A PROTOCOL FOR THE TREATMENT OF VENOUS ULCERS IN PRIMARY CARE

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A PROTOCOL FOR THE TREATMENT OF VENOUS ULCERS IN PRIMARY CARE

By

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ABSTRACT

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By

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Venous ulcers are chronic and recurrent problems that are most often diagnosed and treated in primary care settings. Advanced Practice Nurses working in primary care settings can be key players in assisting clients/patients with venous ulcers and their caregivers to participate in self-care behaviors for the treatment and prevention of these ulcers. A treatment protocol derived from research based literature can promote achievement of optimal outcomes if it includes mutually set objectives and treatment, with comprehensive client education to promote self-care behaviors.

ACKNOWLEDGMENTS

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INTRODUCTION

Leg ulcers negatively impact the lives of hundreds of thousands of Americans and will occur in one to two percent of the adult United States population during their lifetimes (Levine, 1990; Margolis & Cohen, 1994; Phillips & Dover, 1991; Seeley, 1992; Spittell, 1993). The reported prevalence varies from 400,000 to more than one and one-half million people. Women develop leg ulcers three times more than men (Callum, Harper, Dale & Ruckley, 1985; Margolis & Cohen,1994).

There are many different types of leg ulcers. Venous ulcers (VUs) account for 70-90%, arterial ulcers 5-10%, combinations of venous and arterial ulcers 10-21% , and neuropathic (usually diabetic) ulcers make up most of the remaining types (Harris, Brown-Etris & Troyer-Caudle, 1996; Levine, 1990; Nelzen, Bergqvist & Lindhagen, 1991; Phillips & Dover, 1991; Seeley, 1992; Trott, 1992; Zink, Rousseau & Halloway, 1992; Wilson, 1989). Eighty-five percent of all individuals with leg ulcers are over the age of 65 (Callam, Harper, Dale & Ruckley, 1985; Capeheart, 1996; Johnson, 1995; Jones & Millman, 1990; Mostow, 1994; Nelzen-Hallbrook, 1991; Phillips & Dover, 1991; Seeley, 1992). As the elderly population increases, there should be a proportionate increase in the incidence of VUs.

Health care costs in the treatment of leg ulcers are estimated at between $570 million and $1.14 billion each year (Cherry, Ryan & Cameron, 1991; Harris et al. , 1996; Sieggreen & Maklebust, 1996). In addition to these expenses, two million work days are lost annually as a result of employed

individuals with ulcers who are receiving health care treatment. In the United States, from 1990-92, there were more than 1.3 million outpatient visits for VU treatment (Margolis & Cohen, 1994). For purposes of this scholarly project, the following information will relate only to VUs.

VUs are chronic and recurrent, with reported recurrence rates anywhere from 30-90% (Black, 1995; Harris et al. , 1996; Levine, 1990; Monk & Sarkany, 1982; Sieggreen & Maklebust, 1996). Many VUs do not heal for months or years even if correctly diagnosed and treated. It is known that the VUs of compliant patients heal significantly sooner and are less likely to recur (Erickson, Lanza, Karp, Edwards, Seabrook, Cambria, Freischlag & Towne, 1995).

By the year 2000, it is projected that 70% of the population will be enrolled in some form of managed care program as a means of controlling health care costs (Ennis & Meneses, 1996). The focus for the 90's is on controlling outpatient costs via referral authorization, drug formularies, provider incentives, and provider networks. Most VU patients can be managed cost-effectively in the primary care setting utilizing treatment protocols. Protocols must be compatible with current organizational resources, values and provider practice patterns (Ennis & Meneses, 1996).

Consumers have been trying to increase participation and input into the health care delivery system as a means of coping with declining economic trends and perceived powerlessness in influencing health care policy decisions. With increased input, consumers are better able to engage in self-care resulting in more control and responsibility.

THE PROBLEM AND PURPOSE

Statement of the Problem

VUs are chronic, debilitating, expensive and recurrent. Treatment is time consuming and expensive for the individual and the health care system. Leg ulcer patients tend to be treated by health care providers in a variety of specialties potentially resulting in a multitude of interventions with duplication of services (Merli, Robinson, Spandorfer & Paluzzi, 1994).

Variations in VU diagnosis and treatment result from fragmentation of care and often result in expensive choices producing less than desirable outcomes (McGuckin, Stineman, Goin & Williams, 1996). Emotions, personal experience, and access to technology all factor into clinical decisions, often without much scientific support (Ennis & Meneses, 1996).

Purpose

No comprehensive, up-to-date, inclusive protocols were found in the health care literature addressing VU assessment, diagnosis and treatment in the primary care setting with an emphasis on self-care. The purpose of this scholarly project is to develop a protocol for the treatment of VUs for use by health care providers in the primary care setting, based on Orem's Self-Care Theory. The protocol promotes optimal outcome achievement, compliance and satisfaction utilizing research based VU treatment and prevention interventions, mutuality, education, and self-care behaviors.

Clinical practice protocols outline step by step, comprehensive clinical processes to deliver care to a target population (Gawlinski, 1995). A protocol is more comprehensive than a policy, procedure, or guideline. It contains everything that needs to be implemented to achieve desired outcomes and is a prescription for clinical decision making (Nunnellee, 1996).

Orem's Self-Care Theory fits into the development of a primary care protocol for treatment of VUs related to its primary focus on the client/patient experiencing the health care problem. The following section contains pertinent information related to Orem's theory and how it was utilized in the development of the VU Treatment Protocol.

CONCEPTUAL FRAMEWORK

The basic concepts of Orem's Self-Care Theory pertinent to this project are self-care, self-care agency, therapeutic self-care demand and nursing agency. When an individual's therapeutic self-care demand is greater than the self-care agency, a potential or actual self-care deficit exists requiring nursing agency (Wesley, 1995).

Self-care includes those learned, purposeful, independent activities performed by an individual to promote and maintain life, health and well-being throughout life and to recover from or cope with a disease or injury (Chinn & Kramer, 1991; Greenfield, 1985; Orem, 1991; Spitzer, Tar-Tal & Ziv, 1996). Orem (1991) determined that all individuals have some ability for self-care. Self-care is a specialized form of human functioning that depends on basic individual capacities which can be altered by many variables such as age, various disabilities, and pain. (Spitzer et al., 1996).

The self-care agency is an individual's ability to perform necessary self-care activities. There are two agents: the self-care agent who performs the self-care and the dependent care agent who performs the required care for an impaired individual (Wesley, 1995). Chang (1980) defined the self-care agent as an individual who assumes responsibility for his/her own health care, such as recognizing symptoms and managing selected health deviations. The individual characteristics of the self-care agent must be considered since they influence needs, perceptions, response to illness, health care, and treatment (Orem, 1991). These characteristics include age, gender, education level,

occupation, marital status, support network, developmental stage individual beliefs, expectations and attitudes (Chang, 1980).

The therapeutic self-care demand involves self-care actions to maintain health, well-being, or to promote development (Orem, 1991; Wesley, 1995). This demand varies throughout one's lifetime as variables change. The client with a health deviation must adapt and make changes in order to continue to meet his/her self-care requisites to restore balance.

The nursing agency involves the complex actions a professional nurse takes to meet a patient's self-care needs when a self-care deficit exists (Wesley, 1995). Nursing agency is composed of three systems: wholly compensatory, partly compensatory, and supportive-educative, and is determined by the patient's self-care needs and self-care agency. Wholly compensatory nursing actions involve total care of all known care needs when the self-care agent is totally unable to exercise any self care. Partially compensatory nursing actions involve some, but not all, care requisites secondary to the self-care agency requiring some assistance. Supportive-educative nursing actions involve provision of knowledge, skills and support to the self-care agency who is able to perform all required self-care needs (Wesley, 1995).

The defined concepts, as relevant to this scholarly project, are seen in Figure 1. A relationship exists between all Orem concepts and these relationships are depicted by arrows going in both directions.

Orem (1991) postulates that the role of the nursing agency is to move the client toward his/her highest level of self-care with goals and content for self-care education determined by the self-care agency learner. Self-care is the product of negotiation between the self-care agency and the nursing agency based on the patient's perceived needs, preferences, values and goals (Orem, 1991). Self-care is derived from patients' perceived needs and preferences

OREM'S SELF CARE THEORY OF NURSING

AS APPLIED TO VENOUS ULCER TREATMENT

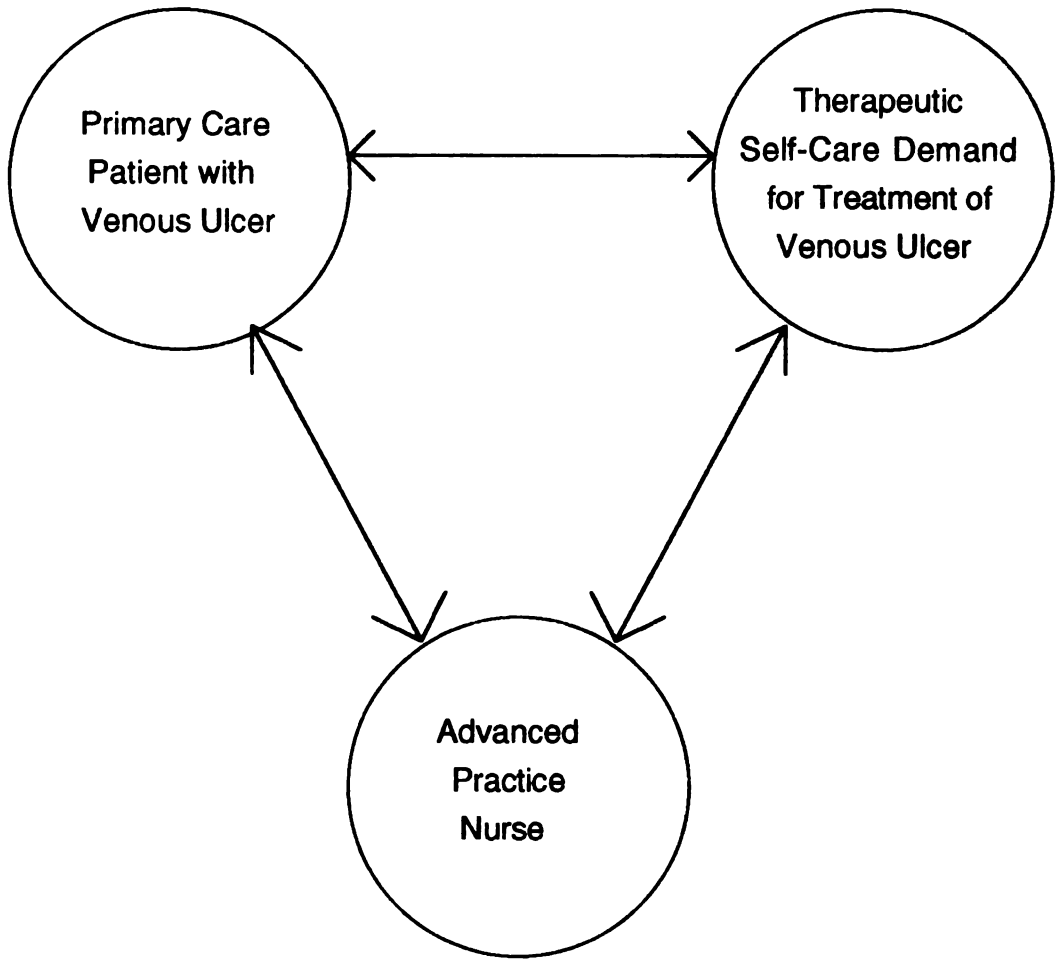


Figure 1. Adapted from Nursing Theories and Models (2nd ed), p. 79 by R. L. Wesley, Springhouse, PA: Springhouse Corporation. Copyright 1995 by Springhouse Corporation.

regardless of whether such needs and preferences conform to professional perceptions of patients needs (Levin, 1978). The self-care agency and nursing agencies are on equal ground in a true, equal partnership (Barofsky, 1978).

A chronic VU influences quality of life, including energy levels, emotional status and social activity (Lindholm, 1995). The self-care agency may be limited in its ability to provide VU self-care secondary to a lack of orientation, resources, knowledge, pain, extremity edema, motivation, dexterity, exhaustion, or a host of other variables. If this is so, a health-deviation self-care deficit exists, and appropriate, individualized nursing agency intervention is necessary.

Figure 2 depicts this authors adaptation of Orem's Self-Care Theory to VU treatment in the primary care setting. The nursing agency is the Advanced Practice Nurse (APN) who assesses, diagnoses, treats and provides self-care education for the primary care patient presenting with a VU (self-care agent). The dependent care agency is the identified care giver who may need to function as the provider of the self-care treatment and may be the recipient of the self-care education.

The therapeutic self-care demand is the need for VU treatment involving education from the APN. The mutual relationship developed in the primary care setting leads to empowerment and self-efficacy, thus increasing quality of life, prevention of VU recurrences and satisfaction with the health care system.

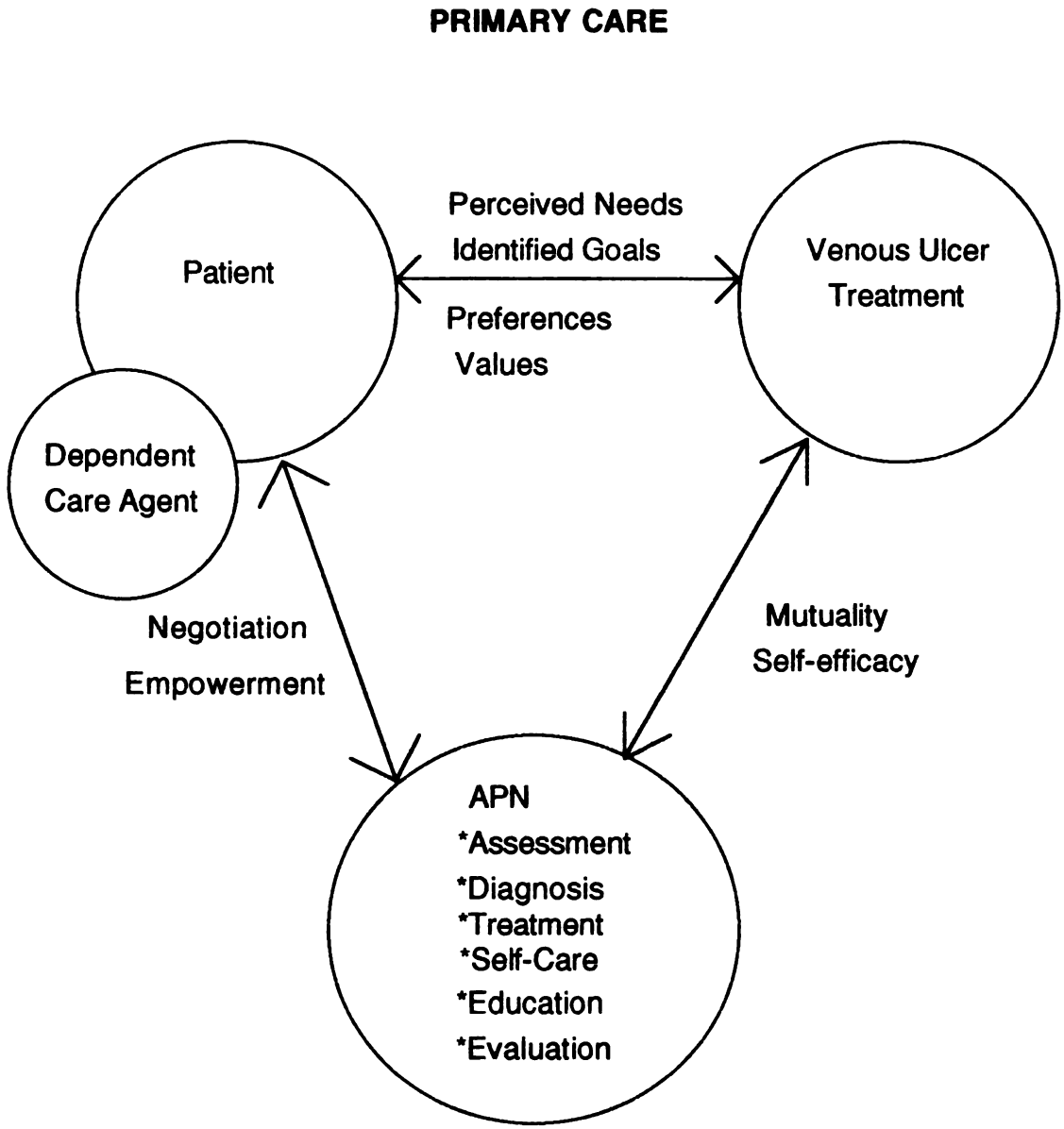


Figure 2. Adapted from Nursing Theories and Models (2nd ed), p. 79, by R. L. Wesley, Springhouse, PA: Springhouse Corporation. Copyright 1995 by Springhouse Corporation.

REVIEW OF LITERATURE

The following section contains information necessary for the health care provider to understand in order to efficiently and effectively educate the health care recipient in self-care treatment for VUs. A basic understanding of anatomy and physiology must be the foundation for advanced knowledge in relation to treatment of VUs.

Anatomy and Physiology

Three distinct groups or systems of veins comprise the venous system of the lower extremities: deep, superficial and perforating. Each is separated by fascial layers, but is directly or indirectly connected. Deep veins (tibial, popliteal and femoral), located beneath leg muscle, carry most of the blood returning from the legs. Superficial veins (greater and lesser saphenous), located in subcutaneous tissue (where there is little structural support), carry the remaining returning blood. Figures 3 and 4 depict superficial veins of the lower extremities. The critical perforating veins, located intermittently between the superficial and deep venous system from the ankle to the knee, allow communication of deep and superficial veins (Black, 1995). The 90 to 200 perforating veins in the lower extremities are small and inconsistent in location (Moore, 1998).

Each leg vein contains multiple one-way, flutter or semi-lunar valves designed to keep returning venous blood from backflowing down the leg while muscle contraction (calf pump) forces the venous blood up the leg, against gravity, and toward the heart. The greater saphenous vein possesses 10-20

Superficial Nerves and Veins of Lower Limb: Anterior View

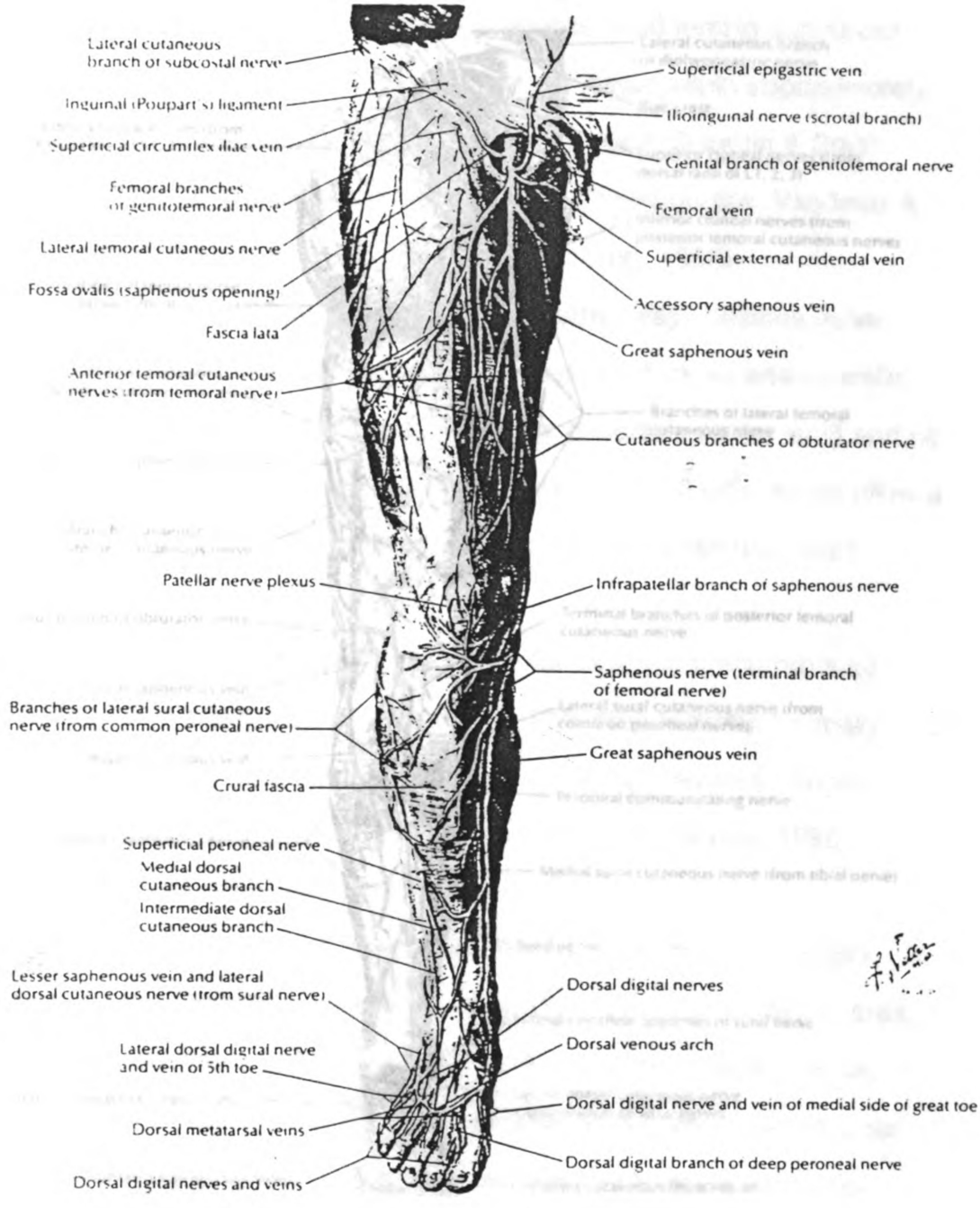


Figure 3. From Atlas of human anatomy by F. H. Netter. Copyright 1989 by Ciba-Geigy Corporation, Pharmaceuticals Division. Reprinted with permission.

Superficial Nerves and Veins of Lower Limb: Posterior View

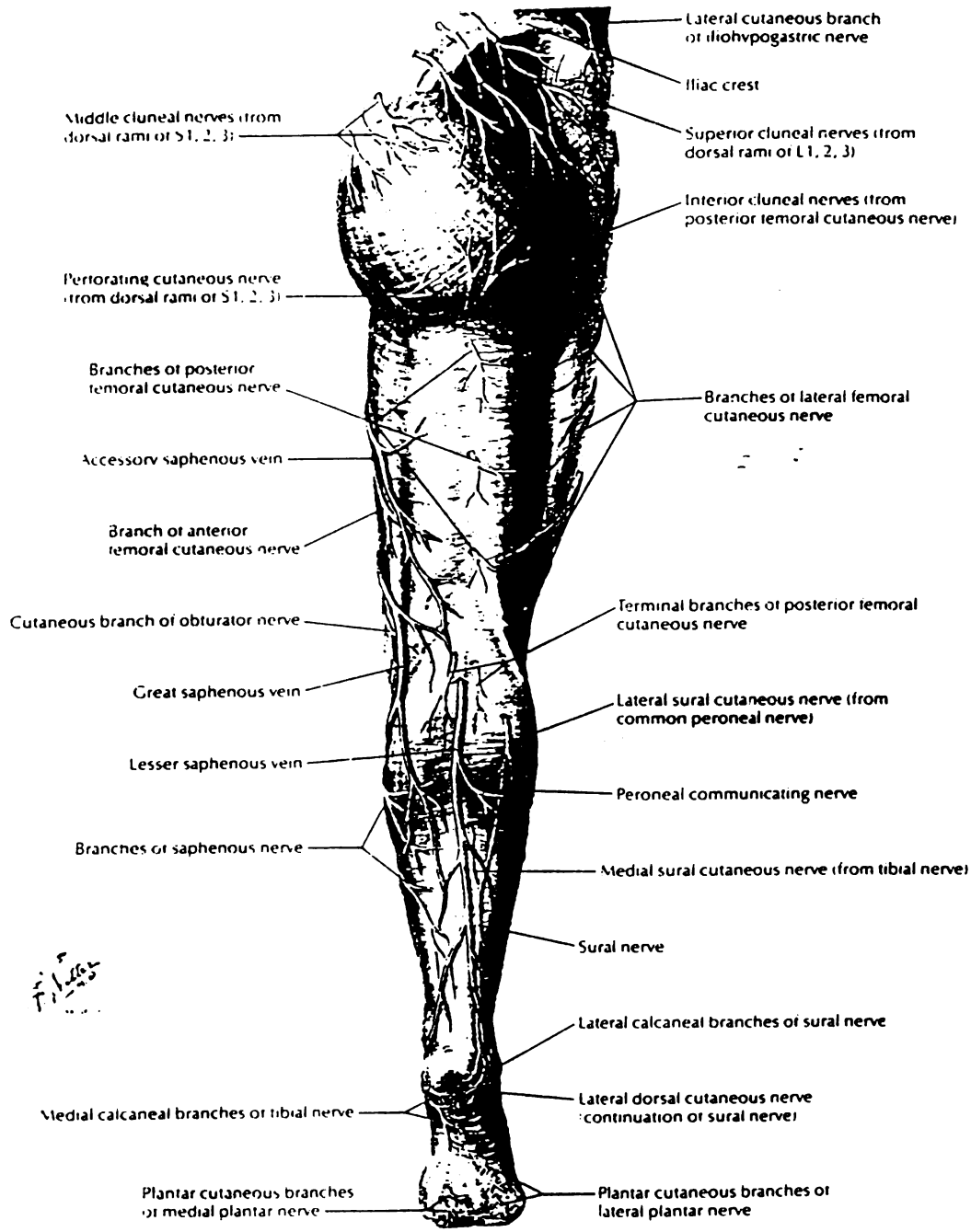


Figure 4. From Atlas of human anatomy by F. H. Netter. Copyright 1989 by Ciba-Geigy Corporation, Pharmaceuticals Division. Reprinted with permission.

valves, while the lesser saphenous has 6-12 ; all open in a cephalad direction (Gourdin & Smith, 1988; Moore, 1998). Normal resting standing venous pressure is 90-100 mmHg at the foot/ankle, reduces to 30 mmHg during calf muscle (pump) activity, and returns to pre-exercise levels within approximately 30 seconds of inactivity (Callum, Eaglstein & Lynch, 1988; Gourdin & Smith, 1988; Moore, 1998; Nicolaides, Hussein, Szendro, Christopoulas, Vasdekis & Clarke, 1993; Phillips & Dover, 1991; Spittell, 1991; Trott, 1992).

Leg veins differ structurally from arteries in other ways besides valve presence. The vein walls are one third to one tenth as thick as arterial walls, possess very little elastic tissue, have a prominent adventitia (outer wall) and no smooth muscle composition (Moore, 1998). These structural differences allow a wide range of flow rates and the ability to collapse when transmural (wall) pressure is low (Moore, 1998).

The calf muscles in the lower extremities act as a pump and produce changes in venous volume, rate, and direction. This muscle pump normally lowers venous pressure in the dependent lower extremity, reduces venous volume in the exercising limb, and increases venous return (Moore, 1998).

Pathophysiology of Venous Ulcer Development

Homans' (1917) Theory of peripheral venous stagnation, secondary tissue anoxia and cell death, leading to VU development in the malleolar area, was accepted for fifty years (Gourdin & Smith, 1993; Pappas, Fallek, Garcia, Araki, Back, Duran & Hobson, 1995). The outdated term "venous stasis ulcer" evolved from this theory. Venous stasis as perceived by Homans, does not exist, therefore the term VU more correctly reflects the underlying pathophysiology (Blalock, 1929; Falanga & Eaglstein, 1986; Gourdin & Smith, 1993).

VUs are currently hypothesized to be caused by a complex series of

mechanical and biochemical events that begin with calf pump failure (CPF) (Trott, 1992). CPF is defined as the inability to decrease the venous pressure in the lower extremity with exercise (Spittell, 1991). CPF produces chronic venous insufficiency (CVI), and is manifested by venous hypertension (VH), decreased venous refill times, increased foot/ankle venous pressure, reflux with calf muscle relaxation, and only occurs during ambulation (Belcaro, Christopoulos & Nicolaides, 1991; Fitzpatrick, 1989; Nelzen et al., 1991). Arterial pressure remains relatively constant in the legs, so VH produces a decreased pressure differential across capillary beds, reducing blood flow. Table 1 contains a glossary of abbreviations commonly used in this paper.

Table 1.

Glossary of Abbreviations

Term	Abbreviation
Ankle Brachial Indexes	ABIs
Advanced Practice Nurse	APN
Calf Pump Failure	CPF
Chronic Venous Insufficiency	CVI
International Association of Enterostomal Therapy	IAET
Methacillin Resistant Staph Aureus	MRSA
Venous Hypertension	VH
Venous Ulcer	VU
Venous Ulcers	VUs
Venous Valve Incompetence	VVI
Zinc Oxide Impregnated Support Dressings	ZOISDS

Venous valve incompetence (VVI), involving the deep or perforating veins, is the most common cause of calf pump failure and secondary VH (Harris et al. , 1996; Spittell, 1991). CPF and VVI are caused by acquired or congenital disorders such as outflow tract regurgitation (e.g. deep vein thrombosis; floppy, incompetent venous valves; post-phlebotic syndrome), outflow tract obstruction (e.g. intraluminal thrombosis; pelvic tumor; pregnancy), perforator vein incompetence, superficial vein regurgitation (varicose vein syndrome) and muscle failure secondary to paralysis and arthritis (Capeheart, 1996; Harris et al., 1996; Spittell, 1991; Trott, 1992; Zink et al., 1992).

The Fibrin Cuff Theory is also currently associated with VU development. VH produces edema secondary to increased capillary permeability (secondary to development of multiple new capillaries and stretching of interendothelial pores), with leakage of fluid containing large protein molecules such as fibrinogen and coagulation factors into the interstitial space (Capeheart, 1996; Gourdin & Smith, 1988; Phillips & Dover, 199l; Seeley, 1992). These protein molecules, in conjunction with tissue factors, lead to the conversion of fibrinogen to fibrin (Moore, 1998). The fibrin accumulates in the tissues, coats the capillaries and creates a fibrin cuff, which interferes with the needed exchange of oxygen, nutrients, and waste removal from the skin and subcutaneous layers in the lower extremity (Phillips & Dover, 1991; Moore, 1998). The end result is tissue anoxia and death of the skin in the affected region producing VUs (Moore, 1998).

Several other theories addressing the assistive role of white blood cells and toxins in VU development emerged when a significantly increased accumulation of white cells was found in the dependent feet of individuals with VH (Thomas, Nash & Dormandy, 1988). One theory suggests that white blood cells (neutrophils) trapped in the fluid of edematous legs activate and release

various substances normally involved in fighting infections, as well as proteolytic enzymes and oxygen metabolites, all which damage and impair capillary function and increase cell membrane permeability (Harris et al., 1996; Papas et al., 1995; Zink et al., 1992). Another speculative hypothesis proposes that trapped leukocytes occlude capillary or post-capillary venular flow, thereby resulting in micro-circulatory and cutaneous ischemia (Coleridge-Smith, Thomas, Scurr & Dormandy, 1988; Pappas et al., 1995). Yet another hypothesis suggests that the breakdown of blood releases toxic by-products which cause tissue breakdown (Black, 1995).

Clinical Signs and Symptoms

Venules and capillaries in the lower extremity elongate and dilate secondary to prolonged, consistent VH, which is observed clinically as "ankle flare" (corona phlebectatica), an area of dilated lesser saphenous vessels usually below the medial malleolus (Trott, 1992). The greater number of capillaries observed in ankle flare, the more extensive the venous disease (Browse, 1988).

Characteristic bilateral or unilateral skin changes precede, accompany, and can predict risk for VU development. Skin changes include brownish to brownish-red hyperpigmentation secondary to hemosiderin and melanin deposits, eczema, lipodermatosclerosis and areas of light avascular skin or atrophie blanche (Callam, Eaglstein & Lynch, 1988; Holloway, 1996; Merli, 1994; Trott, 1992, Zink et al., 1992). Eczema commonly develops in response to xerosis, a chronic dryness secondary to lack of oxygen and nutrients, presenting with erythema, scaling, pruritis and occasional weeping (Zink et al., 1992).

Lipodermatosclerosis, which may be mistaken for cellulitis, precedes and strongly predicts VU development (Phillips & Dover, 1991). It is an induration

and erythematous hyperpigmentation of the leg associated with fibrin deposition (Callum et al., 1988). The combination of lipodermatosclerosis, hemosiderin deposition and epithelial scaling is referred to as venous dermatitis (Phillips & Dover, 1991).

VUs are the most undesirable consequences caused by CPF, CVI and VH (Simon & McCollum, 1996). A combination of persistent or severe edema with capillary obstruction impairs blood flow, causing ischemia and potential for VU formation (Harris et al., 1996). Other factors that encourage the development of VUs are malnutrition, hypoalbuminemia, immobility and trauma (Spittell,1992; Zink et al., 1992). Tissue breakdown (cell death) and necrotic tissue evolve into VUs most often after minor trauma, pressure, but also may occur spontaneously (Black, 1995; Burton, 1993; Holloway, 1996; Merli, 1994; Phillips & Dover, 1991; Seeley, 1992).

An ulcer is the focal loss of epidermis and dermis (Mostow, 1994). A chronic ulcer is one that fails to heal within a reasonable period of time. The reasonable expectation of time for healing is not absolute or clearly defined, and depends on many factors such as wound size, etiology, and general health status of the individual (Mostow, 1994). A VU is a chronic, cutaneous ulceration developing secondary to the pathology of chronic, deep venous insufficiency (Capeheart, 1996; Phillips & Dover, 1991).

VUs most commonly develop proximal to or at the medial malleolus, but can develop anywhere on the lower leg (Black, 1995; Holloway, 1996; Merli, 1994; Trott, 1992). The majority of VUs develop along the long saphenous system (Trott, 1992). VUs are usually pliable, movable with the skin, beefy red in color, irregular in shape with flat borders, and with copious serosanguineous fluid (Holloway, 1996; Merli, 1994; Trott, 1992). Wound depths range from shallow to full-thickness, with possible extension into fascia, muscle and

periosteum (Black, 1995; Sieggreen & Maklebust, 1996).

Pain is usually not present with VUs, but varies in intensity when acknowledged. The severe discomfort described by many with new VUs is often compared to arterial insufficiency pain, and is most likely due to a similar etiology (Holloway, 1996). Other causes of VU pain are close proximity to the saphenous nerve, exposed nerves, engorged veins, inflammation secondary to infection, dessication and/or edema (Harris et al., 1996; Sieggreen & Maklebust, 1996; Zink et al., 1992).

Assessment

The diagnosis of VU is often made exclusively upon clinical presentation with a minimal need for diagnostic tests if the individual at high risk presents with classic VU symptoms (Black, 1995; Falanga & Eaglstein,1988). All possible etiologies and contributing factors must be considered. Many other possible etiologies or contributors to leg ulcer development and/or failure to heal are ischemic arterial disease, mixed arteriovenous disease, external pressure, diabetes, heart failure, malnutrition, hematologic diseases, cutaneous vasculitis, polycythemia vera, insect bites, pyoderma gangrenosum, factitious wounds, malignancies, connective tissue disorders, fungal, bacterial or syphilitic infection, radiation, sickle cell anemia, morbid obesity, medications, injections, neuropathy and trauma (Falanga & Eaglstein, 1988; Harris et al., 1996; Merli et al., 1994). Several studies suggest that malignancy can be a complication of VUs (Yang, Morrison, Vandongen, Singh & Stacey, 1996).

The history obtained by the primary care provider should include risk factor questions about family or personal history of leg ulcers, occupations requiring long hours of standing, history of deep vein thrombosis (DVT), varicose veins, congestive heart failure, angina, pelvic tumor, obesity, pregnancy, intravenous drug use, immobility, paralysis, leg trauma, pain, and

malnutrition (Black, 1996; Capeheart, 1996; Harris, Brown-Etris & Troyer-Caudle, 1996; Pieper, 1996). Questions about social habits, medications and other medical conditions that can cause or retard healing of VUs such as diabetes, autoimmune diseases, malignancy, steroid dependence, arterial insufficiency, DVT, edema, cigarette smoking, alcohol consumption, and previous leg ulcer treatments should also be included (Black, 1995; Capeheart, 1996). Twenty to seventy percent of individuals with a history of DVT are unaware of it, especially if the episode was silent or undiagnosed (Capeheart, 1996).

Comprehensive evaluation and documentation of all VU characteristics should be performed during the initial and subsequent appointments. The VU location, measurement(s) of length, width and depth (in centimeters), periwound skin appearance, wound border, margins and base (whether granulation and/or necrotic tissue is present), exudate (amount and color), odor and general condition of the limb should be thoroughly described. Black (1995) recommends including the wound color in the description.

Wound assessment and documentation should contain quantitative data included whenever possible. Quantitative assessment methods for obtaining data promote increased reliability and validity of wound data, improved communication and continuity of care (Van Rijswijk, 1996).

A tape measure or ruler can be used to measure VUs and obtain quantitative data. This method is easy, inexpensive, fast, has good inter-rater and intra-rater reliability, often overestimates actual wound size, and decreases in reliability with increasing wound size (Van Rijswijk, 1996). Polaroid photos or tracing the VU on transparency film is easy, fast, has excellent inter-rater and intra-rater reliability, decreases in reliability with reducing wound size, and is more costly to perform (Black, 1995; Van Rijswijk, 1996). It is mandatory to

obtain informed consent prior to taking photographs of wounds (Van Rijswijk, 1996) and to label them appropriately.

Assessment for infection must be a part of every wound evaluation. Wound cultures are rarely indicated for VUs (Sieggreen et al., 1996) since these wounds tend to be heavily contaminated with aerobic and anaerobic bacteria (Falanga & Eaglstein, 1988). One study demonstrated that wounds without the classic infection symptoms were just as likely to be infected as those with the classic symptoms, and suggests a return to the routine culturing of wounds (Kravitz, Lyder, Luehrs & Fornek, 1994).

Pathogens in the VU vary, but staph aureus, gram-negative bacilli and others are common (Phillips & Dover, 1991). Methacillin resistant staphylococcus aureus (MRSA) in a wound is significant (Black, 1995). Individual flora in VUs tend to remain constant irrespective of local therapy, and species and concentration of bacteria do not correlate with presence or absence of purulence or the rate of healing (Phillips & Dover, 1991).

Clinical infection develops when the wound, or tissue around the wound, is invaded by a considerable bacterial load, with resulting local cellular injury (Van Rijswijk, 1996). Diagnosis of infection is suggested by one or more common clinical signs: the presence of pus, warmth, pain, odor, erythema, fever, leukocytosis and induration (Harris et al., 1996; Van Rijswijk, 1996). Other findings that may suggest infection are delayed healing, discoloration, friable granulation tissue which bleeds easily, unexpected pain/tenderness, pocketing at wound base, wound breakdown, and epithelial bridging at the wound base (Van Rijswijk, 1996).

Chronic wounds may easily be misdiagnosed as infected secondary to the presence of chronic inflammation, unrelieved pressure or allergic reactions producing classic infection symptoms (Van Rijswijk, 1996). The only sign of

infection in immunocompromised or neuropathic individuals may be a change in the sensation around the wound. Signs of infection may be difficult to interpret in the older population, in part because of their declining immune function (Harris et al., 1996).

A specimen can be obtained for identification of the specific infecting organism(s) in three ways: a swab culture (most common), an aspiration sample, or tissue biopsy (most accurate) (Harris et al., 1996). The specimen must be obtained from within the wound, and not from the wound surface, or results may reflect only surface wound organisms (Burton, 1994, Kerstein, 1996). The culture specimen should be obtained after necrotic tissue is removed, but before antibiotic therapy is instituted (International Association for Enterostomal Therapy [IAET], 1988). A quantitative culture showing 100,000 or more organisms per gram of tissue confirms the diagnosis of infection (Black, 1995). Beta hemolytic streptococci loaded wounds need only 1,000 organisms per gram of tissue to produce infection (Stotts & Wipke-Tevis, 1996). Diabetics tend to have more frequent and severe wound and systemic infections than non-diabetics (Morain & Colen, 1990).

Black (1995) suggests a bone scan with delayed images to rule out osteomyelitis. When results are questionable, the more specific WBC scan can be obtained. Osteomyelitis accompanies many deeper nonhealing leg ulcers, especially in persons with diabetes, and often will not show on plain x-rays in early stages (Black, 1995). An ulcer biopsy may be indicated for non-healing VUs .

Before comprehensive VU treatment can begin, a baseline assessment of pedal pulses, motor and sensory nerve function, palpation of veins, calf tenderness (Homan's sign) and cords is recommended (Black, 1995; Harris et al., 1996). If strong, palpable pedal pulses are present, the arterial

status of the lower extremities is usually adequate and requires no additional diagnostic testing (Harris et al., 1996). The presence of adequate distal pulses, however, does not exclude arterial disease (Falanga & Eaglstein, 1988). Some individuals with uncompromised arterial flow have congenital, nonpalpable pedal pulses, so the absence of palpable pedal pulses does not confirm arterial disease (Harris et al., 1996).

If there is any question about the current presence of a deep vein thrombosis venous disease, a venous Doppler vascular study should be obtained (Black, 1995). Venous Doppler studies may include continuous wave Doppler analysis of venous flow, various forms of plethysmography, venography (ascending or descending or both) and ultrasound imaging combined with Doppler flow studies (duplex scanning) (Black, 1995).

Ankle brachial indexes (ABIs) are indicated when pedal pulses are not palpable, or to screen for the presence of arterial disease when pedal pulses are palpable, especially when a history of hypertension, diabetes, coronary artery disease or stroke is present (Burton, 1995; Merli et al., 1994). ABIs can be obtained at the bedside or in a noninvasive vascular lab. They are obtained by placing a blood pressure cuff over the calf and measuring systolic pressure (with a doppler) in the ankle over the dorsalis pedis and/or posterior tibial arteries, then repeating in the same side arm to obtain a systolic reading over the brachial artery. The ABI readings are calculated by dividing the ankle systolic blood pressure by the brachial (arm) systolic pressure on each side of the body. Right and left sided ABIs are obtained and documented (Burton, 1995).

The normal ABI range is 0.9 to 1.3 (Burton, 1995; Merli et al., 1994). Individuals with a history of intermittent claudication often have an ABI of 0.35-0.9, and those with impending gangrene may have ABIs of less than 0.25

(Merli et al., 1994). A venous ulcer may not heal well if the ABI is under .75 and compression may be contraindicated since additional pressure of 35 mmHg at the ankle (or above) can significantly decrease arterial flow (Harris et al., 1996). If an individual with a VU has an ABI of less than 0.4-0.5 and/or an ankle systolic pressure of less than 60 mm Hg, they should be referred to a vascular surgeon (Harris et al., 1996; Trott, 1992).

A normal resting ABI alone does not guarantee the exclusion of arterial disease, therefore a detailed patient history (as outlined earlier) is required to help identify those at risk. Diabetics may have incompressible, calcified arteries, producing falsely elevated resting ABI readings. Individuals with chronic renal failure and leg edema may also produce unreliable ABI results (Moffatt & O'Hare, 1995).

A Trendelenburg test can be administered to evaluate vein valve competence. This noninvasive test is administered after the patient lies in a supine position, with the affected leg elevated, for approximately five minutes. A tourniquet is placed below the knee (after the veins empty) and the patient is asked to stand. Incompetent perforator valves allow the superficial veins to distend quickly. Incompetent saphenous vein valves allow additional sudden venous distension. (Zink, Rousseau, Halloway, 1992).

Management/Treatment

In this era of cost containment and managed care, primary care providers may find themselves in the position of managing VUs with fewer resources and more demands. Use of multidisciplinary teams, wound care centers or clinics, critical pathways or protocols, state of the art high-tech dressings and equipment are all designed for achieving the optimal goal of rapid wound healing. Specialty services, which can enhance primary care management of wounds, such as physical therapy, nutritional consults, specialist physician, and

mental health are at best being doled out sparingly and some may even be eliminated completely by third party payers (Cahill & Spence, 1995). Regular wound assessment data (showing progression or lack of progression toward desired outcomes) can be a valuable tool when negotiating with managed care organizations for certain referrals and wound care supplies (Richard, 1995).

The history and wound related assessment findings are the foundation for developing specific goals and the plan of care (Van Rijswijk, 1996). VU patients may have a number of concomitant conditions which can impact the healing process, therefore realistic, individualized and clearly defined goals are important (Van Rijswijk, 1996).

Algorithms, guidelines and protocols should not lead one to believe that VUs can be managed with "cookbook medicine" (Sieggreen et al., 1996). Chronic wound care guidelines and protocols can be helpful in a primary care setting, but data providing unambiguous advice on many aspects of care is not currently available.

Healing of VUs is a complex biological process involving a delicate balance and interaction of variables such as sufficient arterial blood flow, venous return, absence of wound infection and debris, nutrition, balanced wound healing factors, and activity level (Cahill & Spence, 1995; Stotts & Wipke-Tevis, 1996). Management of VUs is directed at reduction of venous hypertension and reduction or elimination of pericapillary fibrin (Falanga & Eaglstein, 1986). The four primary treatment goals are: (l) control of underlying medical and nutritional disorders, (2) reduction or elimination of edema, (3) stimulation of fibrinolysis, and (4) maintenance of a moist wound environment (Zink et al., 1992).

Approximately 85% of VUs respond to conservative, non-surgical management (Hurley, 1990). Conservative management generally includes

compression, elevation, and topical wound care (Harris, Brown-Etris & Troyer-Caudle, 1996). A surgical intervention may be indicated when ulcers fail to respond over a period of weeks to months. Some surgical interventions include split- or full-thickness skin grafting, pinch and punch biopsy skin grafting, xenografting, surgical debridement, ligation or removal of thrombosed superficial or perforating veins, valve transfers and transplants and Linten procedures (subfascial perforator ligation) (Black, 1995; Harris et al., 1996; Kerstein, 1996; Zink et al., 1992).

The VUs of compliant patients heal significantly sooner and are are less likely to recur (Erickson et al., 1995). Once a VU heals, preventive, lifelong, compression therapy and good skin care is mandatory (Seeley, 1992). The VU patient must realize that they have a lifelong problem requiring prevention as the best treatment (Harris et al., 1996; Seeley, 1992).

Controlling Underlying Medical and Nutritional Disorders

It is extremely important to recognize and correct or control systemic factors deleterious to proper wound healing (e.g. diabetes, heart and renal failure, anemia). VUs are impacted by the same variables that impact most any other type of wound. Nutritional and immunologic deficiencies, for example, interfere with VU healing. Wound healing depends on the body's ability to create a normal inflammatory response. Various drugs have the potential to impact that response. Systemic corticosteroids (e. g. Prednisone) in doses exceeding 10 milligrams/day are detrimental to the healing of acute and possibly chronic wounds (Oxlund, Fogdestam & Viidik, 1979). Non-steroidal anti-inflammatory drugs (NSAIDS) probably have the same effect (Falanga & Eaglstein, 1988). Topical hydrocortisone preparations, with a potency greater than 1%, have been shown to delay healing as well (Falanga & Eaglstein, 1988).

A low serum albumin and/or transferrin concentration indicate some degree of malnutrition requiring correction with dietary supplementation. In such circumstances, ascorbic acid and zinc deficiencies may be present and require correction. Ascorbic acid is required for the hydroxylation of lysine and proline during collagen synthesis. A deficiency of this vitamin can develop quickly in the elderly and debilitated population. Zinc depletion may result in impaired healing because this element plays an important role in a variety of enzyme activities. Zinc deficient individuals should be treated with one to two grams/day of ascorbic acid and 220 milligrams of zinc sulfate three times daily (Falanga & Eaglstein, 1988). Albumin levels lower than 3.5 gm/dl may increase lower extremity edema by extending the distance between the blood vessels and the dermal surface, and warrant consideration of protein supplementation (Zink et al., 1992).

Cardinal nutrients in the prevention and treatment of VUs are vitamins A, C and zinc (Zink et al., 1992). Treatment with various vitamins and minerals is unlikely to promote healing in those who are not deficient, so indiscriminate use of vitamins and minerals is not recommended (Falanga & Eaglstein, 1988). Zinc can interfere with the immune process unless used judiciously (Falanga & Eaglstein, 1988). The primary care provider must look at many parameters to assess nutritional status, not just serum albumin and ferritin levels (Sieggreen et al., 1996). The obese patient should be instructed and placed on a weight reduction diet, and encouraged to join a weight-loss support group, available through various senior centers, churches or community centers (Capeheart, 1991).

Contact dermatitis causes pruritis, erythema, possibly delays or disrupts VU healing, and may initiate the development of more VUs (Falanga & Eaglstein, 1988). The primary care provider must be careful to detect and

avoid contact dermatitis when treating patients with VUs. Most patients with VUs have multiple allergies to various topical agents such as lanolin, antibiotics, various other components of topical preparations, corticosteroids prescribed for pruritis, and cetearyl alcohol, a common ingredient in creams and paste bandages (Black, 1995; Falanga & Eaglstein, 1988).

All topical medications can potentially cause contact dermatitis, therefore, elicit patient use of topical home remedies. Patch testing may identify specific allergens and preparations that must be avoided (Falanga & Eaglstein, 1988).

Emollients containing urea (10-20%) and lactic acid (5%-12%) assist in removing retained layers of stratum corneum and subsequently reduce the scaliness associated with xerosis (Capeheart, 1996). Prevention of xerosis includes less frequent bathing, addition of oil to bath water, use of mild soaps or soapless cleansers, and application of petroleum based creams after bathing to retard water loss (Holloway, Ooi & Weingarten, 1995; Zink et al., 1991).

The presence of bacterial contamination seems to be of little relevance to VU healing, but infection of these wounds can significantly delay healing (Chung, Cackovic, & Kerstein, 1996; Skene, Smith, Dore, Charlett & Lewis, 1992). Skene and colleagues (1992) found a change in VU bacteria presence from month to month in their study. Vigorous use of antibiotics only changes the wound flora in uninfected wounds (Phillips & Dover, 1991).

The source of infection is usually staphylococcus aureus, pseudomonas, group A streptococcus, or enterobacteria (Burton, 1994; Kerstein, 1996). Unless cellulitis or systemic infection is present, antibiotics should be avoided (Phillips & Dover, 1991; The Alexander House Group, 1992). Depending on the patients clinical situation, they may be treated with topical and/or systemic antibiotics (Harris et al., 1996). The systemic antimicrobial agents of choice are usually the cephalosporins and quinolones (Parish & Witkowski, 1994).

Hospitalization and use of intravenous antibiotics usually isn't necessary, but may be required for more severe cases of cellulitis (Sieggreen & Maklebust, 1996).

Some providers advocate and use trials of topical and systemic antibiotics when heavy bacterial contamination persists despite frequent debridement, or if frank bacterial contamination remains a problem (Falanga & Eaglstein, 1988). Topical antimicrobial agents often provide inadequate penetration of significantly infected tissues (Parish & Witkowski, 1994).

Reduction or Elimination of Edema

Edema control is essential for VU healing since healing cannot take place with continued elevated hydrostatic pressure (Black, 1995). According to Black (1995), edema control not only heals the ulcer, but it saves the patient's life, and therefore, cannot be overemphasized. Trott (1992), however, suggests that edema alone does not impede VU healing.

The cornerstone of effective, conservative treatment of VU is compression and elevation (Gourdin & Smith, 1988; Trott, 1992). Compression improves calf muscle pump function, controls associated edema, may increase transcutaneous oxygenation and tissue perfusion (Black, 1995; Cherry, 1991; Phillips & Dover, 1991). Bourne (1991) recommends application of compression bandages after leg elevation for at least three hours (if possible). Compression is accomplished by using gradient elastic compression stockings or elastic bandages, zinc oxide impregnated support dressings (ZOISDs), or sequential compression devices and alone will heal approximately 86% of VUs (Cherry, 1991; Cordts, Hanrahan, Rodriguez, Woodson, LaMorte & Menzoian,1992; Phillips & Dover 1991). Compression should be graduated with maximum pressure at the ankle and reducing linearly up the lower extremity (Colgan & Moore, 1991).

The exact amount of external compression pressure necessary to heal a VU is unknown. Stemmer (1969) recommends 35-40 mmHg at the ankle to prevent capillary transudation in legs with venous disease. Margolia & Cohen (1994) suggest 20-40 mmHg. McDonagh's research shows that 40-55 mmHg applied externally at the ankle is needed to compensate for internal venous pressures (Cahill & Spence, 1995). Finally, Sieggreen & Maklebust (1996) indicate that an effective compression bandage generates 40-70 mmHg of pressure. A safe and effective level of compression is 30-40 mmHg for VU treatment and 20-30 mmHg for prevention of VUs in clients with mild CVI.

It is generally agreed that high compression bandaging can be used on patients with a resting ABI of greater than 0.8 (Moffatt & O'Hare, 1995; Thompson, 1994). Sieggreen & Maklebust (1996) contraindicate compression therapy when ABIs are less than 0.6, since even slight pressure against the skin can create another ulcer with this much arterial impairment. A VU may not heal well if the ABI is under 0.75 with compression possibly contraindicated since additional pressure of 35 mm Hg or more at the ankle can significantly decrease arterial flow (Harris et al. , 1996). Capeheart (1996) contraindicates use of compression when co-existing arterial insufficiency is present, but does not indicate an ABI parameter.

Compression stockings impact the deep venous system by increasing velocity of flow in the superficial and communicating veins, thus effectively improving microcirculation while reducing VH with its deleterious effects on the skin (Cherry, 1991; Kerstein, 1996). Compression stockings probably reduce edema by emptying periulcer venous channels, often with pressures as low as 18 mm Hg (Zink et al., 1992). Most VU patients can be effectively treated (initially and over the long-term) with elastic compression stockings (Mayberry, 1991; Zink et al., 1992). They should not be measured and fitted (ideally) until

edema has resolved (Cahill & Spence, 1995; Zink et al., 1992). Ankle, calf, and leg length (e. g. from ankle to just below the popliteal fossa) measurements are needed before prescription compression stockings are ordered . Compression hose range in length from knee-high, mid-thigh, thigh-high to panty hose.

Compression hose should be applied before the wearer gets out of bed in the morning, worn all day, and removed at bedtime (Zink et al., 1992). If an individual is unable to tolerate high pressure stockings, consideration should be given to low-pressure stockings for daily, long-term wear (Harris et al., 1996; Zink et al., 1992). These hose may need to be replaced after 4 months rather than the usual six to nine months (if worn daily) to assure that adequate compression is maintained (Sieggreen, Cohen, Kloth, Harding & Stotts, 1996). These hose are not covered by Medicare and many commercial insurances.

Black (1995) recommends elastic leg wraps over compression stockings since they conform to the abnormally shaped legs of those with advanced venous disease, unlike compression stockings. Other benefits of elastic leg wraps are the ability to hold gauze (or other) dressings in place, elimination of potential tape damage on compromised adjacent skin, and the ability to "give" if the leg develops unexpected edema, such as with cellulitis (Black, 1995). Elastic wraps must be wrapped snugly (covering all skin) around the lower extremity, using a figure eight wrapping technique, extending from the toes to the knee. Two fingers should fit snugly beneath the elastic wrap at the base of the toes and then pressure should gradually lessen, in gradient fashion as the wrap progresses toward the knee (Black, 1995).

Elastic wraps should be rewrapped twice daily, and worn 24 hours a day until edema is controlled, and thereafter whenever the leg is dependent (Black, 1995). They should be at least three inches wide and never applied over dermatitic skin (Parish & Witkowski, 1994). Later, compression stockings can

be substituted long-term to prevent recurrence of VUs. Sequential compression pumps can be successfully employed with severe edema, but currently are approved for reimbursement by Medicare only for true lymphedema (Black, 1995).

ZOISDs are commonly used to treat VUs. They consist of a gauze/cloth bandage roll that is saturated with zinc oxide, and possibly calamine, glycerin and gelatin that is wrapped around the affected leg and reinforced with an elastic support bandage. Its healing capability results from a combination of external compression, environmental protection, and skin/wound contact with the moist bandage paste, and not the chemical components of the dressing itself (Barr, 1996). It can remain in place for 3-14 days, with an average wear time of one week (Barr, 1996). It has been found effective for treatment of leg ulcer-associated inflammatory skin conditions such as eczema (Barr, 1996).

The frequency of ZOISDs changes usually depends on the amount of wound drainage present. Most wearers find it messy, smelly, unsightly and painful as it dries and rubs against the ulcer which predisposes to noncompliance (Sieggreen & Maklebust, 1996). ZOISDs that are not changed often enough, can produce maceration of adjacent tissue encouraging fungal overgrowth and wound enlargement. Excessively tight ZOISDs applications have resulted in localized purpura, cyanosis and necrosis (Barr, 1996).

ZOISDs can be modified by adding a hydrocolloid, film, foam, alginate or non-adherent gauze dressing over the ulcer prior to its application . Wound drainage increases (perhaps dramatically) for approximately two weeks after an occlusive dressing is applied, mandating more frequent dressing changes during that time (Barr, 1992; Falanga & Eaglstein, 1988; Sieggreen & Maklebust, 1996).

ZOISDs are contraindicated with significant arterial disease, weeping, friable skin surrounding the ulcer, infection or known sensitivity to any of the components of the boot (Cahill & Spence, 1995; Sieggreen & Maklebust, 1996). ZOISDs are beneficial for individuals who can not or do not want to change compression bandages daily or wear compression stockings, noncompliant patients, or for those who pick at and traumatize their ulcers (Sieggreen & Maklebust, 1996).

Leg elevation is effective generally, since VH is only present while the patient is standing (Fitzpatrick, 1989). Leg elevation above heart level, while the individual is lying down, is desirable since venous blood flows downhill aided by gravity (Cahill & Spence, 1995; IAET, 1988). Continuous leg elevation is not convenient for ambulatory humans, therefore compliance is extremely poor (Black, 1995). Leg elevation usually can be trialed alone (without compression therapy) for nonambulatory patients (Sieggreen & Maklebust, 1996). Leg elevation is of paramount importance for edema relief; but when edema is severe, judicious and short-term use of diuretics is indicated (Falanga & Eaglstein,1988; Myers, Rightor & Cherry, 1972).

Mild and intermittent ambulation is recommended (unless contraindicated) because it promotes beneficial effects of the skeletal muscle calf pump producing variable reductions in VH (Capeheart, 1996; Sieggreen et al., 1996). Walking, toe ups and point flex exercises, marching in place while wearing stockings or compression wraps should be encouraged to increase venous return and collateral circulation, and strengthening of the calf muscle pump (Cahill & Spence, 1995; IAET, 1988). Activity should be alternated with leg elevation higher than the heart when lying down (IAET, 1988). Standing still and sitting with legs below the level of the heart should be discouraged (Cahill & Spence, 1995; IAET, 1988).

Stimulation of Fibrinolysis

Present and future therapy should be directed at eliminating the pericapillary fibrin layer (fibrin cuff) since VUs result from a block in the proper physiologic exchanges between blood and tissue (Falanaga & Eaglstein, 1988). Fibrinolytic mediators are tissue plasminogen activator (the major endogenous enzyme responsible for the cleavage of plasminogen to plasmin), plasminogen activator inhibitor-1 (the major circulating inhibitor of tissue plasminogen activator), and alpha 2-antiplasmin (the major inhibitor of plasmin) (Illig, Green, Ouriel, Riggs, Bartos, Whorf, DeWeese, Chibber, Marder & Francis, 1997). Plasminogen activator, the fibrinolytic activator responsible for the cleavage of plasminogen to plasmin, is rapidly released by endothelial cells in response to venous occlusion, exercise, endotoxin, and arterial ischemia (Illig et al., 1997).

Research based literature supporting specific primary care interventions which promote the elimination of the pericapillary fibrin lay (fibrin cuff) is limited. Compression therapy probably encourages fibrin degradation, as occlusive dressings do (Zink et al., 1992). Occlusive dressings have demonstrated the ability to stimulate urokinase production by fibroblasts in vitro and proteinases from wound fluid which promotes lysis of of fibrin clot and eschar (Chung et al., 1996). Hydrocolloid dressings have been shown to lyse pericapillary fibrin cuffs which may inhibit tissue repair by preventing growth factors, oxygen, and nutrients from reaching the wound, although other theories dispute this (Chung et al., 1996; Mulder et al., 1993). Helfman, Ovington & Falanga (1994) suggest that keeping the wound moist reduces the fibrotic zone.

Maintenance of a Moist Environment, Cleansing, Debridement and Topical Agents

The goal of VU topical therapy is to keep the wound surface clean, moist and free from secondary infection (IAET, 1988). Moisture retentive/occlusive dressings frequently used with healthy, granulating VUs are alginates, hydrocolloids, hydrogels (both solid and viscous), foams or sometimes transparent films, depending on the amount of exudate (IAET, 1988).

The advantages of moist wound healing have been known for some time but the extensive appreciation and use of occlusive dressings for chronic wounds is a fairly recent development. An occlusive dressing is a moisture retentive dressing, that maintains a moist wound surface when in place (Helfman et al., 1994). All occlusive dressings are made from different materials, with varying properties such as adhesiveness and oxygen permeability.

Occlusive dressings reduce pain and discomfort, allow more rapid healing, reduce frequency of dressing changes, exclude bacteria from the ulcer, promote autolytic debridement and facilitate ambulatory treatment of large and numerous ulcers (Alvarez et al., 1989; Falanga & Eaglstein, 1988). Occlusion leads to a 40 percent increase in the reepithelization of partial-thickness wounds secondary to epidermal cell movement, release of growth factors, acceleration of the inflammatory phase of wound repair and new vascularization, which promotes a significantly faster rate of healing in full-thickness wounds as well (Chang et al., 1996; Helfman et al., 1994). Autolytic debridement beneath occlusion results from enhanced leukocyte migration with resultant autolysis of necrotic tissue (IAET, 1988).

Occlusive dressings enhance the growth of resident and pathogenic bacteria in the wound with a shift toward gram-negative bacteria (Mertz,

Marshall & Eaglstein, 1984). This bacterial growth enhancement does not correlate with an increased infection rate (Helfman et al., 1994). Fifty different controlled trials of occlusive dressings demonstrated a statistically significant decrease in infection rates compared with wounds dressed with conventional gauze, xeroform or nonadherent layer treatments (Hutchinson, 1989), most likely due to an enhancement of the host's defense mechanisms and possible protection against entry of exogenous bacteria (Helfman et al., 1994).

Transparent film (polymer film) dressings are nonabsorptive, thin, highly elastomeric, impermeable to bacteria, liquid, and environmental contaminants, and permeable (varying) to atmospheric gases and moisture vapor (Helfman, Ovington & Falanga, 1994). These dressings adhere to dry, clean skin via an adhesive coat on one side, therefore, no secondary dressing is necessary.

Hydrocolloid dressings are compound formulations of hydroactive and absorptive particles, combined with elastomeric agents, which interact with wound fluid and melt into a moist, viscous, colloidal gel (Helfman et al., 1994; Walker, 1996). These dressings may be impermeable to moisture vapor and gases (Helfman et al., 1994). They also adhere to dry, clean skin via an adhesive coat and require no secondary dressing.

Hydrocolloid and film dressings should not be changed more than three times a week, but may be left in place for up to seven days (Walker, 1996). They are contraindicated for highly exudative, malodorous or infected VUs (until the infecting organism if identified and treated), and are more cost effective and convenient than traditional gauze dressings which may require changing up to four times daily (Black, 1995). There are many different versions/trade names of each dressing category available for purchase.

Hydrogel dressings are sheets of crosslinked hydrophilic polymers which can contain up to 96 percent water (Helfman et al., 1994). They are highly

absorbent, dessicate rapidly, and (most) are non-adherent, thus mandating a secondary dressing to prevent dehydration and secure placement.

Calcium alginate dressings are polysaccharide dressings derived from kelp (seaweed) and are available in sheets or twisted staple fiber ropes (Helfman et al., 1994). A gel is formed as the fibers absorb moderate amounts of exudate. These dressings are non-adherent, thus mandating a secondary dressing to prevent dehydration, absorb extra exudate and secure placement.

Polymer foam dressings were developed to absorb larger amounts of exudate than other types of occlusive dressings. They are usually non-adherent and require a secondary dressing to absorb extra exudate and secure placement. Many foam dressings have an outer transparent film overlap to allow for adherence and prevent leakage.

Generally, the use of topical dressings such as a hydrocolloid or transparent film with compression stockings or wraps is accepted (IAET, 1988). Combined therapy of semipermeable film and compression (Unna's boot version of ZOISD) has produced accelerated wound healing versus the Unna boot alone (Davis et al., 1992). Cordts and colleagues (1992) found that VUs healed more rapidly with Duoderm hydrocolloid dressing plus an elastic wrap versus wounds treated with Unna's boot version of ZOISD alone, at least during the initial phase of treatment. Backhouse (1987) found no additional benefit from applying an occlusive dressing with a graduated compression bandage versus coarse mesh gauze and a graduated compression bandage. Some form of compressive wrap must be used over the initial dressing, regardless of the type (Harris et al., 1996).

Critical early steps to promote VU healing are cleansing and removal of any necrotic tissue utilizing debridement measures. Non-viable tissue left in the

wound can serve as a source of infection, prolong the inflammatory response, impede formation of granulation tissue, prevent migration of epithelial cells across the wound and ultimately delay or prevent healing (Walker, 1996). Primary debridement methods are sharp (at the bedside or in the operating room), enzymatic (e.g. topical enzymes), mechanical (e.g. wet to dry dressings, whirlpool, scrubbing), and autolytic (via moisture retentive/occlusive dressings).

A wound dressing must provide other benefits besides just keeping the wound moist to facilitate healing. These benefits are obliteration of open spaces (including tracts and undermined areas), absorption of excess exudate, protection from trauma, provision of thermal insulation, and protection from external contaminants (Alvarez et al., 1989).

Table 2 is a summary of the common types of dressings available for treatment of venous ulcers. The table is not all inclusive.

Wounds should be cleansed with noncytotoxic solutions at a pressure between 4-15 pounds per square inch (Alvarez et al., 1989; Skene et al., 1992; Walker, 1996). Placing harsh solutions into an open wound is generally discouraged, since such solutions probably are toxic to the healing cells and tissue (Burton, 1995). Commonly used topical antiseptics (hydrogen peroxide, Dakin's solution, acetic acid, povidone-iodine) are toxic to the wound, while normal saline is non-toxic and appropriate for all wounds (IAET, 1988; Walker, 1996).

There are topical antimicrobial or antibiotic agents available with broad-spectrum bactericidal or bacteriostatic properties that do not measurably harm the healing wound. These include silver sulfadiazine 1% cream, combination antibiotic ointments such as polymyxin B sulfate, zinc bacitracin, and neomycin ointment or spray, and propylene glycol (Bolton & Fattu, 1994). Silver sulfadiazine (Silvadine) dressings can be used for venous ulcers that appear

Table 2

Commonly Used Venous Ulcer Dressings, Wraps and Compression Hose

Category	Product	Manufacturer	Sizes
Alginates	Sorbsan	Dow Hickham	multiple
	Kaltostat	Calgon Vestal	multiple
	Algosteril	Johnson & Johnson	multiple
Foams	Allevyn	Smith & Nephew	multiple
	Lyofoam	Acme United	multiple
	Hydrasorb	Calgon Vestal	4"x4", 4"x8"
Hydrocolloids	Comfeel	Coloplast	multiple
	Duoderm	Convatec	multiple
	Cutanova Hydro	Beiersdorf	multiple
	Restore	Hollister	multiple
Sheet Hydrogels	Clearsite	NDM	multiple
	Vigilon	C. R. Bard	multiple
Transparent Films	Opsite	Smith & Nephew	4"x4", 4"x8"
	Tegaderm	3M Healthcare	multiple
Compression Hose	Jobst	Biersdorf-Jobst	multiple
	Juzo	Julius Zorn	multiple
	Medi	Medi-USA	multiple
	Sigvaris	Sigvaris	multiple
Leg Ulcer Wraps	Dome-Paste	Bayer	3-4" x 10 yd.
	Gelocast	Beiersdorf	3-4" x 10 yd.
	Setopress	Acme United	3-4" x 120"
	Unna-Flex	Convatec	3-4" x 10 yd.

infected. One study examined silver sulfadiazine dressings and found them to be effective in promoting epithelization of VUs, perhaps due to promotion of a moist environment (Bishop, et al., 1992).

Topical application of growth factors is being studied and utilized with promising results for some patients. Growth factor combinations may be more effective than application of single growth factors (Bolton & Fattu, 1994). Wound repair is thought to be regulated by locally acting growth factors (platelet derived,epidermal growth, transforming growth, and basic fibroblast) which can signal proliferation, increase collagen deposition, stimulate neutrophil and fibroblast migration, angiogenesis or alter the phenotype state of the cell (Black, 1995; Bolton & Fattu, 1994). These growth factors proliferate in a clean, moist environment and normally develop in any open wound (Black, 1995). Growth factors, along with topical or systemic vitamin A, may also remedy the effects of systemic steroids (Bolton & Fattu, 1994).

Outcomes

Skene, Smith, Dore, Charlett & Lewis (1992) developed a prognostic index to predict healing time in VUs. They found that younger persons with smaller ulcers, or ulcers of shorter duration and no deep vein disease, were most likely to heal. Bacterial contamination was not found to delay healing. Four indicators were scored to yield the index. The higher the score the more time required for healing. Individuals at the low end of the index healed in a median time of 40 days, while those at the high end of the index required 120 days (Skene et al., 1992).

A clean VU should develop granulation tissue and the wound should begin to close within two weeks (Black, 1995). The percent of reduction in wound area after two to four weeks of treatment, particularly in ulcers that showed a 20-40% reduction from baseline, is predictive of both treatment

outcome and time required for healing (Arnold, Stanley & Fellow, 1994; Van Rijswijk, 1993). Although researchers are starting to uncover timing sequences for chronic wounds, significant unexplained variability remains (Van Rijswijk, 1996).

In Van Rijswijk's (1993) study, women were more likely to heal than men, diabetics were less likely to heal than non-diabetics, odor was more predictive of treatment outcome than wound size at baseline, and that larger wounds produced more intense odor generally. Van Rijswijk (1993) recommends more effort in diagnosing and treating yet unidentified causes of tissue breakdown if the leg ulcer does not measurably reduce in size during the first two to four weeks of treatment.

Deep full-thickness wounds obviously take longer to heal than do partial-thickness wounds (Polansky & Van Rijswijk, 1994). VU healing rates are dependent on the initial ulcer perimeter (Cordts et al., 1992). Lengthened healing times are associated with aging, liposclerosis, physical immobility, pain and social isolation (Johnson, 1995; Jones & Millman, 1990; Lindholm, 1995; Nelzen, Bergqvist, Lindhagen & Hallbook, 1991).

Healing and recurrence rates are impacted by the degree of venous insufficiency, the presence or absence of a cause (such as a prior deep vein thrombosis), overall patient compliance with the prescribed treatment protocol (including lifestyle changes), poor tissue quality at the ulcer site (Black, 1995) and suboptimal treatment (Erickson et al., 1995; Van Rijswijk, 1993). If VUs fail to heal, the diagnosis should be reviewed, with reevaluation of the total clinical picture and treatment plan, a biopsy specimen obtained, and possibly a referral to a vascular or plastic surgeon made (IAET, 1988; Phillips & Dover, 1991).

VUs are chronic wounds. Chronic wounds are defined as "those that have failed to proceed through an orderly and timely process to produce

anatomic and functional integrity, or proceed through the repair process without establishing a sustained anatomic and functional result" (Lazarus, Cooper & Knighton, 1994, p. 489). Complete healing, or restoring the wound back to sustained functional and anatomic continuity, would seem the optimal goal of wound care. A chronic wound could be considered healed when it is resurfaced, or has function restored, and/or does not recur (for weeks or months). Van Rijswisk (1996) suggests defining chronic wound outcomes individually and mutually, based on patient preference.

Expanding the commonly used goal of chronic wound care (healing) to include quality of life issues and prevention (infection, deterioration, recurrence) is desirable (Van Rijswijk, 1996). VU treatment outcome(s) should be determined by the self-care agency in accordance with their decisions as to which risks they choose to contend with or avoid (Chang, 1980). Decision-making responsibility shifts from the provider to the self-care agency with the self-care approach (Chang, 1980).

Chang (1980) recommended three components for patient outcome evaluation based on self-care: patient compliance, patient satisfaction, and adherence to the care plan. None address the actual healing of the VU. The evaluation focuses on patient's actions in terms of the goal (Chang, 1980).

The overall goal in working with the elderly is to promote quality of life, help to develop and/or maintain independent functioning, to empower and prevent iatrogenic dependency (Harper, 1991). Research indicates that self-care agency characteristics are associated with both client/patient satisfaction and compliance, therefore, outcome evaluation must always consider client/patient characteristics (Chang, 1980).

PATIENT EDUCATION AND SELF-CARE

The role of the primary care provider is to set VU treatment into motion, but eventual healing and prevention of recurrence depend on self-care behaviors. Self-care is learned behavior and usually leads to increased compliance (Zinn, 1986). Once the provider and self-care agency agree on one plan of action, the self-care agency is provided with the education tools for its implementation before assuming primary responsibility for implementation of the plan.

Lifelong lifestyle changes are nearly always needed for the VU patient. Teaching is the means to achieve patient self care (Cahill & Spence, 1995). Mandatory patient education includes pathophysiology of venous disease (a permanent condition), wound and skin care, correct use of compression, appropriate leg elevation, protection from trauma and lifelong prevention of recurrences (IAET, 1988).

Individuals learn in their own, unique ways. A variety of teaching methods should be used to accommodate auditory, visual and multisensory learners. Active participation from the patient in the form of repeating back instructions (saying) and performing the actions being taught (doing) is critical. Research has shown that return demonstrations of newly learned behaviors are the best way to ensure understanding of instructions (Cahill & Spence, 1995).

Several teaching sessions are usually required, combined with follow-up by phone or appointments to assist the self-care agency to establish achievable wound related goals, promote adequate self-care, reinforce instructions, identify and monitor progress, provide feedback and provide additional emotional

support (Black, 1995; Cahill & Spence, 1995; IAET, 1988; Sieggreen & Maklebust, 1996). The self-care agency needs support, encouragement and accountability while initiating and integrating VU care and discipline into their lives.

Measures to monitor and increase compliance will hasten healing of VUs and prevent or delay recurrences (Erickson et al., 1995). Other strategies to motivate the self-care agency and improve compliance have been suggested in the literature. The self-care agency may be motivated to participate in leg elevation, by being asked to log their leg elevation time (above heart level) on a chart (Cameron, 1994). Regular wound assessments and VU photographs may also facilitate motivation and teaching (Harris et al., 1996; Van Rijswijk, 1996). Compressive therapy must continue after the ulcer is healed, probably for the rest of the patients life (Seeley, 1992).

Managed care will promote active participation and partial accountability of the self-care agency for outcomes. For example, via premium rebates for those who wear compression stockings, control weight and glucose, or quit smoking (Ennis & Meneses, 1996); all which have a potential impact on VU healing and prevention.

PROTOCOL DEVELOPMENT

Development of a protocol for the treatment of VUs in the primary care setting promoting self-care behaviors seems most feasible in the current environment of cost-containment, diminishing resources, and consumer demand. Practice protocols are a mechanism for assuring excellence in the primary care clinical setting, and their use helps base clinical practice on current research showing the greatest validity and reliability versus just tradition (Gawlinski, 1995). Primary care providers can and should effectively manage most VU patients with a research-based protocol.

VUs are common, recurrent, expensive, and debilitating problems that demand maximum participation by the self-care agency as well as the primary care provider, and a true, equal partnership. A protocol for VU treatment, utilizing self-care concepts, will provide a comprehensive, outcomes-focused, self-care agency driven guide for clinical decision making that includes minimum requirements for VU treatment.

There is ample literature support regarding venous disease pathophysiology, evaluation, diagnostics, conservative and more aggressive treatment modalities, and prevention measures. The four primary goals of treatment are: (l) control of underlying medical and nutritional disorders, (2) reduction or elimination of edema, (3) stimulation of fibrinolysis and (4) maintenance of a moist wound environment. Other variables to consider are treatment of infection, debridement, skin care, and lifelong prevention.

Clinical outcomes need to be expanded to include quality of life issues, patient preferences, prevention, self-care behaviors and wound healing. The

literature is not specific regarding healing rates, therefore predicted times for VU healing are difficult to project.

The VU patient can maintain (or regain) control and independence by providing as much self-care as possible. Optimal self-care usually leads to increased compliance, convenience and satisfaction, faster healing rates, fewer recurrences, less expense and debilitation. The APN can assist optimal self-care agency participation for increased health care demands via support and education, the level of which is negotiated between the self-care agency and nursing in a true, equal partnership.

A protocol for the treatment of venous ulcers in the primary care setting evolves from current literature with Orem's Self-Care Theory as a base. A step by step approach including target population, purpose, clinical processes, administrative, technical and evaluative aspects of VU treatment is defined. The eight primary goals of treatment are: (1) control of underlying medical and nutritional disorders, (2) treatment of infection if present, (3) debridement of necrotic tissue if present, (4) reduction or elimination of VH and edema, (5) stimulation of fibrinolysis, (6) maintenance of a moist wound environment, (7) skin care, and (8) lifelong prevention/maintenance. A checklist format facilitates full utilization of the protocol. A patient education sheet is provided and explained to each patient/care giver(s) during the first visit for VU treatment to promote self-care.

The protocol for VU treatment, located in Appendices A-G, includes an introductory sheet for the professional staff, algorithm, checklists, patient education sheet and evaluation tools.

Approach, Procedures and Education in Protocol Use

The protocol will be introduced to all professional (e. g. physician; APN; PA; RN) staff in a primary care setting with instruction for its use provided via

brief in-service presentations. In-servicing will include an introduction of the potential benefits of protocol use, patient eligibility criteria, protocol content and use, location of protocol, and checklist completion. A copy of the literature review and reference list will also be available for staff use. The staff will be instructed to measure each VU, consult and utilize the protocol during every patient visit. They will also be instructed to document in the patient record each time the protocol is used and exactly where they are at in the protocol.

Evaluation of Protocol Effectiveness

The first measure of effectiveness of the protocol is determination of the extent to which it is used. In an effort to determine how many of the protocols were used in the primary care setting, a number may be assigned to each protocol included in a set so it will be easy to determine how many, if any, were removed for use by the professional staff. After a period of time (e.g. one to four months), it can be determined how many protocols are absent from the original set. A sign-out sheet and flags for each chart will be provided with the set of protocols to make patient identification and chart auditing possible. The flag will remind the staff to use the protocol contained in the chart each visit.

Chart audits may then be completed with criteria such as average healing time of ulcers, length of time between the first visit for VU treatment and healing, recurrence, staff time involved in use of protocol, and staff and client satisfaction with the protocol. Brief provider and patient/caregiver interviews during or after the completion of an evaluation tool may be used to evaluate ease of use of the protocol. A Likert scale will be used for some client/caregiver responses on evaluation forms. Data collected from chart audits, interviews, and evaluation tools will determine the protocol's overall effectiveness.

Implications for Advanced Nursing Practice, Education, Research and Primary Care Practice

The VU treatment protocol suggests many implications for the APN in the primary care setting. Assessment of all individuals, especially the elderly who present for physical examinations or problems with the lower extremities, should include a focused history for VU risk factors and meticulous inspection and palpation of the lower extremities. The assessment phase is an excellent time to provide patient education regarding normal anatomy of the venous system with deviation from normal for patients with CVI.

Once a patient is identified to be at high risk for VU development or actually presents with one, mutuality in goal setting and establishment of an individualized plan of care is of paramount importance. This true partnership between the APN and patient/caregiver(s) facilitates adherence to the prevention and treatment plan and promotes satisfaction. The APN individualizes comprehensive patient/caregiver(s) education to promote understanding and compliance.

Health care professionals in primary care settings need to pay more attention to VU risk factors and identify patients at high risk for the development of VUs. Those patients deemed to be high risk need to have a prevention plan implemented as soon as possible. An educational tool should be provided to each of these patients, making them more responsible for preventive self-care behaviors and self-assessment. The mass media can be utilized to educate the public regarding risk factors, self-assessment and preventive behaviors. These interventions may begin only after the patient presents with a new or recurring VU.

APNs can be instrumental in initiating patient and professional staff education in primary care settings regarding prevention and treatment of VUs. Nursing and medical colleges and physician assistant programs need to look at

the curriculum which may need more content regarding wound prevention, assessment, and treatment. This subject is often addressed only in a one or two hour lecture, with the rest, if any, of the student's wound education obtained from clinical experience. APNs can also contribute articles to professional journals which can heighten awareness of prevention in patients at high risk for VUs.

The APN integrates the research component into protocol development and can bridge the gap between research and practice (ANA, 1981; Gawlinski, 1995). There is a paucity of research regarding the effectiveness of mutually determined versus provider determined VU objectives and treatment plans, as well as studies which determine the cost and benefit of various types of VU prevention interventions. Clinical trials of this protocol need to be carried out in various primary care settings to facilitate this research. This protocol will be disseminated to APNs working in primary care settings via mail, professional conferences and journals. This protocol can also be converted into clinical pathways for use in primary care, acute care, and managed care systems. The extended care facility population, which has a substantial number of VU patients, should be considered for use of this protocol.

The potential value of a protocol of this kind for prevention and treatment of VUs cannot be overlooked. It is imperative that a protocol like this one be followed considering the staggering annual cost of treating leg ulcers in this country. APNs can have a great impact on this segment of the population by identifying patients at risk, promoting self-care behaviors and mutuality, providing prevention and treatment education to patients/caregivers, and encouraging other providers to do the same.

APPENDIX A

INSTRUCTIONS FOR USE OF VENOUS ULCER TREATMENT PROTOCOL

This new protocol was developed to promote maximum client participation in venous ulcer treatment which can improve compliance, patient satisfaction and optimal outcome achievement and prevention of recurrences. Please consult and utilize this protocol for all patients you are seeing who present with a venous ulcer. Begin utilizing this protocol during the first visit and continue using it during the entire time of treatment. Complete checklists.

Please document in the patient record when you are utilizing this protocol and where you are at in the protocol so charts may be audited to evaluate the protocol's effectiveness.

INCLUSION CRITERIA:

1) Diagnosis of leg ulcer(s) due to venous insufficiency
2) Patient and/or caregiver(s) able and willing to participate in decision making and venous ulcer treatment
3) Resting ankle-brachial index equal to or greater than .8

EXCLUSION CRITERIA:

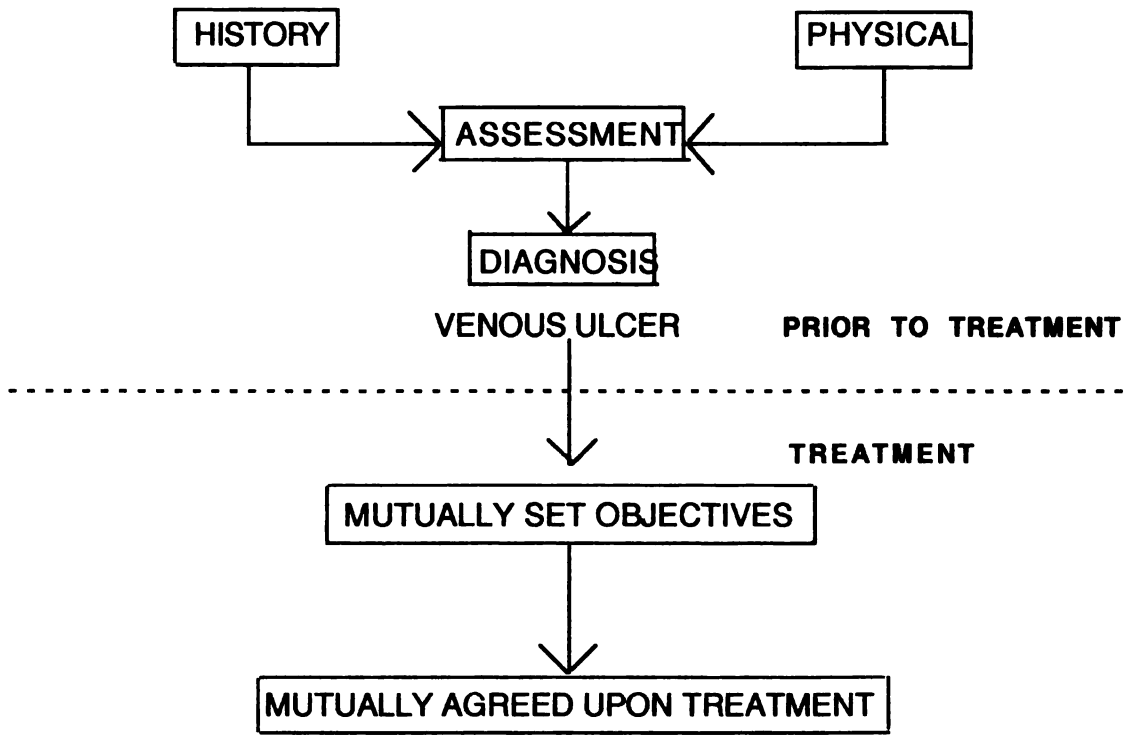
1) Diagnosis of arterial insufficiency/peripheral vascular occlusive disease
2) Resting ankle-brachial Index of less than .8
3) Leg ulcers due to all other etiologies besides venous insufficiency
4) Patient and/or caregiver(s) unable or unwilling to participate in decision-making and venous ulcer treatment

Please review the protocol with the patient/caregiver and complete the checklist during each visit. Provide and review the education sheet during the first visit and prn.

******PLEASE SIGN OUT EACH PROTOCOL YOU TAKE AND INDICATE FOR WHOM IT WILL BE USED.

APPENDIX B

PROTOCOL FOR VENOUS ULCER TREATMENT



CONTROL OF UNDERLYING MEDICAL AND NUTRITIONAL DISORDERS

TREATMENT OF INFECTION

DEBRIDEMENT OF NECROTIC TISSUE

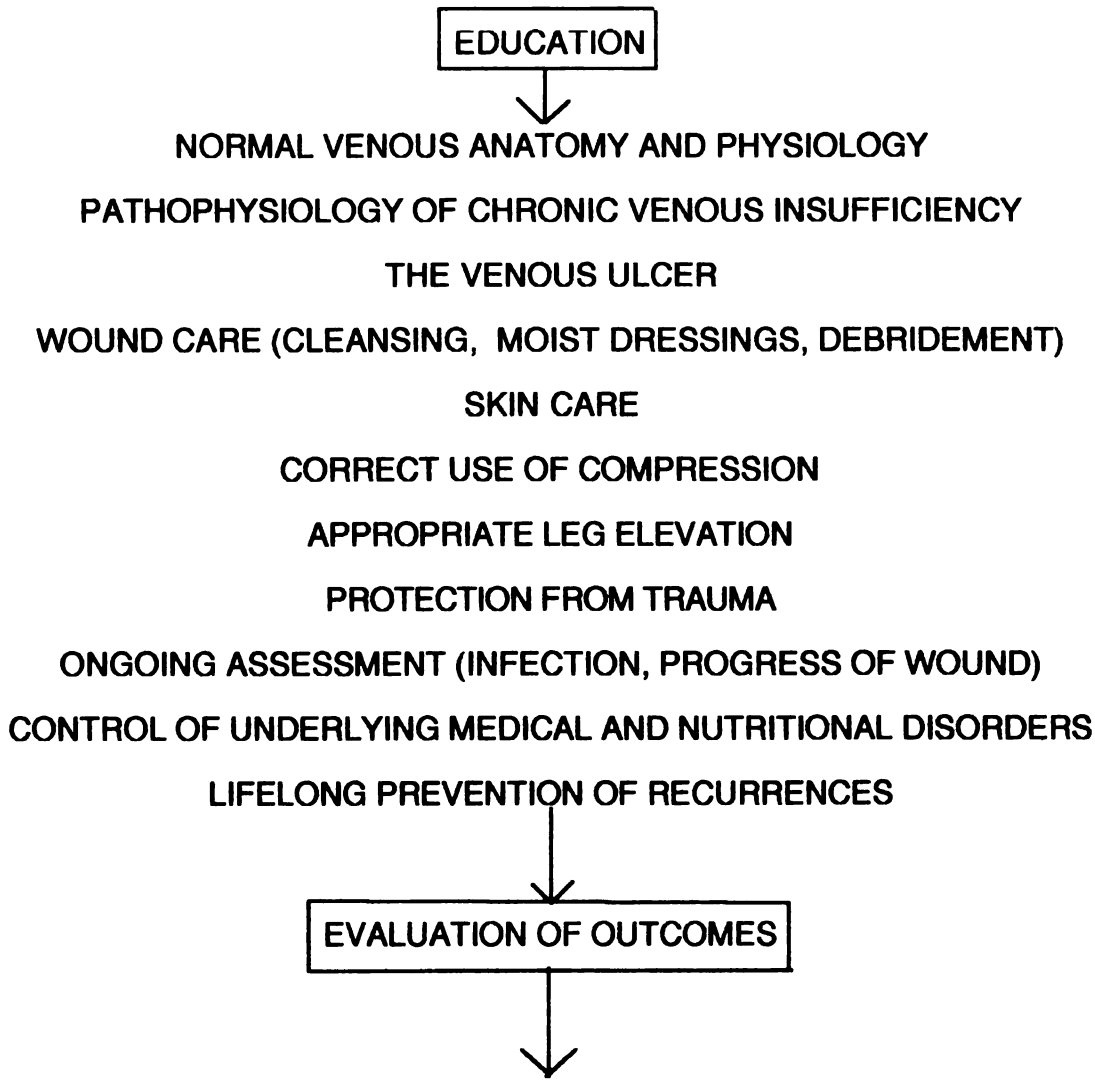
REDUCTION OR ELIMINATION OF EDEMA

STIMULATION OF FIBRINOLYSIS/

MAINTENANCE OF A MOIST WOUND ENVIRONMENT

SKIN CARE

LIFELONG PREVENTION/MAINTENANCE


EDUCATION
NORMAL VENOUS ANATOMY AND PHYSIOLOGY
PATHOPHYSIOLOGY OF CHRONIC VENOUS INSUFFICIENCY
THE VENOUS ULCER
WOUND CARE (CLEANSING, MOIST DRESSINGS, DEBRIDEMENT)
SKIN CARE
CORRECT USE OF COMPRESSION
APPROPRIATE LEG ELEVATION
PROTECTION FROM TRAUMA
ONGOING ASSESSMENT (INFECTION, PROGRESS OF WOUND)
CONTROL OF UNDERLYING MEDICAL AND NUTRITIONAL DISORDERS
LIFELONG PREVENTION OF RECURRENCES
EVALUATION OF OUTCOMES
CLIENT/CAREGIVER SATISFACTION
ADHERENCE TO CARE PLAN
ACHIEVEMENT OF MUTUALLY SET GOALS
ABSENCE OF INFECTION
REDUCED WOUND SIZE/COMPLETE HEALING
PERMANENT LIFESTYLE CHANGES TO PREVENT RECURRENCES
NO RECURRENCES
NEED FOR FURTHER ASSESSMENT?

APPENDIX C

TREATMENT PLAN FOR VENOUS ULCER(S)

PATIENT NAME:	ID#

Document with each contact: date, intervention and initials of provider.

	date	initials
1. Objectives set mutually with patient/caregiver.		
2. Treatment Plan mutually agreed upon with patient/caregiver.		
A. Control of underlying medical conditions		
B. Control of nutritional disorders		
C. Treatment of Infection as indicated.		
1. Wound culture obtained?		
2. Topical or systemic antibiotics.		
D. Debridement of necrotic tissue as indicated.		
1.Sharp, blunt, enzymatic, autolytic		
E. Reduction or elimination of edema		
1. Graduated Compression Hose		
2. Ace Wraps		
3. Zinc Oxide Impregnated Wraps		
4. Leg elevation above heart level		
5. Encourage walking, leg movement		
F. Stimulation of Fibrinolysis/Maintenance of a Moist Wound Environment		
Type of dressing: hydrocolloid, foam, alginate, etc. with frequency of change		
G. Skin care		
H. Discussion of lifelong prevention measures		
I. Referral(s) made as indicated		

I agree with the above treatment plan: _____
(Patient/caregiver signature and date)

APPENDIX D

TEACHING GUIDE FOR VENOUS ULCER TREATMENT

Patient (Caregiver) Name:			ID#	
Document with each contact: Date of instruction, initials of instructor and client response code.	#1	#2	#3	#4
1. The Client/Caregiver has a basic understanding of leg anatomy and physiology.				
2. The Client/Caregiver has a basic understanding of chronic venous insufficiency.				
3. The Client/Caregiver can describe a venous ulcer.				
4. The Client/Caregiver can verbalize/demonstrate basic managementof a venous ulcer.				
A) Edema control via elevation, compression, and ambulation/leg movement				
B) Moisture retentive dressing use				
C) Debridement prn				
D) Protect from trauma				
E) Good skin care				
F) Treatment of infection prn				
5. The Client/Caregiver can verbalize symptoms to report to provider.				
A) Infection				
B) Allergic reaction				
C) New or larger leg wound(s)				
D) Anything unusual				
6. The Client/Caregiver can verbalize basic lifelong prevention behaviors.				
A) Edema control as above				
B) Good skin Care				
C) Protect from trauma				

Initials Signature

____ _____

____ _____

____ _____

Any follow-up phone calls? Y N

Dates:

Client Response Codes:

1. Good Comprehension
2. Fair Comprehension
3. Poor Comprehension
4. More Instruction Needed
5. Materials Given
6. Patient able to read

APPENDIX E

PATIENT/CAREGIVER EDUCATION FOR VENOUS ULCERS

Leg Veins

Arteries and veins are elastic tube-like vessels that carry blood to and from every cell in the body. Arteries in the legs take the blood down or away from the heart while veins carry blood back to the heart. Arteries and veins are connected by capillaries (very tiny blood vessels). There are many veins in the legs, some shallow and some deep, some large and some small; but each one contains one-way valves that keep the blood moving back toward the heart against gravity while you stand or sit with your legs down. Standing still increases the blood pressure inside the leg veins, while walking reduces the pressure since leg muscles act as a pump to keep blood moving in the veins.

Why a Venous Ulcer Develops:

Sometimes the muscles of the leg can no longer pump the blood in the veins back toward the heart efficiently because the valves aren't closing tightly or because some other problem has developed in the veins. The arteries keep bringing the blood down the leg, but the veins aren't taking the blood back fast enough when you stand. The pressure in the veins goes up and stays up. This high blood pressure in the veins causes fluid (not blood) to leak out of the capillaries into the tissues of the leg which causes swelling. This fluid can cause a material to develop around the capillaries that interferes with food, oxygen and waste exchange that cells in the leg need to live. Skin and fat cells that don't get enough nourishment and oxygen can die, which can cause an ulcer or wound to develop. You will see enlargement of the tiny veins around the ankle, swelling in the lower leg and foot, and maybe even skin pigmentation changes with itching and scaling long before an ulcer develops.

What is a Venous Ulcer?

A venous ulcer is a break in the skin (a wound) of the lower leg, that can develop after you lightly bump your leg or even for no apparent reason. The ulcer(s) usually develops around the ankle, can be shallow or deep, big or small, very painful or not painful at all, usually drains a lot, often smells, and may get infected. These ulcers take weeks, months, or even years to heal.

Venous Ulcer Treatment

Once you see your health care provider about your venous ulcer, treatment needs to begin immediately. You are an equal partner with your provider in deciding what your goals and treatment plan are. Your participation in the office and at home is critical so that your venous ulcer(s) can heal, and that you will reduce your risk of developing more ulcers in the future.

Things that need to be done now:

***Treatment of infection (if present)**--antibiotics may be ordered.

* **Wound care**--including wound irrigation each time the dressing is changed, applying the correct moisture-retentive dressing and changing it as needed, and cleaning any dead tissue out of the wound (which will be done as needed by your health care provider). You will learn how to do this yourself.

***Control of leg swelling**--a critical part of the treatment. This is achieved by (1) elevating your legs frequently above the level of your heart when lying down, (2) wearing graduated compression hose or other leg wraps (even over dressings) except when you are in bed for the night , and (3) walking often with compression hose or leg wraps on and keeping your legs moving.

***Protecting your legs from injury**--avoid contact sports, jumping dogs, scratching cats, biting insects, razors, heat, bare feet, tight shoes, etc.

***Getting other medical conditions that you may have under good control**. Diabetes, anemia, heart and kidney failure, malnutrition, obesity, cancer and even certain drugs can delay or prevent ulcer healing .

***Taking good care of the skin of your legs**. You can prevent the skin of your legs from drying out by reducing bath frequency, using mild soaps or soapless cleansers, and applying petroleum-based creams and emollients to intact skin right after your bath. Keep your legs clean. Don't apply lotions or creams on your leg ulcer.

***Observing your wound and legs closely at home**. Look for signs of infection in your wound or leg such as increased pain, heat and redness, pus, foul odor, or fever. Look for increased or new swelling or color changes in the feet, ankle or legs. Look for signs that you might be allergic to the dressing or cream or lotion you apply to your wound or leg. You might notice a rash, increased redness, weepy, itchy skin that involves the area that came into contact with the dressing or ointment or lotion. Look for new ulcers or enlargement of your existing ulcer. If you notice anything unusual, call your health care provider.

Lifestyle Changes for Lifelong Prevention:

Once your ulcer heals, things you do at home everyday can prevent another venous ulcer from developing. You have a lifelong problem that cannot be ignored. You have probably already made the necessary changes. This is not the time to go back to old habits of not wearing compression hose, standing for long periods of time, not taking walks and elevating your legs, not taking good care of the skin of your legs and avoiding trauma to your legs. Critical things you need to do every day are elevating your legs above your heart several times for 10-15 minutes, applying compression to your legs, and keeping your legs moving. Good skin care and prevention of leg trauma to your legs will help prevent ulcers. Stop smoking (if you do) and avoid weight gain.

It is up to you!!!! Venous ulcers are expensive, inconvenient, unsightly, messy, smelly, can get infected and even can disable you. The good news is that they often are preventable!!! We want you to be the best that you can be in partnership with your health care provider.

Patient Name:_____Date:_____

APPENDIX F

PATIENT EVALUATION OF VENOUS ULCER TREATMENT

Patient Name (Caregiver)	Date	Date Plan Initiated	ID#

Please complete the following questions by circling the number as it most closely represents your feelings for each question.

1. None of the time
2. Less than half of the time
3. About half the time
4. More than half the time
5. All of the time

A. I am satisfied with the treatment plan for my venous ulcer. 1 2 3 4 5

B. I believe that I was provided enough education to make the right decisions, follow the plan of care, and feel in control. 1 2 3 4 5

C. I am able to follow the plan of care. 1 2 3 4 5

D. I am following the plan of care. 1 2 3 4 5

E. I believe that the goal(s) will be met. 1 2 3 4 5

F. I plan on changing my behaviors for life to prevent more future ulcers. 1 2 3 4 5

Please answer the following questions by circling yes (Y) or no (N).

G. The goals and treatment plan were mutually established by me and my health care provider. Y N

H. Did infection develop in your wound after treatment was initiated? Y N

I. Has your venous ulcer(s) reduced in size since treatment began? Y N

J. Has your venous ulcer(s) healed since treatment began? Y N

APPENDIX G

PROVIDER EVALUATION OF VENOUS ULCER TREATMENT PROTOCOL

Please answer each question honestly. Please circle yes (Y) or no (N). Your answers will be anonymous unless you choose to identify yourself. Please add any comments on the reverse side.

1. Were you adequately prepared to begin using the protocol? Y N
2. Were you satisfied with the protocol for leg ulcer treatment? Y N
   Why or why not?
3. Does the protocol include too many items? Y N
4. Does the protocol include too few items? Y N
5. Did you mutually set goals with your patient/caregiver? Y N
6. Did you mutually decide upon a treatment plan with your patient/caregiver? Y N
7. Was/were the objective(s) met? Y N
8. Did the protocol take up too much of your time? Y N
   How much more time did it take, than you would have otherwise spent, with this patient/caregiver?
9. Did your patient/caregiver comply with the treatment plan? Y N
10. Did your patient develop a wound infection? Y N
    If yes, did the infection develop before the treatment plan was implemented? Y N
11. Did the venous ulcer(s) reduce in size? Y N
12. Did the venous ulcer(s) heal completely? Y N

Please add any additional comments on the reverse side.

Optional Signature_____ Date_____

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