulation because I had two internal valvuloplasties that thrombosed while not receiving anticoagulation. I know some of the other panelists don't anticoagulate. They were the only deep venous reconstructive procedure in my experience that's thrombosed. Now all of my deep venous reconstructions get therapeutic heparin.

DR. KRYLOV: And the patient is ambulated anyway?

DR. O'DONNELL: Yes. The patient after a procedure like this is ambulatory with a little difficulty because they have a groin incision.

DR. KRYLOV: You are not keeping him in bed?

DR. O'DONNELL: No, I'm not keeping him in bed. Why should I keep him in bed?

DR. KRYLOV: And a question to Dr. Kistner. In this case if you will find the valve below the profunda you could repair that, but if you will open the vein and you will not find the valve, what do you do in that case? The phlebogram is not 100 percent distinctive. You just see something which could be the valve. It is especially bad that we are more and more relying on the duplex data, and the duplex is even less accurate than a phlebogram. What would you do in a case if you will not find the valve but the vein is already open?

DR. KISTNER: I think that brings up a good point. If you're going to do venous reconstruction, you need to know a fallback procedure, and in this man I think there is an excellent fallback procedure because he has a competent profunda system and could well be treated by a transposition.

DR. O'DONNELL: But what would happen if you didn't have that valve function and you got in there? What would you do next? I think that's Dr. Krylov's point.

DR. KISTNER: The profunda valve does function.

DR. O'DONNELL: But he's saying in a hypothetical case it doesn't. You get in there. It doesn't. Then what would you do?

DR. KISTNER: Then your fallback position is probably an axillary transplant.

DR. KRYLOV: With false positive data on the phlebogram, due to twisting of the vein, the picture will indicate that the valve is competent, but actually the valve is not present.

DR. KISTNER: Are you speaking of the superficial femoral valve or the profunda valve?

DR. KRYLOV: No, superficial femoral.

DR. KISTNER: Yes, I agree with you. Sometimes you get there and it's not what you thought. The first thing we would do as we approach the vein is an adventitial dissection to look for the white line of insertion of the valve cusp. If that line is not complete, then we know that we're not going to get a competent valve if we try to repair it. Then I would go probably to the transposition procedure.

DR. KRYLOV: In my situation I prefer to open the vein in the sinus, and in this case I can simply close the vein and make something else including this transposition.

DR. BELCARO: I think that one of the important requisites of valvuloplasty is that it be done only in subjects without severe conditions or complications like obesity or diabetes. I think it should ideally be done only in normal subjects.

DR. O'DONNELL: If they were normal subjects, we wouldn't be doing them.

DR. BELCARO: By "normal" I mean normal weight, body mass index, not diabetics, and so on. I mean only patients with pure primary incompetence. Also, I want to say to Professor Perrin that the follow up of valvuloplasty (which we call limited external valvuloplasty) now include more than ten years including more than 60 patients. We think it's a very useful procedure if you limit valvuloplasty mainly to patients with primary incompetence. About secondary incompetence due to post-thrombotic syndrome, most of these patients (maybe 35 percent) may have a thrombophilia or some situation which can predispose them to new episodes of thrombosis. I don't think that (unless we have special cases) in post-thrombotic syndrome we should actually use valvuloplasty. In primary incompetence we can perform valvuloplasty and it's very effective. In secondary incompetence due to post-thrombotic syndrome we really need some guidelines because only a very limited amount of patients can benefit from this procedure.

DR. KISTNER: I guess my question is whether there is a primary incompetent valve in association with post-thrombotic disease. If so, I treat them as a primary valve problem with valvuloplasty and expect to get good results. If it is a scarred valve, I have not been successful with freeing up the scarring and having it become functional. I think maybe Raj has a better experience and would like to hear what he has to say about it.

DR. RAJU: I think some post-thrombotic valves can be repaired, but you should feel good about it. If the quality of repair is good, then it's reasonable to expect a good result. I do not think general surgeons should be doing valve reconstruction, not because they are general surgeons. I think anybody who does this has to focus on venous disease to a large extent. I think otherwise the patient gets the short shift.

DR. O'DONNELL: I think we've all seen three or four patients who have had vascular surgeons who do occasional venous surgery have this short shift, as you say.

DR. NEGLEN: I have a question for you, Bob. Have you ever had to do an axillary vein transplant in a primary sufficiency patient, and if you had, how often do you find that the axillary valve appears insufficient too?

DR. KISTNER: Peter, I don't have enough experience to tell you. If I've done them, it's been just once or twice that I've had to take an axillary vein to put down there. So I don't know, but I think you have better data on that. Why don't we reverse the question?

DR. TRIPATHI: This is a question for Bob. Nobody is talking about venous ligation, and in patients who have got valvular dysplasia or aplasia, what is the role of venous ligation if the profunda axis is quite good and competent?

DR. KISTNER: If you have aplasia without any other competent axial segment, ligation doesn't play a role. I think you're just stuck in that situation. The only possibility that I know of is a cryopreserved homograft valve.

DR. TRIPATHI: What are the options when the profunda is okay or competent and what are the options when the profunda is not competent?

DR. KISTNER: If the profunda is competent, you always have the alternative of a transposition which is my second choice. If the profunda is not competent, then I don't think reconstructive surgery, would play a role, unless you have a very severe advanced case, and certainly not in this kind of a patient.

DR. TRIPATHI: And how does ligation compare in a case where profunda is competent with transposition?

DR. KISTNER: If the profunda is competent, a compromised (partially thrombosed) superficial femoral vein can be ligated with good results. If there is still good outflow through the SFV, we prefer transposition to ligation.

DR. DEPALMA: There's one way of looking at that, and that is on the operating table to occlude the superficial femoral vein and measure the pressure in the foot and in the arm the way that Raju does, then change the position. And see if you tilt the table down or if you clamp the vein in the supine position and the pressure in the foot goes up, do not ligate the superficial vein.

DR. DALSING: Just one comment. If the question was, can you ligate a superficial femoral vein with a competent profunda? It would probably be okay. Bob has actually looked at that question and found no long-term disability. If you have a clot within the SFV and you ligate it, even if it doesn't involve the entire vein and the profunda is normal, you should be all right. However, if the profunda is incompetent, then you have a problem long-term.

DR. GARCIA-RINALDI: I'd like to ask Dr. Kistner that if we feel that the concept of overflow is the cause of venous dilatation and incompetence, how many of the transposition cases into the deep femoral have indeed developed insufficiency of the valve.

DR. KISTNER: The concept that overflow in the venous system causes venous dilation of the unobstructed deep system makes little sense to me. I really don't understand it. If you really have valvular pathology, I don't believe cutting off saphenous inflow is going to cause that valve to work again unless there is proximal obstruction. I don't understand that physiologic concept very well, so someone else will have to speak to that.

DR. O'DONNELL: Well, we didn't understand it well because it's similar to the state of carotid disease before the availability of duplex scans to detail the extent of carotid occlusive disease. Similarly there was valvular incompetence in many of these patients whom we thought had primary varicosities. Certainly in my practice it's not unusual to encounter a number of patients with incompetence of the superficial femoral valve with reflux by duplex scan. Following a standard ligation and stripping the reflux disappears post-operatively.

DR. KISTNER: Is that a total axial reflux, Tom, or segmental?

DR. O'DONNELL: It's segmental, maybe includes the mid-thigh valve, but the popliteal is usually competent.

SURGICAL MANAGEMENT

DR. EKLOF: This patient comes from Western Samoa. We found out that he had eight male relatives, all uncles that had the same problem as this young man had. Since their early teens they developed recurrent venous ulcers of both legs. Our patient came to Hawaii about eight years ago. He is a very healthy, big, strong Samoan. Typical football player with a perfect calf muscle pump function. He has no ankle problems. He has never had DVT. He had been treated for a long time with Unna boots without any effect on healing. So when he was referred to us we did all the investigations that I showed to you, and we tried to continue conservative treatment to at least try to heal his ulcers before surgery. We were not successful. So after marking his perforators with a scanner the day before surgery, we did high ligation and stripping of the GSV. We also did perforator ligation with small incisions on the medial and the lateral side, where we found big perforators four to five millimeters wide. We explored the common femoral, and the deep and superficial femoral veins in search of the valve that was quite beautifully shown on the venogram. We did a very careful excision of the adventitia, and could only see one cusp. Since we couldn't identify the opposing cusp, we couldn't do an external valvuloplasty. We decided to divide the superficial femoral vein, and we looked into its proximal part and found that he had only one valve cusp. We did a transposition of the superficial femoral vein into the competent deep femoral vein. This was in July. His pain disappeared immediately after his reflux was repaired. We have done several scans and it is completely competent. We skin grafted his ulcers and they are completely healed (Fig. 4). He has decreased his weight by about 20 pounds and he plans to go back to work.

(Scientific Articles continue on next page)

IV. POST-THROMBOTIC DISEASE: DEEP AND SUPERFICIAL

CASE OF SECONDARY DEEP VENOUS DISEASE-SOMETHING CAN ALWAYS BE DONE

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Compression therapy of deep venous insufficiency is associated with high recurrence.[1] In a carefully conducted study from Sweden, Nelzen, et al reported a 56% recurrence, the majority occurring in the first year. Apart from the high recurrence and primary non-healing (30%), noncompliance is a major factor in many compression regimens.[2,3] Noncompliance is associated with nearly 100% recurrence.[2] The reasons for noncompliance are many, including lack of self discipline, poor fit, a sensation of "binding" or "cutting off the circulation", cosmetic considerations, warm weather, high recurrent cost, and other more compelling reasons such as contact dermatitis, infirmity or arthritis that prevents the patient from applying the device without daily help. In addition to these drawbacks it is our impression that many patients under chronic compression regimens seldom get *complete* relief of symptoms particularly pain and swelling. A surgical approach provides a more definitive therapy with superior symptom relief. The majority of patients after successful valve repair discard their stockings,[4] and the remainder who continue to use them after surgery have a greater latitude and freedom in utilizing the device compared to patients on primary compression therapy. The surgical option should, therefore, be considered in patients in whom compression therapy had failed or cannot be applied.

Relatively minor improvement in hemodynamics can lead to remission with healing of stasis ulceration, even though substantial improvement in reflux parameters (see below re. obstruction) appears to be required for total relief of pain and swelling. This argues for an aggressive surgical approach in patients with secondary or post-thrombotic disease. This is the basis of the premise that a comprehensive correction of obstructive and refluxive pathologies as is practically feasible offers the best chance of symptom relief for the post-thrombotic patient. Hard data to support this philosophy is however not yet available and the approach is strictly empirical at the present time. However there have been technical advances in venous surgery, allowing a greater number of patients, many with pathologies previously considered inoperable to benefit from a surgical approach. Several different techniques[5] of valve repair are now available, allowing repair of even small caliber veins or multiple repairs if desired. There is little difference in the clinical result between the various techniques.

Similar ulcer healing is obtained as long as valve competency was restored irregardless of the individual technique used. Valve reconstruction techniques can now be applied to even post-thrombotic trabeculated veins[6] and axially transformed profunda femoris veins.[7] Cryovalves have become available for salvage cases. Secondary saphenous varicosities can be safely stripped[8] providing improvement in the overall reflux without affecting outflow. The advent of endovenous stenting has provided a means of a simple percutaneous technique in nearly an outpatient setting to afford significant symptom relief in the large subset of patients with stenosis or obstruction of the iliac veins. Relief of pain and swelling with this simple procedure has been impressive.[9] Approximately 30% of ulcers appear to heal with the stenting procedure alone.

In the last five years, >85% operability was achieved in post-thrombotic patients, even though no preselection was made based on severity of venographic appearance, size, extent or duration of the ulcer, or presence of procoagulant abnormalities. Employing this aggressive approach, actuarial ulcer healing of >60% at 10 yrs was achieved even in those with severely mangled and trabeculated post-thrombotic veins.[5]

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CASE OF SECONDARY DEEP VENOUS DISEASE – VALVE TRANSPLANTATION

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Introduction

It has been estimated that 2.7% of the U.S. population (~ 6 million people) have advanced chronic venous disease with approximately 800,000 new cases recognized per year.[1,2] These symptomatic patients often have deep venous disease (~ 70% isolated or 80-100% combined with superficial disease).[2] Approximately 85% are due to insufficiency and possibly 50% have an etiology classified as secondary (eg. post-phlebitic).[3,4] A rough estimation would suggest therefore, that of these six million patients about two million would have deep venous insufficiency due to a secondary cause and may require venous valve transplantation to free them from a life of disabling symptoms.

Indications/Preoperative Evaluation

Patient selection for venous valve transplantation is based upon symptoms, anatomy/physiology and the failure of other more conservative medical and surgical options designed to alleviate the patient's disability.

Patient symptoms coincide with a CEAP classification of 4 or higher.[5] The patients typically have severe lower extremity edema, lipodermatosclerosis and recurrent venous ulceration. Recurrent

ulceration unresponsive to medical management afflicting a motivated compliant patient constitutes a prime candidate for this surgical approach.

Anatomy/physiologic considerations are defined by the preoperative work-up. The initial history and physical examination can eliminate patients with insignificant venous disease while arterial disease (lower extremity arterial doppler examination) and a preexisting hypercoagulable state (Antithrombin III, Protein C, Protein S, Factor V leiden deficiencies, etc.) can also be investigated. The latter does not necessarily eliminate the patient as a candidate for venous surgery but does stress the need for stringent anticoagulant therapy if a surgical procedure is undertaken. The next step is a venous duplex study to define the precise location of disease throughout the leg (obstruction or insufficiency). In addition, a plethysmographic evaluation, usually air plethysmography, provides an overall hemodynamic estimate of calf pump function, venous obstruction or valvular insufficiency. Confirming insufficiency as the major problem, an ascending venogram with intravenous pressure measurements confirms the noninvasive findings and provides a roadmap of the venous anatomy. If obstruction is a significant problem, it would be addressed at this time. Lastly, a descending venogram performed by the method of Kistner[6] determines the presence and location of venous valves as well as the degree of reflux. Grade 3 and 4 reflux are considered abnormal.

Prior to considering a valve transplantation to alleviate the patient's complaints, all superficial and perforator disease should be addressed. I tend not to perform simultaneous major superficial and deep venous surgery due to the bleeding which may occur at the time of heparinization for the valve transplantation. Certainly, the less invasive nature and long-term results of valvuloplasty[7] make it a surgical approach which should be attempted prior to transplantation if applicable. The post-phlebitic patients considered for valve transplantation generally do not have the proper anatomy to allow this option. Valve transposition is also a viable first approach but is possible in less than 3% of patients evaluated.[8] Valve transplantation, due to its need for multiple incisions and more extensive operative repair, is the final option offered to patients who have exhausted all other avenues. For those 30-40% of patients who have no appropriate autogenous valve for transplantation,[9] cryopreserved venous valve transplantation is considered.

Technique

The goal of therapy is, of course, to prevent grade 3 or 4 venous reflux. When considering venous reflux, it is critical to evaluate the profunda femoral vein. Profunda reflux can allow grade 3 plus reflux and recurrent symptoms following the correction of superficial femoral vein incompe-tence.[7,9,10] Often, only one autogenous venous valve is available for transplantation. In this situation, the valve must be positioned below all thigh reflux. The superficial femoral vein will suffice if the profunda is competent,[7,9-11] but others have championed the popliteal location for most cases.[12-15] The popliteal location must be chosen if the profunda is incompetent. If two or more valves are available, correction of reflux in both the superficial femoral and profunda femoral veins may be appropriate.[16] The duplex scan and ascending venogram may help one decide which area of the vein is most normal in caliber allowing optimal flow and ease of surgical implantation.

The operation is routinely performed under general anesthesia to allow dissection in the leg and axillary region. A valve containing vein segment demonstrated to be competent by preoperative duplex scanning is harvested through an infra-clavicular incision positioned over the neurovascular bundle. Sometimes the only available vein is in the upper arm venous system but the size discrepancy makes this less desirable. Harvest is not performed until the standard groin or medial leg incision has allowed dissection of the appropriate superficial femoral or popliteal vein segment. In addition, systemic heparinization is provided prior to any venous interruption. At harvest, the axillary vein is simply ligated and divided to provide a 4-6 cm length of vein with the valve lying safely within. The lower leg venous segment is transected after applying vessel loop or vascular clamp control. The vein edges spring apart and residual vein is then removed to accommodate the length of upper extremity vein available. The anastomosis is performed with interrupted 6-0 or 7-0 prolene sutures in an end-to-end fashion. It is often useful to perform the cephalad anastomosis first, release the proximal clamp, and allow retrograde flow. The valve, if competent, will prevent reflux of the blood and the caudal anastomosis can be completed with proximal clamps removed. This confirms valve competence and allows more native femoral or popliteal vein to be removed if required for proper matching of length. The distal anastomosis is flushed, irrigated with heparinized saline, the last sutures tied and the distal clamp is removed. The "strip-test" confirms valvular competence. If incompetent, the valve is made competent by the closed technique of Kistner.[17] Careful hemostasis is mandatory. Suction drainage of the subcutaneous tissue is optional but often used because postoperative anticoagulation will be utilized. Incisions are closed in standard fashion. Intermittent lower limb pneumatic compression is begun in the operating room and continued until the patient is ambulating well. Postoperative anticoagulation may be with fractionated or unfractionated heparin followed by Coumadin therapy for 3-6 months or for life if the patient has a hypercoagulable state. Elastic compressive support is encouraged to control any residual edema.

For those patients with no autogenous venous valve available for transplantation, the use of cryopreserved tissue has been studied. The technique is essentially the same as for an autogenous transplant without the need for an axillary incision. The cryopreserved valve containing vein segment is ordered by blood type, diameter and length. It was originally procured from qualified donors with an acceptable superficial femoral vein valve. The allograft is shipped at –70°C and must be thawed for surgical use within 72 hours. The thawing process is a 4 step protocol generally requiring 30 minutes and, therefore, should be started significantly early during the groin or medial leg incision. Once thawed, the valve is checked for competence by injecting heparinized saline retrograde in the vein. Valvuloplasty may be used if required, but the company is willing to send more than one valve to ensure an immediately competent valve. I personally tend to add a distal arteriovenous fistula if the valve is placed low in the popliteal fossa. Anticoagulation is continued for the life of the valve. All other technical considerations are similar to those used for an autogenous valve transplant.

Results

The competence of upper extremity donor valves, when defined as

absent or only mild reflux and substantiated by duplex scanning or descending venography, range from 75-100% at six months.[7,10,13,14] With follow-up of 6-48 months, the reported competency rate was 34-100%.[7,10-14] Although not always in a direct one to one relationship, a competent valve translates into a symptomatically improved limb. Restricting the patients to those with prior or concurrent recalcitrant ulcers, ulcer healing occurred in 95% and recurrence was prevented in 60% up to 10 years post-transplant.[11]

Cryopreserved tissue has only recently been used for this clinical scenario, the single published paper suggests a 6 month valve competency rate of approximately 60% with ulcer healing/prevention of recurrence of approximately 67%.[18] Issues of rejection and the need for long-term anticoagulation make this a secondary choice for most patients.

Summary

The number of patients suffering from disabling chronic venous insufficiency is not insignificant and is generally treated with conservative medical maneuvers. There are alternatives including surgical procedures to prevent massive venous reflux. Proper patient classification and a regimented diagnostic evaluation including non-invasive and invasive imaging can define the patient who may benefit from a specific surgical approach. The transplantation of valves from the upper to lower extremity is generally reserved for patients with secondary causes of deep venous insufficiency who have no other options. The environment into which the valve is placed is a damaged, scarred conduit which probably explains the less impressive long-term function of these valves when compared to primary valvuloplasty. Nevertheless, one can expect a clinical benefit defined as ulcer prevention in approximately 60% of patients. For those lacking an autogenous valve, a cryopreserved valve may substitute with encouraging early results.

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ANGIOPLASTY AND STENTING OF THE OBSTRUCTED ILIAC VEIN

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Chronic venous insufficiency of the lower extremity is a complex disease with several etiological factors. In approximately one-third of the limbs with postthombotic disease an obstructive component is predominant. The treatment of the outflow obstruction has been hampered by difficulty in identifying hemodynamically significant obstruction and by a rather extensive surgery to correct it. Available surgical procedures like the crossover femoro-femoral bypass or axial iliocaval bypass graft constitute major surgery, always followed by long-term anticoagulation. The interest in venous obstruction is now rising owing to the development of the new technology to diagnose and treat stenosis and occlusion by percutaneous dilatation and insertion of stent. This study presents the technical aspects of this procedure and the results when applied in limbs with postthombotic disease.

Material

A prospective study of 78 limbs with post-thrombotic disease had balloon dilatation and insertion of stent to correct iliac vein obstruction (median age 47 [range 18-77], male/female ratio 1/1, left/right lower extremity 2.3/1). All patients had a thorough history taken and clinical examination performed. A visual analogue pain severity scale from 1-10, in which 10 is the most severe pain, was used to assess pain intensity. The clinical score as per the guidelines of the SVS/ISCVS was also used to assess swelling and pain.

The presenting limb complaint was active (24%, 24/79) or healed ulcer (8%, 6/79); lipodermatosclerosis, pigmentation and/or dermatitis (12%, 9/78); and swelling (50%, 39/78). Concomitantly, 35% of the patients had severe pain (> 5/10 as assessed by the analogue pain severity scale) and required analgesics. Only 16% of patients denied any pain. In addition, 97% of the patients complained of swelling. A comprehensive work up was performed prior to the intervention.

The following preoperative indicators of obstruction were used: occlusion or obvious stenosis on ascending or antegrade transfemoral venography, increased arm/foot pressure differential and/or abnormal hyperemia-induced pressure rise, and presence of pelvic collaterals on venogram. Positive preoperative pressure measurement was present in 38% of the limbs, radiological obstruction in 81%, and collaterals were visualized in 63%.

Intervention

All interventions were performed in a dedicated interventional room with ceiling mounted ISS equipment in the surgical suite with complete sterile precautions. The procedures were done under general anesthesia or local infiltration analgesia in combination with

monitored sedation. Initially, cannulation of the femoral vein was blind, but later it was performed with guidance of ultrasound. After cannulation and insertion of a pinnacle, an antegrade venogram was performed followed by intra-vascular ultrasound (IVUS). Degree, length, and site of obstruction were recorded, and the presence and type of collaterals were noted. Transverse vessel area and diameters were measured by IVUS. Intraoperatively pull-through pressure from the inferior vena cava to the femoral vein was then obtained. Femoral pressure distal to the presumed obstruction was obtained before and after injection of 30 mg papaverine intra-arterially to increase the venous flow. The obstruction was dilated with a balloon and the degree of waisting was noted. The venogram and IVUS investigation were repeated to measure any recoil. The dilated segment was then traversed with a stent of appropriate length and diameter (Wallstent). The venogram, IVUS investigation and pressure measurements were repeated to assess the final result. The sheath was removed and pressure applied for 10 minutes. The patients received 3-5,000 units heparin and 30 mg ketorolac intravenously during the procedure. All patients were admitted for less than 23 hours. Postoperatively, a foot compression device was used, dalteparin 5,000 units s.q. administered once daily; and a ketorolac injection repeated in the morning before discharge. Aspirin (81 mg p.o.) daily was started immediately postoperatively and continued. Only patients already on warfarin preoperatively owing to prior deep vein thrombosis and thrombophilia were anticoagulated postoperatively. Warfarin was not routinely discontinued prior to surgery.

The patients were followed clinically after 6 weeks and with repeat ascending/antegrade transfemoral venography and functional studies later.

Result

The deep system alone was involved in 42% of lower limbs. The minority (32%) had only obstruction and the remaining lower extremities had a combination of reflux and obstruction. The results of the hemodynamic studies performed before surgery were obviously affected by the high rate of concomitant reflux observed in these post-thrombotic limbs.

Intra-operative: Only one stent was used in the majority of cases, but as many as 6 stents were used in one patient. The median length of stented area was 8 cm (range: 4-26) and the median diameter of the inserted stent was 16 mm (range: 10-20 mm). The combined involvement of common iliac vein and external iliac vein (42%) was almost as common as of the common iliac vein alone (45%). The venogram underestimated the degree of narrowing by 10% compared to the findings of the IVUS investigation. Obvious waisting of the balloon on inflation, indicating the presence of resistance at the narrowed portion of the vein, was found in 87% of limbs. Minimal change was observed in 1% and no waisting occurred in 12%.

Venous collateralization was found on the intraoperative venogram in 78% of patients. Transpelvic collaterals were most commonly seen (89%), followed in frequency by a visualized ascending lumbar vein (32%), and axial collaterals (18%). After balloon-dilatation and/or stenting, the collaterals disappeared completely in 72%, substantially decreased in 17% and remained essentially unchanged in 11% of limbs. A subgroup of 17 limbs with diffuse narrowing of the iliac vein with no collateral formation was identified. After balloon dilatation the vessel in most instances recoiled towards the original dimension to varying degrees. The mean recoil percentage was 86% (± 26, SD). No iliac vein pressure gradient was observed in many of the limbs during the intraoperative measurement. A resting gradient of $\geq$ 2 mmHg was seen in only 18% of limbs. This rate increased to 54% after papaverine injection. The cross area increased from 0.44 ± 0.33 to 1.51 ± 0.41 cm^2 poststenting.

Post-operative: The early complication rate was low and there was no mortality. Thrombosis of the stented area occurred in 7 limbs within 3 weeks of the surgery. Thus, the postoperative occlusion rate was 8%.

During the initial part of this study (16 limbs) care was taken not to insert any stent into the inferior vena cava, but rather to place it slightly beyond the stenosis even when the narrowing was at the ilio-caval junction. Five of these patients (38%) had full relief of symptoms after treatment, but returned during the follow-up period with symptomatic recurrence 6-8 months later and restenosis central to the stent. All limbs were restented successfully with placement of the stent well into the inferior vena cava. When this late complication of stenting was realized, all subsequent stents have been placed well into the inferior vena cava. Neither deep vein thrombosis of the contralateral limb nor any recurrence of stenosis of the common iliac vein has occurred.

Clinical: The median clinical follow-up of 62/78 limbs (79%) is 9 months (range: 1-27). Antegrade transfemoral or ascending venograms was performed in 54 patients. Three stents had irregularities within the stent, indicating hyperplasia or partial rethrombosis but no obstruction to flow. During follow-up one limb was found with recurrent stenosis distal to the stent, and late occlusion occurred in 2 limbs (4%). The remaining patients (8) had duplex Doppler ultrasound performed, which showed no indication of obstruction of the iliac segment. The primary, primary assisted, and secondary patency rates at 1-year were 75%, 87%, and 89%, respectively, as per reporting standards of the SVS/ISCVS.

There was substantial pain relief after surgery. The rate of patients free from pain increased from 16% to 64% postoperatively. The mean value of the pain intensity scale decreased from 4.3 ± 2.5 (SD) before the intervention to 1.5 ± 2.4 (SD) ($p < 0.001$) on follow-up. The leg edema also improved. Prior to surgery 97% of patients complained of varying degree of swelling; this rate was reduced to 57% after the procedure. The clinical score of swelling decreased from 1.4 ± 0.5 (SD) to 0.8 ± 0.8 (SD) ($p < 0.001$).

Twenty-four limbs had active ulcer before the balloon dilatation and stenting. In eleven patients, the ulcer healed after the stenting before additional reflux surgery was performed (46%). The ulcer healed in two additional patients, but recurred quickly; four venous ulcers never healed, and seven limbs were not yet evaluated.

Conclusions

1. Venoplasty and stenting of the iliac vein is a safe procedure with no mortality, low morbidity, and substantial clinical benefits.
2. Iliac vein obstruction is a painful lesion, significantly improved by balloon dilatation and stenting.

3. The significant recoil after simple dilatation warrants a stent insertion in all cases following venoplasty.
4. Stents should be inserted well into the IVC to prevent recurrence of central stenosis.
5. The definite objective preoperative test to use for selection for this procedure needs to be defined.

CASE OF SECONDARY DEEP VENOUS DISEASE

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This 47-year old healthy male was referred with a 4-year history of chronic venous ulceration of the right lower extremity due to post-thrombotic disease of the femoral-popliteal-tibial veins.

The problem began 4 years ago with the spontaneous onset of itching, discoloration, and a small sore on the posterior aspect of the calf. Swelling and chronic aching with dependency were progressive, and the ulcer grew to a large, encircling lesion of the entire circumference of the calf. Treatment by a series of 8 physicians entailed many medications during this time, but elastic support was not prescribed. No objective tests were done. During this time the problem was progressive.

A clinically apparent DVT of the SFV was diagnosed one year ago and treated with heparin-coumadin. Workup for a hypercoagulable state was negative.

3 months prior to this referral, he consulted a new physician who diagnosed severe deep venous reflux and prescribed elevation of the leg, pneumatic compression and pumping of the leg, and limitation of activity. His ulcer closed over the next three months.

The dominant symptoms were aching and swelling of the leg, and venous claudication with walking. The need to pump his leg interfered with his occupation as a traveling sales person. He had to stop his hunting activities. The problem was that the treatment prescription to control the ulceration required a life-style change that was incompatible with his desired way of life, and with his occupation.

P.E.: Healthy, strong 47-year old male. 5'10" tall, 230 lbs. Normal physical examination including arterial pulses, except for venous findings in RLE: Right calf 1" larger than left; large discoloration encircling the leg, 6" in length on post-lat and 2" on medial sides; no induration of skin, good turgor. V.V. in leg, mild.

Doppler: Mild local reflux in Posterior tibial vein, local and sustained 4-second Valsalva reflux in the popliteal vein. Late onset reflux in GSV, as found with perforator reflux. Normal phasic flow in common femoral vein.

Phase II.
Duplex scan: Occlusion of SFV to adductor canal.
PFV-popliteal connection, with 2 second reflux
SFV-popliteal collateral, with 4 second reflux
Incompetent greater saphenous vein, with low velocity 4-second reflux
Popliteal, crural, and short saphenous reflux, low velocity, 4 second duration
Incompetent, 2.0 mm perforators, medial and lateral lower calf, single
Summary: Post-thrombotic extremity with mixed obstruction and reflux. Occlusion of entire SFV, and low velocity reflux in all veins.

APG: O.F.-normal at 50%
VFI - 2.72 ml/min
2.10 ml/min with superficial occlusion
VV - 75 ml
Ejection Vol - 58 ml Ejection Fraction - 77%
RVF - 51%
Summary: Normal outflow and reflux volumes. Calf muscle pump satisfactory. Elevated RVF

Venous Pressure:

AVP: GSV: sustained high pressure without fall while walking
Dorsal toe vein: Normal fall to 30mm HG, rapid rise (<10 sec)
Arm-foot: Resting: <4 mm. Hg. Difference
Post hyperemia: 7 mm. Hg difference
Summary: Sustained venous hypertension and rapid return to baseline
(Difference between GSV and toe tracings not explained)
Consistent with deep venous obstructive and reflux disease

Summary of Phase II workup and case analysis:

Findings diagnostic of post-thrombotic disease with elements of reflux and of obstruction shown on duplex. Physiologic confirmation of significant obstruction and reflux lacking in APG. Venous pressure consistent with venous disease, both obstructive and reflux.

Further workup needed to find a way to improve his symptoms since his present way-of-life was unsatisfactory.

Phase III.
Ascending venogram: Ascending flow preferentially by superficial veins, even with tourniquet at the ankle. Ankle and upper calf tourniquets required to force flow into severely distorted tibial, popliteal, and SFV-PFV veins. Popliteal vein distorted, becoming obstructed above popliteal space.
Large GSV, main outflow tract from the calf.
The LSV (SSV) ended in a Giacomini vein which ascended to join the saphenofemoral junction. There was no connection between the LSV and the popliteal.
Normal CFV-Iliac-IVC.
No significant calf or thigh perforator veins seen.

Descending venogram: Upright examination with Valsalva revealed:
Non-visualization of SFV
Reflux in distorted PFV down through PFV-popliteal branch into popliteal vein, and then reflux down into anterior tibial and muscular veins. One large lateral branch of the CFV (common femoral vein) showed a competent valve.
GSV showed slight reflux in thigh only. (Valve leaflets seen in GSV.)

CEAP: C2,3,4,5(s); Es; As,p,d; Pr2,3,4,5,12,14,15,18; o12,13,14,15

Analysis of workup:
Life-style limiting venous claudication, pain, and swelling in an otherwise healthy and athletic 47 year old male. Healed ulceration and significant skin changes. The problem was due to post-thrombotic disease which had virtually wiped out the deep venous return in the deep veins. Most of the obstruction was in the lower thigh, extending down through the popliteal into the calf veins. Reflux of greater or less degree was present in all veins except two, and these were the Giacomini vein and the lateral branch of the PFV. The GSV and the perforator veins were not of great importance in the process. The dominant symptoms which limited his lifestyle appeared to be due to poor emptying of the calf. Outflow from the calf was limited by deep vein obstruction in the SFV, poor collaterals, and reflux in the PFV. Outflow channels were the GSV, the Giacomini extension of the SSV, and possibly the competent lateral branch of the PFV (Fig. 1).

Figure 1
Diagram of pre-operative condition determined by duplex scan and venography. A, upper end Giacomini vein with competent valve. B, junction of LSV with Giacomini vein at popliteal level, showing obliteration of branch to popliteal vein. C, incompetent profunda femoris vein with large communication to poplital vein. D, permanently occluded superficial femoral vein. E, lateral branch of common femoral vein with competent valve.

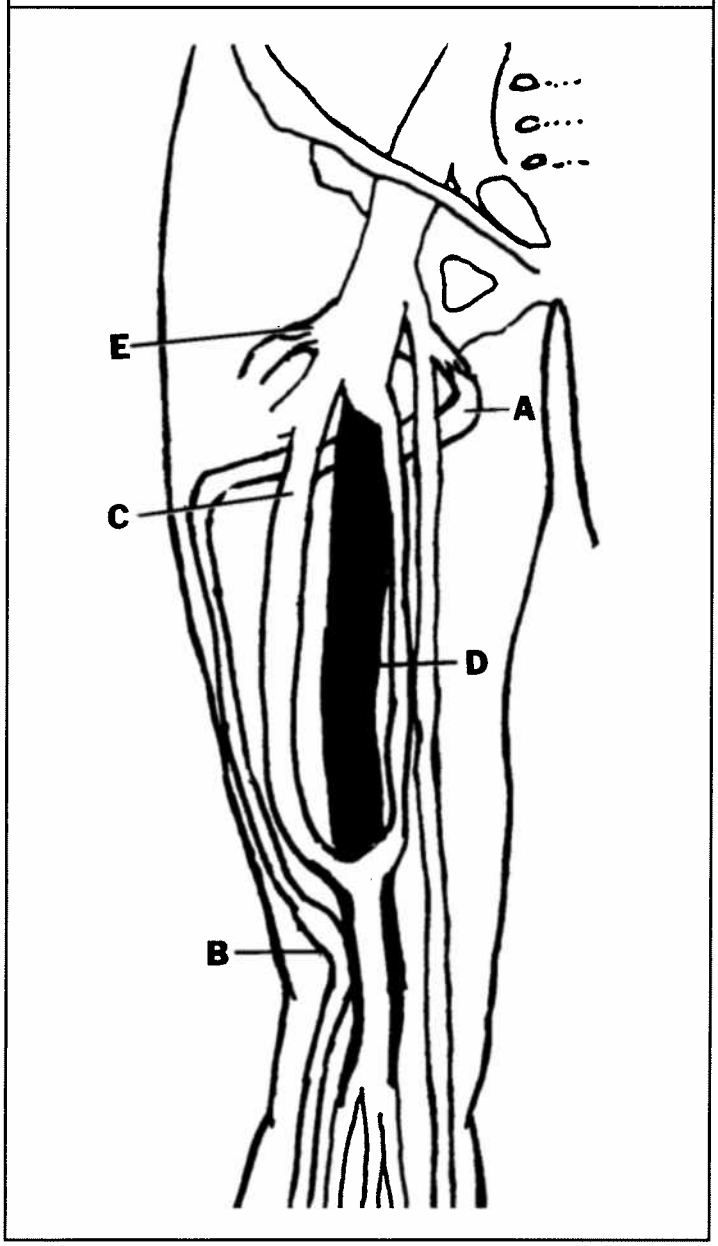
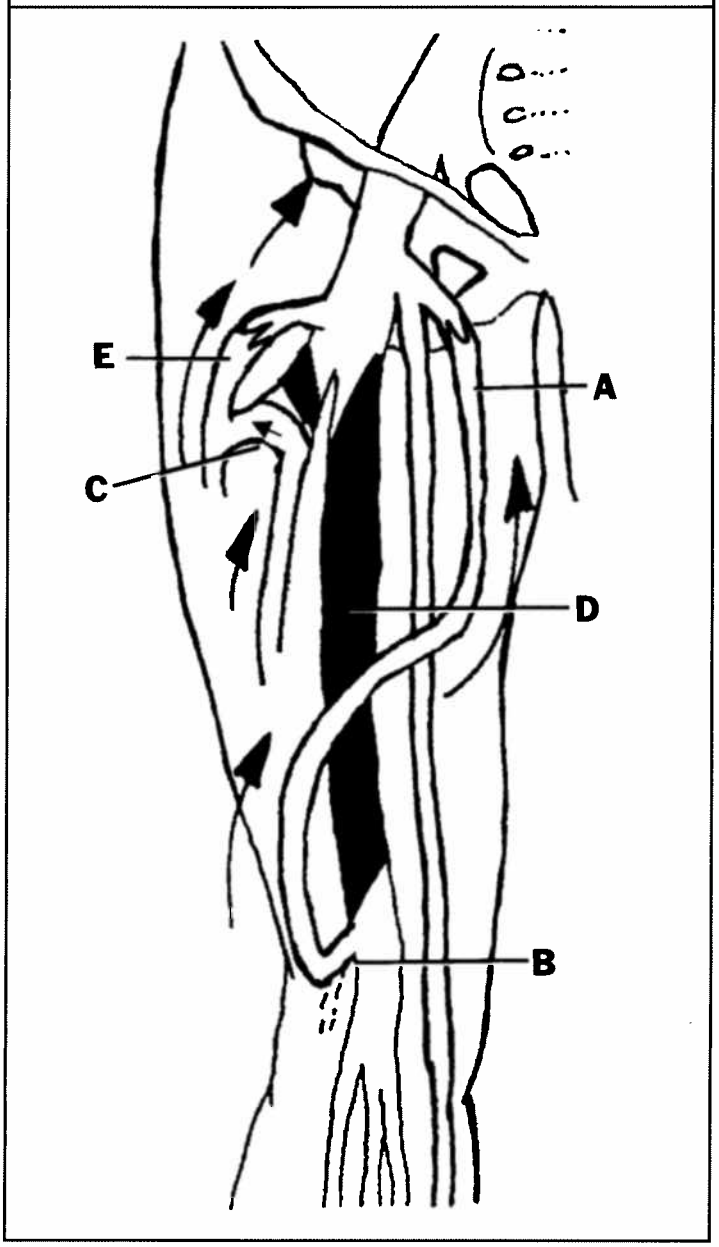


Figure 2
Diagram of surgical procedure. A, upper end of Giacomini vein with competent valve utilized as outflow for the popliteal vein. B, lower end of Giacomini vein disconnected from LSV and anastomosed to popliteal vein to serve as popliteal outflow. C, incompetent PFV disconnected from CFV and transposed to the competent lateral branch of the CFV. D, permanently occluded SFV. E, Competent valve in lateral branch of CFV.



DISCUSSION

DR. O'DONNELL: That was an excellent presentation, and represents one of the largest experiences with this problem.

DR. GLOVICZKI: Well, this is an extremely complex case, no question about it, and I may follow a little bit what Mike was suggesting, try to stay on the conservative side before I would decide on any type of deep vein reconstruction. I'm even thinking of some kind of a May-Husni operation to use the saphenous vein to bypass the obstruction.

DR. O'DONNELL: Do you think they're missing something in the iliac segment?

DR. GLOVICZKI: I certainly would look very, very carefully into that venogram. I don't have large experience repairing deep vein valve incompetence of the profunda femoris vein, that's another option that could be done in this case.

DR. DEPALMA: Tom Wakefield pointed something out that Mike Dalsing emphasized too, that this patient may be suffering from repeated episodes of thrombophlebitis. I would seriously consider long-term anticoagulation and Heparin or low molecular weight Heparin therapy with elevation to see if these would improve his symptoms. If not a surgical intervention can be considered. Dr. Husni was a friend of mine in Cleveland. I did six of these operations. None of them worked so I quit. So I don't see why the May-Husni procedure would help this man, and if the saphenous thrombosed he'd lose his outflow tract.

DR. PERRIN: Conservative treatment and anticoagulation. I have been very disappointed by saphenotibial bypass. I have performed I would say 20 years ago 25 or something like that, and the results were not good. May in Austria and Chris in Germany have the same poor results.

DR. GARCIA-RINALDI: I'd like to propose to Dr. Dalsing that maybe the approach of taking the entire circumference of the vein has been your problem. We have approached this problem by taking a monocusp patch made of the pulmonary artery of human beings, cryopreserved, and we have implanted this in over 50 cases. They've been followed for up to five years. We have not had a single case of thrombosis even though these patients go home on aspirin, and I would propose that as an alternative to the entire circumference replacement. There seems to be a mechanism not known to me that protects this particular patch from thrombosis if you leave the posterior aspect of the host of vein.

DR. RAJU: I think I am not fully understanding what you are saying.

DR. GARCIA-RINALDI: But I'm not proposing a model. I'm telling you I've done it.

DR. RAJU: I am familiar with your earlier work. I urge you to publish your latest results.

DR. GARCIA-RINALDI: If you take a monocusp and you place it properly, you will get competence. The problem we've seen or the problem we've had in some of these patients is the actual sizing of the prosthesis because we have had to use whatever LifeNet will send us. However, the bioprosthesis comes as a patch, not as a pulmonary artery trunk. So we are implanting a patch, and this we tested to be totally competent at the time of surgery by releasing the proximal clamp and placing it in the common femoral vein.

DR. DALSING: I really have no specific information how synthetic grafts fail. I feel that the problem with cryopreserved tissue is probably some low-grade rejection response. Certainly if you do not aggressively anticoagulate these patients or if the anticoagulation levels drop or if the patient fails to take his medication, the allograft will fail. I know, at least in animal models, that if you place any kind of synthetic in the venous systems that they'll fail. So I'd be interested to see your data because I would expect to see different results.

DR. TRIPATHI: My first question is for Dr. Dalsing. There is a high incidence of incompetence of your cryopreserved vein valve at six months. I also notice that there is no external support for your vein. Have you tried external support and are the results better with external support?

DR. DALSING: We have not tried external support. I can tell you a little bit about what happens to these veins when they fail. It doesn't seem to be a dilation problem. They seem to fibrose. So I think putting an external support around them would probably make little difference.

DR. TRIPATHI: My second question is for Dr. Raju. It has been seen that nearly 30 to 50 percent of all axillary veins are incompetent. Do you always use axillary veins for femoral vein valve transplant or do you look for superficial femoral vein and popliteal veins of the contralateral limb whenever there is a duplication?

DR. RAJU: No, I don't think you should go to the contralateral limb. You are dealing with a disease which is bilateral in a large percentage of cases. Incompetent axillary vein can be repaired before you insert it in large number of cases.

DR. O'DONNELL: Using the external valvuloplasty technique that Bob has developed, we usually take it from the nondominant arm.

DR. TRIPATHI: My last statement is a comment about the iliac angioplasty. Learning from our techniques of iliac angioplasty, especially at the aortoiliac junction, we found that unless you use a kissing balloon technique you are going to decrease the lumen of the opposite side.

DR. RAJU: Not true in the veins.

DR. TRIPATHI: That's what I want to know, whether you are using a kissing balloon technique, and whether you are evaluating the opposite side common iliac vein after doing the ipsilateral common iliac angioplasty, especially when you are advocating putting the stent way up into the IVC?

DR. RAJU: We have not looked at the opposite side in every case. There has been no problem with the opposite side. I don't think the kissing balloon is necessary in the venous system because of lack of rigidity. It's not the same thing as in the artery.

DR. HASANIYA: This question is for Dr. Dalsing. Do you think that the failure rate could be related to other immune process and do you think in your experience that immune suppression might help decrease the failure rate?

DR. DALSING: I can only tell you a little bit about the arterial system where there seems to be cytotoxic T cells that causes much of the problem. So there has been some suggestion that immunosuppressive drugs directed to this T cell population may improve results. I have not had any personal experience with this, but I do believe it is being carefully considered by those involved with cryopreserved tissues.

DR. THORPE: This case represents, I think, a good example of how video phlebography, if you will, could help us understand this patient's problem because by looking at the still images, I can't tell exactly where the stasis is. Is it mostly in the calf, or mostly in the

thigh, or is there any clearance problem through the iliac, as well? Hopefully, in the future, we'll advance towards phlebography with digital imaging. I'd like to ask Dr. Neglen if you've used duplex velocities in the femoral before and after therapy, particularly after stent placement, to assess the flow? This might help you determine whether or not you've got enough stent opening to remain patent.

DR. NEGLEN: Yes. All the patients have duplex ultrasound before and after surgery. Many of them do have decreased augmentation and a lack of respiratory changes before which is abolished by the stenting. However, I think ultrasound is too insensitive a test to detect slight borderline stenosis. For example, the cases of hyperplasia were not detected by ultrasound. They were detected by venogram, and they didn't have any symptoms whatsoever.

DR. GLOVICZKI: Peter, this is a wonderful series that you presented, but the indication seems to be different from our conventional indications of reconstruction for obstruction. I mean, you only had 18 percent of the patients who had greater than two millimeters of mercury pressure difference at best. That to me looks like maybe the circulation in most of these patients is sufficient and you did not really have functional obstruction in most of your patients. So what did you base your indications on?

DR. NEGLEN: Well, as I told you, we're trying to find the actual venous pressures which detect significant stenosis. I think the biggest problem is that in a supine position on an operating or radiology table, it is difficult to increase the venous flow enough to detect significant obstruction. It's a low flow/low pressure area as we talked about yesterday. The question is when you have collaterals, does that mean you have compensated the outflow obstruction, or does it mean that collaterals actually indicate that you have an ongoing outflow obstruction? So these are the kinds of questions we are trying to resolve. Unfortunately, there is no correlation between the intraoperative findings, the preoperative findings pressure-wise, and the postoperative clinical results. Unfortunately, with present diagnostic methods there are patients who have normal pressures in the presence of stenosis, who experience very good results post stenting.

DR. GLOVICZKI: You had ankle-arm pressure measurements? Or did you have any type of outflow obstruction on plethysmography?

DR. NEGLEN: Oh, yes. These patients were fully investigated with ankle-arm pressures and hyperemia pressures and so on. As I showed, the pickup rate for the proximal stenosis by reverse pressure testing, even if they are severe, is not very high. Even though we have been proposing the arm/foot hyperemia test to be the best available test presently, we don't think it is good in all situations, and we're still looking for a better test.

DR. DALSING: I just have one question for Peter too. How do you inject the papaverine?

DR. NEGLEN: Intra-arterially at the level of the femoral artery.

DR. OSMAN: I am one of those unfortunate souls who occasionally have to see 50 patients on a Tuesday afternoon. I have a question to Professor Raju. I may have misheard him, but I understood in this last case that he would have stripped the long saphenous vein which I thought in this particular patient may have made things a lot worse. Did I mishear you or is that the case?

DR. RAJU: No, you didn't misunderstand me. We presented data yesterday to show that you can do it in similar type of situations without any clinical mal sequelae and oftentimes with clinical improvement if there is significant reflux. The paper was in Surgery last year.

SURGICAL MANAGEMENT

DR. KISTNER: We didn't want to operate on the patient, but the patient would not leave without having something done. He said, "My quality of life is so impaired, do whatever you can do. I'm not leaving here until you do something." This led to surgery, even though there were few encouraging findings for a surgical approach. We did have extensive venography of the pelvis and there was nothing abnormal in the iliac vein, so proximal obstruction was not part of the syndrome. We analyzed the case as being severely obstructed in the thigh, compounded by post-thrombotic reflux. Please recall that the patient had a Giacomini vein visualized on the ascending venogram. We disconnected the popliteal termination of the lesser saphenous and moved it down to a more advantageous soft spot on the popliteal vein; this converted the Giacomini vein, which had a valve in it, to an outflow tract for the calf to help the obstruction. We left the greater saphenous vein intact because it was a good outflow tract for the leg. The really non-standard thing we did was to interrupt the profunda femoris vein which was refluxing, and perform an end-to-side transposition between the PFV and the lateral femoral branch because this lateral femoral vein provided a valved outflow for the profunda femoris vein. These procedures were thought to be low risk. The patient post-op felt improved right away. He's now three years post-op. On duplex scan done elsewhere, both of these reconstructions are patent. I spoke to him on the phone this weekend because he lives elsewhere. The patient has returned to hunting. He's back to full-time work. He discarded his pump. He continues to use stockings. At least for three years to this point, he has obtained the result he was seeking.