Venous ulcer diagnosis, treatment, and prevention of recurrences

David L. Gillespie, MD, on behalf of writing group III of the Pacific Vascular Symposium 6,
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The high incidence and prevalence of chronic venous disease has a considerable impact on health care. Advanced venous disease manifested by edema, skin changes such as lipodermatosclerosis (LDS), and leg ulceration affects over 2.5 million patients per year in the United States.[1] Using the CEAP classification the severity of venous disease may be categorized from the most benign form of the disease telengiectasia and reticular veins (C1), through LDS (C4) to the most debilitating form of the disease, chronic venous ulceration (C6).[2] Studies report that as the average venous ulcer requires as long as 6 to 12 months to heal completely and as many as 70% will recur within 5 years of closure.[3] These ulcers are often painful and cause loss of an estimated 2 million workdays because of disability. The cost for treating patients with chronic venous ulcers per year in the United States is estimated to exceed $3 billion per year.[4,5]

The charge of our group was to present current principles in venous ulcer diagnosis and management that will lead to a reduction in the incidence and prevalence of venous ulcers in the future. It will be organized under three major headings of: Diagnosis and Definition of Venous Ulcer; Treatment of Venous Ulcer; and Prevention of Venous Ulcer Recurrence.

DEFINITION, DIAGNOSIS, AND BRIEF PATHOPHYSIOLOGY OF THE VENOUS ULCER

There is no current established official definition of a venous ulcer, yet we know that venous ulcers constitute the largest segment of leg ulcers, thought to comprise 50% to 70% of cases. These estimates are currently agreed upon, even in the absence of agreement about the elements needed to classify an ulcer as of venous origin in whole or in part.

According to the revised CEAP classification of chronic venous disease published in 2004, a venous ulcer is defined as a full-thickness defect of skin, most frequently in ankle region, that fails to heal spontaneously and is sustained by chronic venous disease.[6]

The currently proposed initiative plans to decrease venous ulcers by 50% in 10 years. This proposed ulcer reduction plan will utilize the following working diagnosis of a venous leg ulceration: A full thickness defect of the skin (1) located in the lower leg, typically with pigmentation and/or skin changes; (2) presence or history of venous disease: documented history of DVT, or documented axial venous reflux or deep vein obstruction; and (3) absence of another condition that could be the essential cause of the ulcer.

Systematically, we can summarize the definition of a venous ulcer as a defect in the skin with surrounding pigmentation and dermatitis, located in the lower leg (usually in the gaiter region) that has been present for greater than 30 days, characterized by persistent venous hypertension and abnormal venous function (result of venous reflux and/or obstruction confirmed by hemodynamic and/or physiologic assessment), without a primary or associated arterial, immunologic, endocrine, or systemic cause. It is recognized that ulcers can be caused purely by venous pathology such as venous reflux or obstruction. When these abnormalities are combined with additional pathologic conditions, they contribute to the causation and perpetuation of the ulcer. The latter situation includes comorbid conditions such as arterial ischemia, swelling and lymphedema, trauma, autoimmune disorders, neurotrophic conditions, and diabetic vascular disease.[7] These ulcers are categorized as mixed origin, in which the venous component may or may not play a dominant role. Successful treatment of such ulcers includes not only the venous component but also concomitant management of the comorbid condition.

A diagnosis of venous reflux or obstruction must be established by an objective test beyond the routine clinical examination of the extremity. Many conditions result in discoloration of the skin. Tissue changes alone such as those seen in a classical "post-phlebitic" appearance are not adequate for a diagnosis without definitive documentation of reflux or obstruction in the veins of the extremity. Currently, duplex scanning, which is widely available, is considered the standard test of choice.

Further complicating the situation is the wide practice variation and levels of expertise of those individuals performing the duplex scans within the United States. The scan required for the diagnosis of venous hemodynamic abnormalities requires a complete assessment of the venous

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Correspondence: David L. Gillespie, MD, Program Director, Vascular Surgery, Integrated Residency and Fellowship, Professor of Surgery, Division of Vascular Surgery, University of Rochester, School of Medicine and Dentistry, Rochester, NY 14642.
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segments for reflux in addition to the compression evaluation, standard for diagnosis of venous thrombosis. This "extended" scan of venous reflux is time consuming and not practiced routinely in hospital and non-hospital based vascular laboratories in this country for a variety of reasons. To begin, the time needed and incremental cost for the additional reflux examination are barriers to widespread adoption. In addition, there is a lack of demand from the medical profession for this additional reflux information. The need for a "standardized chronic venous scan" that includes analysis of the extremity for axial venous reflux is a practical problem that must be solved before a 50% reduction in venous ulcers can be achieved.

Complete duplex examinations of lower extremities with venous disease have been widely reported in patients with venous ulceration. These studies have shown that the distribution and extent of reflux is strongly associated with the clinical severity of chronic venous insufficiency (CVI). Distal venous reflux is present in at least 80% of the symptomatic limbs. Patients with venous ulcers exhibit involvement of the superficial veins in 90% or more of cases, perforator involvement in 80% or more, and deep vein involvement in 70% or more.[8-18] Of note, deep venous thrombosis (DVT) is not a prerequisite for the development of skin changes or ulceration. Labropoulos et al found that 86% (37/43) of patients with venous ulcers have some degree of reflux in the local area, the pattern of which may differ from the axial vein disease and in support of the concept that treatment of the local hemodynamic abnormalities is an important factor in the healing of the ulcers and in the prevention of their recurrence.[9]

To date, there are no effective therapies preventing skin fibrosis or identifying patients who may be at risk for developing venous ulcers. Early identification of patients at risk for fibrosis associated with venous insufficiency may lead to therapies that could prevent the progression of benign disease to lipodermatosclerosis (LDS) and non-healing ulceration. A potential clue in the pathogenesis of progression to LDS and non-healing ulceration can be found in research evaluating the important regulators of tissue remodeling.

One important factor identified is transforming growth factor-β (TGF-β), which has been widely studied in wound healing. The expression of both the type I and type II TGF-β receptors has been shown to be elevated in acute wounds.[19] In addition in the acute wound, TGF-β has been shown to stimulate the synthesis of extracellular matrix proteins[20] and suppress their degradation by decreasing protease expression.[21] These regulatory mechanisms allow for normal wound healing to occur. Conversely, venous ulcer fibroblasts demonstrate a lack of viable TGF-β receptor complexes, which may contribute to the chronicity of these wounds.[22]

Various studies have implied that there are abnormalities in the TGF-β signaling pathway in chronic venous ulcerations.[23-27] The degree of skin hardening or fibrosis associated with LDS is proposed to relate directly to skin breakdown and venous ulcer formation as well as to a subsequent delay in ulcer healing.[19] In fibroblasts derived from patients with C6 disease, there is a lack of type II TGF-β receptor present in ulcer biopsies.[22] Interestingly, TGF-β-RII is observed in the fibroblasts and inflammatory cells surrounding the ulcer. Healing ulcers were shown to express both type I and type II receptors suggesting that the presence of a viable receptor complex is necessary for healing. These results, however, have not been correlated with the clinical data found in patients with C6 disease. Most recently, analyses of fibroblasts from chronic wounds have shown alterations in the TGF-β signaling pathway and decreased TGF-β-RII expression.[28] Furthermore, venous ulcer fibroblasts have been shown to be unresponsive with respect to collagen synthesis.[29]

Recommendations and actions regarding the diagnosis of a venous ulcer

For more patients to receive an accurate diagnosis and effective treatment for venous ulcerations, an increase in clinician education is required. The educational process should be directed to clinicians who treat patients as they access the health care system, such as primary care clinics, nurse practitioner, and physician assistant run centers, and even the emergency room. An initial diagnostic workup for a patient with a leg ulcer should include a venous duplex scan and an arterial evaluation (ABI) to ensure that compression therapy can safely be employed. The importance of early and accurate venous duplex scanning when skin changes or ulcers first occur is fundamental to prompt treatment and limitation of dense scarring of the lower leg tissues. Both of these tests should be performed by a qualified diagnostic vascular laboratory (eg, ICVAL accredited). The clinical history should focus on signs and symptoms that elicit a venous cause of the ulcer. These criteria may include leg heaviness and cramps, swelling, leg pain after ambulating that is relieved with rest and elevation, and a history of deep venous thrombosis.

The role of the primary care physician in this process is critical because the treatment of early skin changes or ulcers increase the chance to preserve the tissues of the lower leg. A major educational program is needed to enroll primary care clinicians as key members of the team to reduce venous ulcers through recognition of these early cases. The educational message to primary providers is that chronic venous disease is a lifelong progressive degenerative process, much like peripheral arterial disease. Effective management prior to irreversible skin changes will prevent marked disability in the patient's later life.

A call to action for all primary care providers to perform the necessary venous duplex scans and classify leg ulcers as venous, nonvenous, or mixed etiology is sorely needed. Effective management in the early stages of chronic venous insufficiency involves more than just getting the ulcer to heal. It requires that a cause of the ulcer is established, and the necessary steps to eliminate or control venous reflux or obstruction are taken. The primary care provider should refer patients to a center where a definitive diagnosis and proper treatment can be made if they lack the expertise.

Potential sites that currently offer this type of organized care includes wound care clinics and a growing field of venous specialists.

A strong public awareness campaign is also needed to alert patients with progressive venous disease to contact their physician for diagnostic evaluation and definitive care. Too often, the patient's early inquiry may be overlooked by the physician as a minor problem that can delay definitive evaluation and preventive management.

Specific means to improve the diagnosis of venous ulcer

The need for development of standardized diagnostic evaluations and increased education for primary care providers throughout the USA will take several years. This proposal is setting a goal for achieving the initial diagnosis by venous duplex scan within 1 week of presentation by 50% of wound practitioners in 3 years. The 5-year goal increases this benchmark to 75% of practitioners diagnosing venous disease through duplex scanning within the first week of presentation. The methods for achieving this include a major program of awareness, utilizing medical resources from wound care clinic organizations, Health Maintenance Organizations, the Veterans Administration, Medicare, and intense efforts from medical societies.

The adoption and dissemination of this information could be fostered through the creation of a patient "venous ulcer card" that documents the ABI (or presence of arterial pedal pulses) and the presence of reflux or obstruction in the leg/pelvic veins. This could extend to a Website documentation form and other mechanisms to store and disseminate the data.

Monitoring progress of this project over the next decade is paramount but is challenging. Recording, on a national scale, the number of leg ulcers referred for venous testing, the results of the testing categorized for pure and mixed venous ulcers according to a single definition, monitoring the time to referral for venous testing, and the numbers of recurrent ulcerations would be ideal. This might be possible as data abstraction improves and as more and more hospital systems engage the electronic medical record. The progression of venous disease could be captured according to a definitive diagnosis, and the results of various treatments could ultimately be compared.

TREATMENT OF VENOUS ULCERS

While the primary aim for the treatment of venous ulcers requires a paradigm change from healing to one of prevention, the discussion in this section will be restricted to appropriate therapy targeted to ulcer healing. Definitive care of the ulcer patient now needs to be extended from just healing the ulcer to correction of treatable venous reflux and obstructive states and to ongoing surveillance of those whose disease is not correctable.

The literature on venous ulceration has focused largely upon means to reverse the inflammatory changes around the ulcer and achieve epithelial covering of the ulcer defect. The vital importance of compression to eliminate swelling and reverse the derangement of the microcirculation is well recognized by many informed physicians but poorly practiced across the spectrum of primary care providers. It is not uncommon to encounter patients who have been under a physician's care for persistent venous ulceration for weeks, months, and even years using a variety of therapies, but without objective testing for venous disease or treatment with any compression modality.

Judging from experience because accurate reports are not available, it could well be that over half of patients with venous ulcers in the general public are managed by their physician without the use of compression. The reason for this is failure to disseminate basic knowledge about the effects of swelling and venous hypertension exists through our entire system of medical education. The majority of medical schools do not include lectures on this subject in their curriculum. The same is true for postgraduate training programs in general medicine, and for qualifying examinations in general and specialty medicine, in spite of the occurrence of chronic venous disease in over 20% of the adult population. Thus, the busy practitioner is likely to be uneducated about chronic venous disease and poorly equipped to provide proper care for patients with VU.

The importance of reducing edema is central to the appropriate management of all lesions of the distal lower extremity in the erect human being. For the venous ulcer patient, it is absolutely vital that lower leg and ankle swelling be controlled. Elimination of swelling is a prerequisite for the effective use of all other medications including antibiotics and both medicinal and physical applications to the wound surface in the ambulant care of the venous ulcer.[30,31]

The management of venous ulceration has progressed from recommendations for prolonged bed rest and elevation of the leg to ambulatory treatment using effective support. The principles of effective compression and active ambulation have been advanced through the studies of Partsch, Stemmer, and others in Europe, and the developments of the stocking and bandaging industries.[32-34] Evidence-based guidelines exist for the treatment of venous ulcers.[35, 36] The Cochrane Collaboration review of 39 randomized controlled trials examining various forms of compression in venous leg ulcers concluded that compression clearly increases ulcer healing rates compared with no compression.[37] Multicomponent systems are more effective than single-component systems, and most studies found that multicomponent systems with an elastic bandage were more effective than those composed mainly of inelastic components.

Compliance with compression is difficult to assess in long-term follow-up. According to a survey including a large cohort of patients under the care of primary care physicians and specialists, 63% of patients did not use the compression stockings prescribed, with no differences between the C0-C2 and the C3-C6 group.[38]

The role of various medications and applications for the wound surface is beyond the scope of this document. Proper wound bed preparation, including controlling bac-

terial colonization, is important in optimizing the wound to facilitate the healing process.[39] However, topical therapies are usually unsuccessful in stimulating wound healing without definitive care of the underlying venous abnormality, and concomitant compression.

The role that correction of superficial reflux and iliac venous obstruction plays in promoting ulcer healing remains to be defined. Prospective, randomized studies have not confirmed that venous intervention will result in faster ulcer healing, but clearly show that ulcers treated in this way recur less often.[30,40-42] However, we believe that evaluation of the iliac venous system should be considered if persistent ulcer and edema exist, despite correcting superficial venous reflux and compliance with compression garments.[43] In addition to superficial and iliac vein disease, other sources of primary axial reflux include incompetent perforator veins, focal reflux through the small saphenous vein, and deep axial reflux with focal incompetence through local perforator veins.

Studies are not available to show whether corrective venous procedures should be done while the ulcer is active or delayed until the ulcer heals. Logically, the optimal time to intervene would be as early in the course of chronic venous disease as one can determine that the extremity is at risk for the development of advanced leg tissue changes. In this way, the integrity of the soft tissue in the lower leg could be preserved. For the present, it can only be said that surgical intervention to correct treatable reflux and obstruction is essential to decrease the overall incidence of VUs.

The problem of ulcers related to postthrombotic syndrome (PTS) is more complicated and will be addressed in a separate manuscript later in this issue. There are those with combinations of PTS and superficial primary reflux disease where elimination of the superficial reflux is of great benefit in limiting progression of the subcutaneous soft tissue changes.[44] For PTS, level 1 evidence exists for the benefit of long-term use of graduated support in decreasing recurrences of the ulcer and in providing patient comfort.[30,32]

While not able to be addressed easily because of multiple socioeconomic reasons, many venous ulcer patients are infirm, live alone, and are often morbidly obese without adequate support systems. It seems that a disproportionate percent of CVU patients become wards of the community because their health habits are chronically deficient, their diets often result in obesity, and their access to informed medical care early in the course of the venous disease has been limited. For these, programs to heighten awareness and promote education of the medical and para-medical professions are the best hope within our present medical system.

Recommendations and actions for treatment of venous ulcer

(1) When an ulcer patient is initially seen by a primary care provider, an accurate diagnosis is the most important first step. This may require referral to a source such as a wound care clinic or a venous-schooled practitioner who will obtain the needed venous and arterial testing and institute immediate application of effective compression therapy. Specifically, an accurately diagnosed VU case to receive effective compression treatment within the first week is the goal. This is necessary to eliminate swelling in the extremity because all other modalities are largely ineffective until the swelling is corrected. It is equally important to diagnose cases where arterial insufficiency coexists with venous disease complicating treatment due to the risk of compression therapy in this patient group. These patients should be referred initially to specialist centers for management.

(2) Specific correction of treatable sources of axial reflux and deep vein obstruction are critical to the control of venous hypertension and its long-term destructive effects upon the soft tissue of the lower leg. Early referral to a venous specialist is advised if the practitioner is not capable of more advanced management.

(3) Active cooperation with the wound care clinics is needed to adjust treatment algorithms for early diagnosis and definitive treatment of venous ulcerations. Wound care clinics, along with the movement to develop informed specialists in venous diseases, are the best sources for unifying a definitive approach to venous ulcer management.

(4) A strong educational program is needed to inform primary care providers about their role in guiding the patient with his/her first venous ulcer to achieve an accurate diagnosis. Providing this initial effort, definitive care can be instituted before the lower leg tissues become irretrievably scarred. Programs to improve treatment of VU patients could include web-based guidelines about VU treatment and risks of recurrence of ulcer to heighten awareness, programs with CME accreditation for physicians at various levels, incorporation of questions about chronic venous disease on board certification examinations for primary care physicians, and residency review committee physician training documentation.

(5) Major programs of patient and patient's family awareness are needed to treat the venous ulcer case early and effectively, and to prevent the chronic progression of the venous hypertension to the level that results in lower leg tissue scarring and ulceration effects.

PREVENTION OF ULCER RECURRENCE

The primary goal after a VU has healed should be the avoidance of recurrence. Chronic venous patients are prone to develop new sites of involvement over time, and the ongoing venous dysfunction tends to become more destructive to the lower leg tissues as aging occurs in the erect human. Progression from asymptomatic varicose veins to severe lipodermatosclerosis is estimated to occur in about 20% of cases. The aim of treatment in all patients needs to focus on preventing the gradual progression of the venous hypertension to the point of lower leg tissue scarring with

advanced skin and ulcer effects. In the case of the VU, in contrast to other types of leg ulcer, healing of the ulcerated skin defect does very little to prevent a new ulcer from forming if the underlying cause for the ulcer remains intact. For example, a VU is much more likely to recur if the underlying reflux or obstruction is not treated and/or the patient is noncompliant with compression therapy. Ulcer recurrence is a huge problem for which effective remedies are available for a significant number of cases, but not for all.

As stated in the prior section, the ideal treatment for venous ulceration is to remove or repair the veins with reflux or obstruction so the underlying cause of the ulcer ceases to exist. This will also significantly decrease the recurrence rate. When the ulcer is first diagnosed, it will be found to be due to either primary reflux disease or post-thrombotic deep vein obstruction/reflux disease. In the case of primary reflux, the venous abnormality is nearly always correctable in whole or in part by superficial venous ablation of the routes of axial reflux. When this is done, the pathologic process is eliminated or decreased, and the venous physiology is returned to a more normal state. Since it is established that 40% to 70% of VUs are secondary to primary reflux disease, it is clear that this treatment alone will result in marked reduction of venous ulcers.[45] These patients should be referred initially to specialist centers for management. The frequency of recurrent ulcer will be markedly reduced by this treatment and that is superior to treatment by compression alone.[30,41,42]

The rate of VU recurrence has been found by some to depend upon the degree of compliance with compression treatment, but has also been reported to vary with the etiology of the disease, the extent of deep vein involvement, the type of therapy, and the duration of follow-up.[36,46-50] Current evidence suggests that recurrence of venous ulceration is decreased by ablation of reflux in the superficial venous system more effectively than compression alone.[36,40,42,51-53] For example, the beneficial effect of venous surgery over compression alone comes from Gohel et al [36] in a study where 500 patients with open or recently healed leg ulcers and superficial venous reflux were randomized to either compression treatment alone or in combination with superficial venous surgery. Twenty-four-week healing rates were similar in both groups (65% vs 65%, hazard 0·84 [95% CI 0·77 to 1·24]; $P = .85$), but 12-month ulcer recurrence rates were reduced in the compression and surgery group (12% vs 28%, hazard −2·76 [95% CI −1·78 to −4·27]; $P < .0001$). Longer follow-up of the same patient series reported similar superiority at 3 years when the recurrences had mounted to 56% for those who had only compression compared to 31% recurrence when surgical treatment was added.

The availability of this treatment may present the best single opportunity for reduction of venous ulcers, as evidenced by the successful Swedish report of VU reduction by Nelzen et al.[45,54] A protocol that identifies patients with superficial reflux among the VU population and treats them effectively could reduce the incidence of VU by a significant portion of the 50% goal. In fact, it is probable that the goal to decrease the incidence of VU cannot succeed until the several routes of reflux are routinely identified by accurate ultrasound diagnosis and effectively treated in the community setting.

The technique of venous ablation is not critical as long as the venous reflux is addressed. In the prospective randomized study, Zamboni et al treated 47 legs with venous ulcers with either superficial venous surgery, using a minimally invasive surgical hemodynamic correction of reflux (CHIVA) or with compression.[42] All patients had primary superficial vein incompetence. Patients with PTS, deep vein reflux or obstruction, or those with excessive ulcers (>12 cm) were excluded. At 3 years, healing was 100% (31 days) in the surgical and 96% (63 days) in the compression group ($P < .02$). The recurrence rate was 9% in the surgical and 38% in the compression group ($P < .05$).

Elimination of other routes of axial superficial and local perforator reflux in primary disease is also readily done by minimally invasive procedures through accurate noninvasive ultrasound tracing of reflux patterns in the ulcer patients. These include small saphenous reflux, focal perforator reflux serving the ulcer bed, especially when accompanied by axial deep reflux, and various cases of recurrent ulceration after prior reflux ablations have developed new sites of reflux. Patients with combined superficial and deep axial reflux are candidates in selected instances for elimination of the superficial axial reflux combined with ongoing use of external support to decrease the incidence of recurrent ulceration.

Recommendations and actions to prevent ulcer recurrence

(1) Compression, correction, and surveillance are the three key actions to effective prevention of ulcer recurrence. Compression is needed in virtually all cases of VU at the beginning of treatment and continuing as long as the risk exists for recurrence. Correction of venous reflux and obstruction is important because it eliminates the basic cause of the sustained venous hypertension and may effectively cure the problem. Surveillance refers to ongoing follow up of the VU patient to detect new sites of reflux or thrombosis, and to prompt treatment of new problems before they cause tissue damage. Long-term maintenance of effective external compression is needed for all who have swelling in the legs.

(2) The VU case does not end with epithelialization of the wound. Educating the primary and specialty physicians that the VU patient has a chronic disease that needs a program of ongoing external compression and surveillance against recurrent reflux or thrombosis.

(3) Education is needed in the medical profession and in the public perception of chronic venous disease. Two widely held and seriously misleading concepts are that chronic venous disease is not treatable and ulcers are not preventable. These need to be corrected by the understanding that chronic venous disease is readily

diagnosed by simple, affordable, noninvasive ultrasound scanning and that a large proportion of the ulcer cases can be effectively treated. Chronic venous disease has to be understood to be a lifelong degenerative condition whose management belongs in the responsibility of the primary physician as well as the interventional specialist.

Means to prevent recurrence of venous ulcer

(1) Creation of a guideline for fundamental change in the responsibility of those who treat VU patients to include prevention of ulcer recurrence along with healing of the skin ulcer defect. Early referral to a venous specialist is advised if the practitioner is not capable of more advanced management
This would embrace the following principles of treatment:
   (a) Correct axial superficial reflux and other readily correctable sources of reflux and obstruction in all ulcer cases where practical during the treatment phase of the first ulcer.
   (b) Maintain effective compression therapy on the ulcerated extremity as long as the underlying cause of the ulcer remains uncorrected and as long as swelling occurs in the dependent leg.
   (c) Manage the VU patient as any other chronic debilitating disorder by ongoing surveillance as long as underlying reflux and obstruction persists in the extremity.

(2) Develop cooperation with the wound care clinics of the United States to incorporate accurate venous reflux scans in all leg ulcer patients that are suspect for a venous etiology, and add correction of the treatable sources of venous reflux and obstruction in their treatment algorithm.

(3) Develop guidelines for ongoing surveillance that apply to all who care for patients with chronic venous disease and especially for patients with venous ulceration. These guidelines should embrace correction of treatable venous reflux and obstruction, and ongoing effective compression for PTS.

(4) Develop an adequately powered research program that will provide high-quality evidence to confirm or deny that correction of axial superficial reflux (C2 disease), and perhaps other specific sources of ulcer development, results in delay or prevention of advanced venous (C4-C6) disease.

(5) Incorporate the primary care physician in the early diagnosis and effective management of chronic venous disease. Chronic venous disease should be viewed by the medical profession as a lifelong progressive disorder with the threat of becoming a debilitating disorder in its later stages for a significant proportion (20%) of cases. As such, it requires the attention of the primary care physician to diagnose its presence and manage its early stages.

BENCHMARK MEASURES AND TIMELINE OF SUCCESS FOR VENOUS ULCER HEALING AND RECURRENCE

(1) A 50% compliance with early employment of compression therapy in VU care in 2 years, and 75% in 5 years.

(2) Guidelines for diagnosis of VU via chronic duplex scan with compliance of 80% in wound clinics and specialty venous clinics in 3 years, and 100% in 5 years.

(3) A 50% compliance by wound care clinics and venous specialty clinics with interventional treatment to eliminate superficial venous reflux and other selective sources of focal axial reflux in 3 years, and 80% compliance in 5 years.

(4) Awareness programs for the public and educational programs for the health care profession within 1 year.

(5) Adoption of the concept that chronic venous disease is a lifelong progressive degenerative disorder by 50% of primary care medical societies in 5 years.

PVS6 writing group III members: David Gillespie, MD, Bob Kistner, MD, Carolyn Glass, MD, Brad Bailey, MD, Arun Chopra, FICA, Bill Ennis, MD, Bill Marston, MD, Elna Masuda, MD, Greg Moneta, MD, Olle Nelzen, MD, PhD, Joe Raffetto, MD, Seshadri Raju, MD, Suresh Vedantham, MD, David Wright, MD, Vincent Falanga, MD.

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