



# Lymphedema, lipedema, and the open wound

## The role of compression therapy

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Lymphedema is a chronic, incurable condition (with the exception of localized lymphedema secondary to trauma and wounds). It is characterized by an abnormal collection of fluid as a result of an anatomical alteration to the lymphatic system. It is estimated that 1 person in 30 is afflicted with lymphedema worldwide, though this figure does not include the millions suffering from chronic venous disease [1].

Lymphedema can be categorized as either *primary* or *secondary* lymphedema. Primary lymphedema is a congenital malfunction of the lymph system resulting in impaired lymph node or lymph vessel development and accounts for 10% of all lymphedema patients. Milroy's disease (or Nonne-Milroy disease) is lymphedema that is present at birth, and it comprises 15% of primary lymphedemas. The symptoms of primary lymphedema may not be apparent until the second or third decade of life. This form of primary lymphedema may present as lymphedema praecox, which appears in adolescence, and lymphedema tarda, which begins after 35 years of age. The distribution in cases of primary lymphedema between the sexes is reported to be 87% in women and 13% in men [2]. Primary lymphedema occurs most often in the legs and is rare in the upper extremities and face.

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Secondary lymphedema can be caused by many factors. The most recognizable are associated with lymphadenectomy, radiation, venous disease, and numerous postsurgical complications. Lymphedema secondary to vascular reconstruction, joint replacement, and venous harvesting in conjunction with coronary bypass comprises an ever-growing problem. Periwound (localized) lymphedema in acute and chronic wounds is now recognized as a major inhibitory factor in wound healing [3]. The most common cause of lymphedema is filariasis, a disorder caused by infection with larvae that is transmitted to humans by mosquitoes and infects more than 90 million people worldwide. In the Western Hemisphere, filariasis is now confined to isolated areas of the Caribbean and South America, predominately Haiti and Brazil.

### **Lipedema**

Lipedema is a chronic disease of lipid metabolism resulting in the symmetrical impairment of fatty tissue distribution and storage combined with hyperplasia of individual fat cells. This abnormal distribution of fat is usually seen between the pelvic crest and the ankle so that unless the lymphatic system has been damaged, the feet appear normal (Fig. 1). Swelling often progresses during the day as the diminished tissue resistance of fatty tissue permits the accumulation of orthostatic edema. A positive family history has been reported in 20% of patients diagnosed with lipedema [4,5]. Eventually, in many patients, mechanical insufficiency of the lymph system occurs, leading to true lymphedema. Lipolymphedema is a combination of impaired fat distribution and an impaired lymphatic system.

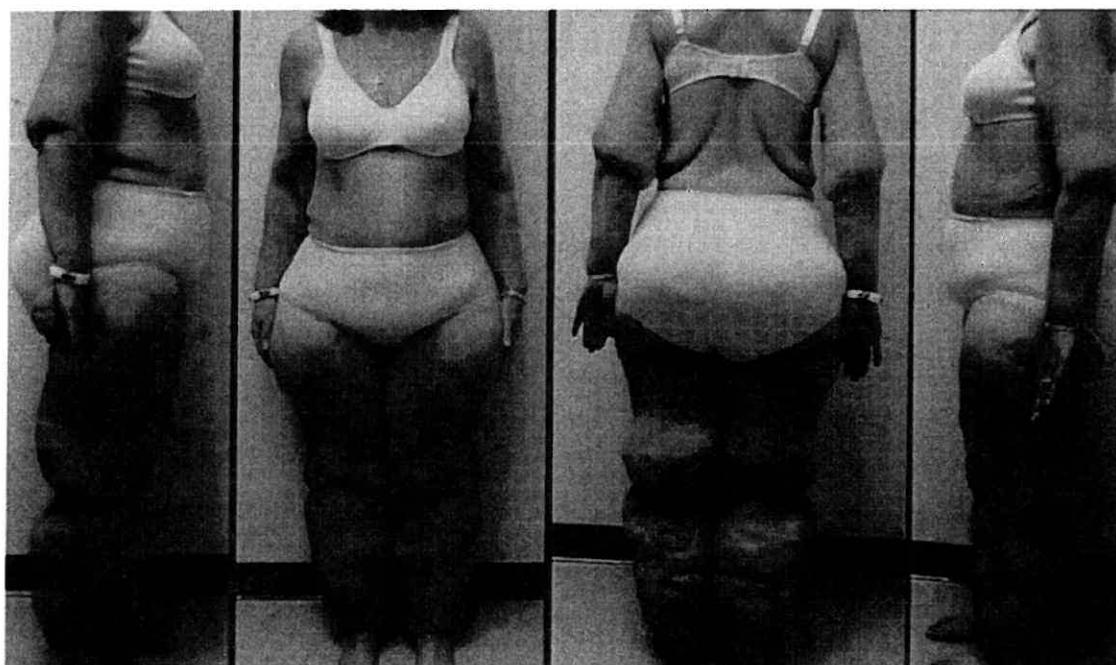


Fig. 1 Lipedema

Lipedema occurs almost exclusively in women. Men develop this condition only in association with feminization (ie, hepatic cirrhosis, hormonal therapy in prostatic carcinoma). In most cases, lipedema develops during puberty. Heavy hips and thighs are obvious signs. Simultaneous incidence in the upper extremities is rare, but when involved, there is often a large fold of loose hanging skin visible when the patient elevates her arm to the horizontal position (Box 1).

Medical history and physical examination make the diagnoses of lipedema. Special diagnostic procedures or additional laboratory tests are rarely necessary to establish the diagnoses. The main differential consideration is lymphedema. In contrast to lymphedema, lipedema is symmetrical, often painful to palpation, susceptible to easy bruising and the patients rarely develop cellulitis. The Stemmer Sign (thickened cutaneous folds on the dorsum of the toes that are difficult to lift), which is considered pathognomic for lymphedema, is negative [6]. The patient may show varying degrees of obesity and may report many unsuccessful attempts at dieting. Often the patient will relate that dieting resulted in weight loss only in the upper part of the body, while the tissues of the lower body remained soft and rubbery as opposed to the hard and fibrotic tissue seen with chronic lymphedema.

Therapy for lipedema is largely palliative or is directed at comorbidities. Lipolymphedema and chronic venous insufficiency are treated by the usual methods. Perhaps the most important service provided by the physician is emotional support and reassurance that this disability is not the patient's fault, and that lipedema truly is a congenital abnormality. Dietary measures are advised only for control of superimposed obesity. At the present time, lipectomy and liposuction would seem to be contraindicated for fear of iatrogenic damage to the lymphatics resulting in deficient lymph drainage. These procedures should be used with extreme caution. Until recently, bariatric surgery has not been found to significantly affect the abnormal fat distribution and metabolism seen in lipedema.

### **Box 1. Lipidema—clinical findings**

- Females almost exclusively
- Develops during puberty
- Heavy hips and thighs most common
- Swelling ends at ankles
- Palpation usually painful
- May be associated with lymphedema
- Cellulitis infrequent
- Symmetric
- Stemmer sign negative
- Frequent black and blue marks

## Anatomy of the lymphatic system

A detailed description of the anatomy and physiology of the lymphatic system is beyond the scope of this review. However, some fundamentals are necessary to understand the rationale of current therapy for lymphedema and wound-related lymphedema. The lymphatic system consists of lymph vessels and regional and central lymph nodes. *Initial lymph vessels* is the collective term given to the valveless lymphatic capillaries and the single and double valve precollectors. The initial lymphatics provide liquid resorption, while the lymph collectors are transport vessels. The lymph collectors lead to lymphatic ducts that form the main parts of the transporting vessels and the thoracic duct. The lymphatic ducts do not have resorbing function. The extremities contain a superficial (above the fascia) and a deep (below the fascia) lymphatic system. The transport of lymph occurs not only from distal to proximal, but also from the superficial to the deep system and vice versa. The collectors generally follow the course of the blood vessels and lead to the regional lymph nodes. However, throughout the body, variations in the vessel course and directional flow are of clinical importance.

The body—both the internal organs and the skin—is divided into a series of lymphatic drainage regions by “lymphatic watersheds.” Watersheds are dividing areas free of lymph-collecting vessels that contain only initial lymph capillaries. On either side of these dividing lines, the flow of lymph drains in different and, in general, opposite directions (Fig. 2). These lymphatic drainage areas are called *lymphotomes*. Variations in lymphatic vessel course are frequent. Knowledge of the regional anatomy of the lymphatic system enables the therapist to redirect lymphatic flow into functioning collateral circulation after lymph node removal or destruction. For example, in drainage of the lower extremity, the inguinal lymph nodes may be circumvented by lymph collectors that course along the sciatic nerve and end in the internal iliac lymph nodes. Likewise, the cephalic lymph vessels that originate in the region of the wrist, anastomose with nearby lymphatic pathways and end directly in supraclavicular lymph nodes. This allows functioning collateral lymphatic circulation to be used after the removal of the axillary lymph nodes during the management of breast cancer.

## Lymphedema versus edema

Protein content defines the difference between edema and lymphedema. Lymph is waste material that is transported from the interstitium back to the venous system. It consists primarily of proteins, fats, and cells. Water functions as a transport medium. Protein cannot be reabsorbed in the interstitial tissue and must be transported by the lymphatic vessels. Resorption of excess water occurs through venous drainage. Lymph also contains chemicals, organic and inorganic cell products and fragments, and bacteria and viruses. The filtration and resorption process are controlled by



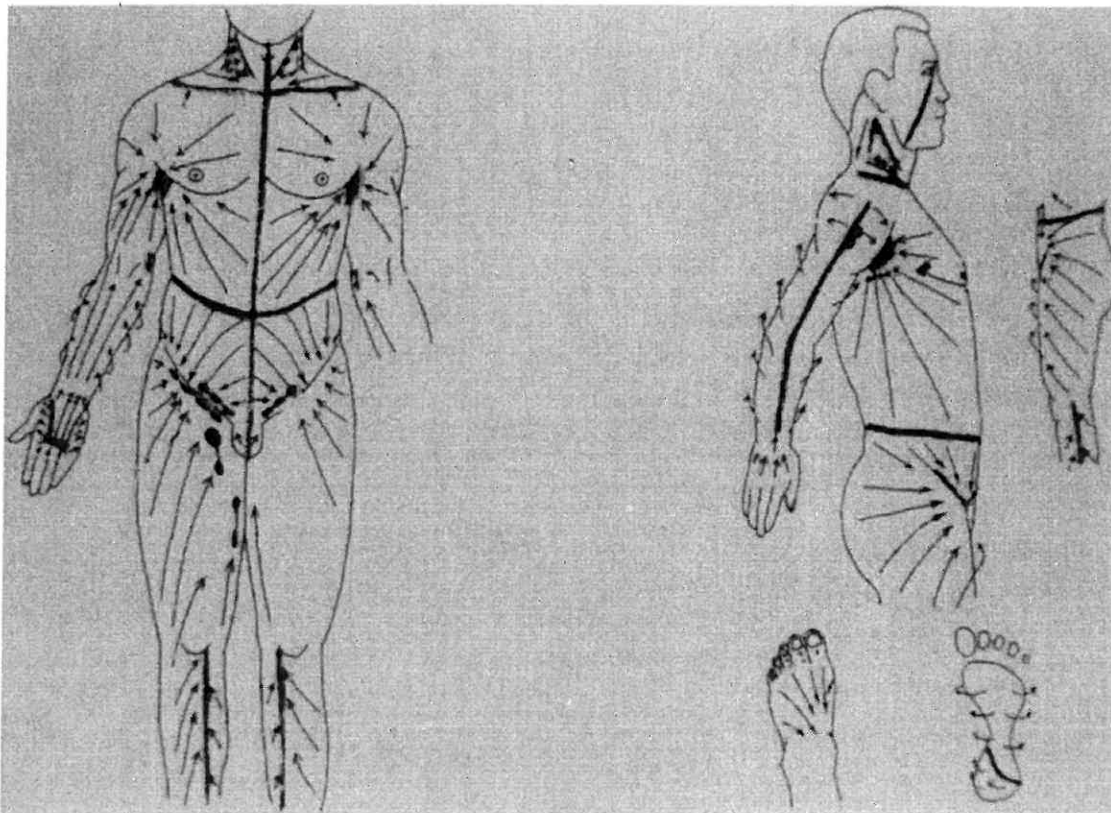


Fig. 2. Lymphatic watersheds.

capillary pressure, surrounding tissue pressure, and colloid osmotic pressure (Starling's Equilibrium).

Traditionally, the term *edematous* is used to describe any limb or organ that becomes swollen. An intact lymphatic system can accommodate an increased amount of lymph with a higher transport capacity; however, this functional reserve is limited. When compensation possibilities are overwhelmed, the system will fail to cope with the increased amount of lymph and, in an already damaged lymphatic system, even normal amounts of waste material can exceed transport capacities. The increased protein concentration in the tissue results in chronic inflammation with subsequent cell proliferation (macrophages, fibroblasts, lymphocytes), which leads to fibrosis in the edematous tissue. Ultimately, this causes dilatation of the afferent lymph vessels resulting in lymphatic valve insufficiency, endothelial damage, reduced lymphflow, and a further increase in protein-rich interstitial edema. Thus, two classifications of edema—high-protein edema and low-protein edema—should be used [7]. Lymphedema is a high-protein edema, which is the result of damage or absence of the normal lymphatic system. Edema, in contrast to lymphedema, is mostly water. The arbitrary dividing line of protein concentration in edema fluid is 1 g/dl (ie, 1 g%). The concentration of protein in high-protein edema may actually be lower than the concentration of normal tissue (approximately 2–3 g/dl).

**Box 2. Lymphedema classification***Primary*

- Birth
- Praecox-adolescent
- Tarda-Age 35+

*Secondary*

- Surgery
- Infection
- Trauma
- Chronic Wound
- Tumor
- Radiation
- Venous Disease
- Neurological
- Filariasis

Lymphedema is the result of a low-output failure of the lymphatic system (Box 2). In all forms of lymphedema, primary and secondary, the common denominator is that lymphatic transport falls below the capacity required to handle the presented load of normal microvascular filtrate. Edema, on the other hand, is a high-output failure of the lymph circulation (Box 3). This takes place when a normal or increased transport capacity of the intact lymphatics is overwhelmed by an excessive flow of filtrate. Common examples

**Box 3. Edema classification***Passive hyperemia*

- Chronic venous insufficiency
- Congestive heart failure
- Pregnancy
- Inactivity

*Hypoproteinemia*

- Malnutrition
- Malabsorbtion
- Renal disease

*Active hyperemia*

- Inflammation
- Allergy

of edema include congestive failure, hepatic cirrhosis (ascites), chronic venous insufficiency (peripheral edema), and nephrotic syndrome (anasarca).

The lymphedema associated with acute and chronic wounds is sometimes called *posttraumatic lymphedema*. Perhaps *posttraumatic lymph stasis* would be a more appropriate term. Acute trauma is usually followed by a transudative low-protein edema. If the lymphatic collecting anatomy is initially damaged (eg, in an open wound), true lymphedema rapidly develops. Unless the lymphedema is pre-existing, collectors proximal and distal to the lesion are normal [7].

### Classification of lymphedema

Clinical staging has proven useful for the classification of lymphedema [8]:

Stage I: spontaneous, reversible, tissue swelling leaving indentations; negative or borderline Stemmer sign; no palpable fibrous tissue.

Stage II: spontaneous, irreversible tissue swelling with moderate or pronounced fibrosis. Indentations are difficult to produce. Stemmer sign is positive; lymphostatic dermatosis.

Stage III: lymphostatic elephantiasis, usually with pronounced skin alterations.

Severity is based on differences in limb volume are assessed as minimal (<20% increase), moderate (20%–30% increase), or severe (>40% increase).

### Diagnoses of lymphedema

An accurate diagnosis of lymphedema and its underlying cause is essential for appropriate therapy. This can be accomplished in most patients through a detailed history and physical examination. In many patients, however, comorbidities such as venous insufficiency, metastatic disease, morbid obesity, and repeated infections may complicate the clinical presentation. It is especially important when analyzing unilateral extremity lymphedema that significant venous disease and occult visceral tumors are considered. Related diseases such as diabetes mellitus, congestive heart failure, and peripheral vascular occlusive disease will also influence the therapeutic approach. With this in mind, it seems obvious that informed physician input is required before beginning lymphedema therapy.

Imaging for diagnoses can be helpful if further definition is required. Noninvasive duplex-Doppler studies and, rarely, phlebography, may be required if venous disease is suspected. Computed tomography (CT), MRI, and ultrasonography are also useful in some patients. Lymphoscintigraphy (LAS) is useful for demonstrating the detailed lymphatic pathology. LAS provides images of lymphatics and lymph nodes as well as semiquantitative

data on radiotracer (lymph) transport. However, the limited availability of experienced specialists in nuclear medicine and the limited ability of LAS to influence therapy would seem to restrict its use to research programs.

Genetic testing can define a limited number of hereditary syndromes as in some forms of Milroy's disease. In the future, such testing may become routine and could hold promise for specific gene therapy.

### **Therapy for lymphedema**

Untreated lymphedema is most often a progressive, debilitating, and chronic disease. The complications seen with lymphedema are the result of lymph stasis, excess fibrosis, and contraction (Box 4). Ultimately, untreated lymphedema produces severe physical sequelae as well as psychological and functional impairment. A consensus document on the diagnosis and treatment of peripheral lymphedema was published in 2001 by the International Society of Lymphology Executive Committee; their recommendations and discussion are cited in this article [9].

Therapy for peripheral lymphedema is divided into nonoperative (conservative) and operative methods. This discussion does not apply directly to wound-related lymphedema, which will be detailed in a following section. The principles of modern lymphedema treatment, however, have direct application to the lymphedema–wound-healing equation.

#### *Nonoperative therapy*

Comprehensive decongestive physiotherapy (CDP) is the gold standard of treatment for primary and secondary lymphedema [9,10]. CDP involves a two-phase treatment program. The first phase consists of daily therapy sessions of specialized manual lymph massage and drainage, range of motion exercises, skin care, and compression wrapping applied with multilayered short stretch bandages. The patient wears the bandages during

#### **Box 4. Consequences of chronic lymphedema**

- Protein-rich interstitial edema (lymphedema)
- Fibrosis, sclerosis
- Disturbance of local metabolism
- Increased infection rate (cellulitis)
- Progressive lymphatic damage
- Lymphatic arthropathy
- Chronic wounds
- Malignant degeneration



the intervals between clinic visits. The therapy sessions vary between 1 and 2 times per day and may be continued for 2 to 4 weeks or longer, depending on the severity of the disease.

The second phase, which is initiated immediately after completion of the first phase, is programmed to conserve and optimize the benefits achieved from the start of therapy, and is a lifetime commitment by the patient. Fitted compression garments are used daily. Exercises designed to improve systemic lymphatic flow and self-administered manual lymph drainage (MLD) enable the patient to not only maintain the achieved lymphedema reduction but also, in many cases, to improve upon the initial reduction. The success of CDP depends on the availability of physicians (ie, clinical lymphologists,) nurses, and therapists trained and certified in this specialized technique (for reference: National Lymphedema Network (NLN), 1-800-541-3259). Of perhaps greater importance is the compliance and willingness of both the patient and family members to continue therapy. New medical compression devices (eg, Reid Sleeve<sup>®</sup>, Circ-aid<sup>®</sup>) have often been helpful in relieving some patients of nighttime bandage requirements. A dramatic example of the effects of CDP over a 4-month period is shown in Fig. 3.

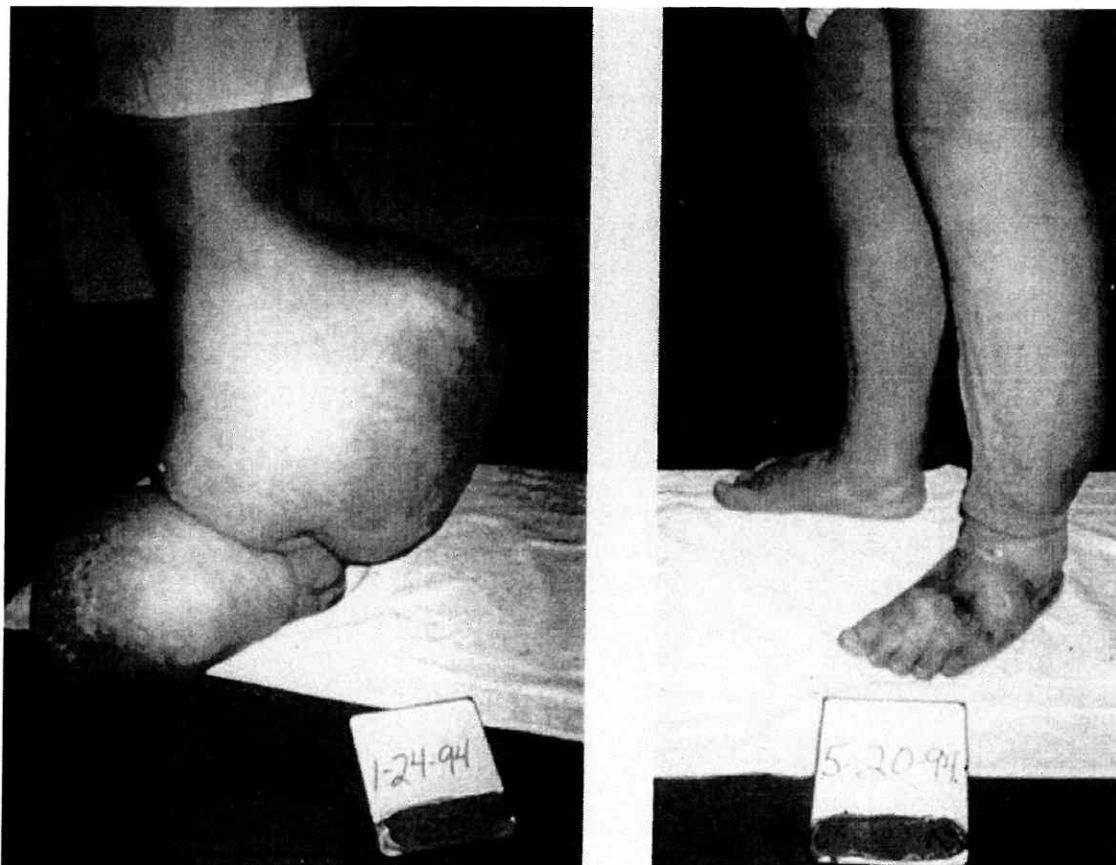


Fig. 3. Comprehensive decongestive physiotherapy. Treatment duration: 4 months. (Courtesy of Gunter Kloese, Danville, PA.)

CDP as practiced today was introduced, applied and refined by Drs. Michael Foldi and Ethel Foldi in Germany [11]. This method has been further modified through the contribution of the Casley-Smiths in Australia [12]. The Foldis have treated 2500 patients annually with CDP. Limb volume reductions averaged 50% after completion of the course of therapy. More than 50% of patients maintained their reduction during the second phase of treatment [11]. The Casley-Smiths reported volume reductions of over 60% in 618 lymphedematous limbs [13]. The pioneering work of Lerner and Boris introduced CDP to North America in the 1980s [14,15]. Boris et al [16] published results in 119 consecutive patients; the affected limbs included both arms and legs. Lymphedema reduction averaged 62.6% in 56 patients with one affected arm and 68.6% in 38 patients with one affected leg. After 36 months follow-up, the average reduction in the arms increased to 63.8% and remained at 62.7% in the affected legs.

Intermittent pneumatic compression by a sequential gradient pump, if prescribed, must be used with caution and under strict supervision. The indiscriminant use of pneumatic pumps without the added resource of MLD can result in further lymphatic damage as well as significant pelvic and opposite limb swelling [17]. MLD should be used to decrease fluid proximal to the obstructing lesion, thereby improving the efficiency of lymph mobilization toward the thoracic duct and venous circulation. The role of thermal therapy and pulsed radio frequency energy in the treatment of lymphedema remains unclear.

### *Drug therapy*

Diuretic agents may sometimes be useful during the early phase of CDP. However, long-term administration of diuretics in the treatment of lymphedema is of little benefit. Diuretics may induce fluid and electrolyte imbalance by decreasing the water content of the lymph fluid and increase the viscosity, thereby hindering the mobilization of lymphedema [9]. Oral benzopyrones (coumarin) are thought to hydrolyze tissue proteins and facilitate absorption while stimulating lymphatic collectors [13]. Benzopyrones have not been approved for use in the United States because they have been linked to liver toxicity, and dose regimens are still in question. The efficacy of nutritional supplements in treating lymphedema has not been proven.

### *Operative therapy*

Surgical approaches to alleviate extremity lymphedema by improving lymph return have not been widely accepted. Reconstructive methods such as interposition vein segment and autologous lymph vessel transplantation are very demanding and require extensive experience in lymphology and microsurgery. Long-term patency and improved lymphatic transport require future confirmation

Resection or “debulking” may be useful in some cases of advanced elephantiasis. After aggressive CDP, redundant skin folds may be suitable for excision. However, extensive surgery has the major disadvantage of removing or damaging superficial lymphatic collaterals; postoperative problems with healing and infection can be formidable. Omental transposition, enteromesenteric bridge operations, and the implantation of tubes or threads to promote substitute lymphatics have not demonstrated long-term value. Liposuction has recently been modified to successfully treat nonfibrotic upper extremity lymphedema [18]. Short-term results seem encouraging, but strict patient compliance is required with continued use of low-stretch compression garments and supportive CDP.

### **Lymphedema and wound healing**

To appreciate the relationship of lymphedema to wound healing, clinicians should review their knowledge of chronic venous insufficiency and venous stasis ulceration. Chronic venous insufficiency leads to venous hypertension, which results in a high filtration pressure causing increased fluid to appear in the tissues (ie, increased lymphatic waterload). When the lymphatic transport capacity is exceeded by the waterload, a state of low protein edema occurs as a result of this dynamic failure. Constant lymphatic hypertension causes infiltration of lymph into the perilymphatic tissue, resulting in fibrosclerosis and lymphangitis. Protein permeability increases and lymphatic damage follows. Subsequently, lymphedema (high protein edema) becomes the underlying pathology that contributes to the formation of venous stasis ulcers.

Venous stasis ulcers often exhibit many of the characteristics of the nonvenous chronic wound: normal arterial blood supply, colonized bacterial contamination, and healthy granulation tissue. With compression and control of the lymphedema, these wounds will heal in most cases. Given the same parameters in nonvenous, acute, and chronic wounds throughout the body, controlling the peri-wound lymphedema will result in enhanced wound healing [19].

In the authors’ clinic, more than 80% of patients presenting with nonvenous, chronic wounds of the lower extremity have demonstrated generalized or peri-wound lymphedema (data accumulated from April 2000 to March 2001). The degree varied from trace to 4+ pitting. These findings were seen in many types of wounds (eg, ischemic, diabetic, and traumatic). Eliminating lymphedema appeared to improve healing. In many instances of long-standing wounds, the elimination of lymphedema enhanced the rate of healing dramatically.

Although lymphedema control in the therapy of venous ulcers is widely accepted, the same principle as applied to nonvenous ulcers has rarely been discussed. Lymphedema/edema is rarely listed as an inhibitory factor to wound healing in journal articles and textbooks. A recent preliminary

Internet search using the phrase “wound healing + lymphedema” resulted in just two matches [3,20]. In most articles, the relationship is discussed only in relation to venous disease.

### **Clinical observations**

How do clinical observations translate into evidence-based conclusions at the clinical and basic science level? Future research will be required to define exactly how lymphedema inhibits wound healing. However, considerable information currently exists to support these assumptions.

The most obvious effect from lymphedema is swelling. This can result in abnormal function at both the tissue and cellular level. Distance between tissue channels can affect metabolic exchange, causing a shift toward anaerobic metabolism. Because cells are more widely separated, the exchange of gases between plasma membranes is likely to be affected. In chronic venous insufficiency, the removal of lymphedema results in a significant increase in transcutaneous oxygen tension [21]. Capillaroscopy studies have shown that the density of skin capillaries increases as a result of edema reduction [22].

Alterations in tissues produced by simple injections of protein are almost identical with the changes observed in subacute and chronic lymphedema [23]. Mani and Ross [24] state: “The chronic effects of edema on the visco-elastic properties of connective tissue are unknown. It is reasonable to assume that pools of edema will squash, squeeze, stretch or affect the crimping and orientation of dermal collagen bundles.”

Open wounds studied by the injection of dye have demonstrated significant reduction in lymphatic channel regeneration compared with arterial and venous angiogenesis [25]. Trauma increases lymphatic flow, and outflow obstruction with the accumulation of waste products generated in the wound-healing process is a likely inhibitory factor in wound healing [26]. Tissues surrounding acute and chronic wounds are characterized by collections of interstitial or third space fluid. This collection of fluid mechanically compromises the microvascular and lymphatic system, thereby increasing capillary and venous afterload. Consequently, the delivery of oxygen and nutrients and the discharge of toxins and inhibitory factors are affected [27,28].

It is believed that removing excess chronic wound fluid also removes inhibitory factors present in the fluids. Studies have shown that fluids removed from chronic wounds suppress the proliferation of keratinocytes, fibroblasts, and vascular endothelial cells *in vitro* [29,30]. Argenta and Morykwas [31], in their investigations related to vacuum-assisted closure of wounds, have provided valuable insight into the consequences of lymph stasis and the healing wound. Their technique removes chronic lymphedema, which contributes to increased localized blood flow and enhanced formation of granulation tissue.



## Lymphedema therapy and the open wound

MLD and continued compression are the cornerstone of treatment for nonwound-related lymphedema. However, using MLD to alleviate lymphedema associated with the open wound is neither timely nor cost-efficient. After the wound has healed, CDP may be indicated if persistent swelling is a problem. In the treatment of chronic venous leg ulcers, improving the efficiency of the calf muscle pump by using compression bandages is widely accepted as essential for proper care. Thus it is reasonable to postulate that compression is the appropriate therapy for lymphedema/lymph stasis associated with the open nonvenous wound. Limb elevation, when practical, is obviously helpful. In addition to the accepted dictums of modern wound care, the purpose of compression is to create a dynamic wound dressing. As has been mentioned, diuretics are rarely indicated as primary therapy and can actually impair fluid mobilization by extracting water from the lymph [9]. Diuretics are useful for treating limb swelling when a significant degree of edema superimposed on the underlying lymphedema is evident, as in chronic congestive failure.

## Compression physiology

When considering the use of compression therapy in the management of wound-related lymphedema, basic principles should be understood. The law of Laplace is a mathematical formula that defines the relationship between pressure  $P$ , tension  $T$ , radius  $R$ , and bandage layers  $N$ . While accepting the fact that the leg is not a fluid and is not truly cylindrical and that the uniform tension and the number of layers as a simple multiple is questionable, a “modified law of Laplace” is useful as a clinical guideline (Box 5) [32]. In particular, this principle warns of the higher, “relative” pressures that can be transmitted to small limbs, the ankle and prominent bone ridges. As long as bandage tension is constant from the toes to the knee, the highest pressures will be seen at the ankle because of the smaller radius. The opposite application is useful when increased external pressure is

### Box 5. Modified law of Laplace

$$P \text{ relative to } \frac{N T}{R}$$

P = pressure

N = number of layers

T = tension applied

R = radius of limb



desired when compressing a specific area. In this case, creating a sharper curve with molded padding decreases the radius and increases the direct pressure on the wound [33].

The concept of working pressure versus resting pressure is an important consideration. Working pressure is the pressure exerted on the leg by calf muscle contraction against the bandage material. This intermittent pulse pressure is the force that propels venous blood and lymphatic flow centrally; in other words, working pressure is directed outward from within the leg. Resting pressure is the pressure exerted from the tension of the bandage as it rests against the calf muscle. Resting pressure is directed inward from the bandage onto the surface of the leg. When the patient moves, working pressure develops. Resting pressure occurs with relaxation. The amount of working pressure is influenced by the degree of elasticity (resistance) in the compression bandage (Fig. 4).

The impact of external limb compression on vascular dynamics is a concern. A common yet erroneous belief is that all forms of compression impair arterial perfusion; however, when properly applied, external compression has been shown to increase cutaneous vasodilatation and oxygen tension. Gaylarde et al [34] demonstrated that after prolonged standing, even normal volunteers lowered their transcutaneous oxygen tension to very low levels, after which an orthopedic splint was applied and inflated to different compression pressures. It was noted that as compression was applied, the transcutaneous oxygen tension increased until a maximum was achieved at 40 mmHg external compression. Mayrovitz and Larson's [35] report of increased leg pulsatile flow affected by compression bandaging may also have special significance.

### Compression bandage guidelines

A variety of compression bandages are used in the treatment of wound-related lymphedema (Box 6). Bandage types are categorized as short-stretch (<50% extensibility), medium-stretch (70%–140% extensibility), and long-stretch (> 140% extensibility). The extensibility of a bandage can be simply determined by stretching it out along a ruler [36]. Multiple combinations of bandages are available, and their application is dictated by patient presentation. The ideal bandaging system should provide high working

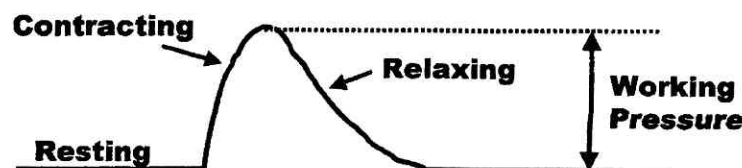


Fig. 4. Resting pressure versus working pressure—stylized representation. Working pressure increases with contraction, then returns to resting pressure upon relaxation.

**Box 6. Commonly used compression bandages***Short-stretch*

Unna boot (zinc-oxide plaster)  
Crepe  
Comprilan (Beiersdorf-Jobst)

*Medium-stretch*

Coban (3M)  
Co-plus (Smith & Nephew)

*Long-stretch*

Ace<sup>®</sup> bandage  
Surepress (ConvaTec)

*Four-layer bandage*

Profore (Smith & Nephew)

pressures and relatively low resting pressures to safely remove peri-wound lymphedema without compromising microcirculation.

Short-stretch bandages have many advantages for standard wound care [37]. They reduce deep venous reflux more effectively than long-stretch bandages and produce high-pressure amplitudes when the patient is walking. A pressure decrease of approximately 20 mmHg occurs when the patient is lying down. Their main disadvantage to short-stretch bandages is that pressure loss occurs after a short period; this can be explained by loosening of the bandages with edema reduction [38–40].

The advantage of long-stretch bandages is that they maintain pressure for longer periods. However, a higher interface pressure (60 mmHg) is necessary to effectively reduce venous reflux. The pressure amplitude during exercise is smaller, and there is only a small pressure decrease when the patient lies down [38–40].

The four-layer bandage system (containing short-stretch, medium-stretch, and long-stretch material) reduces venous reflux, though less effectively than short-stretch bandages [40]. It produces high-pressure amplitudes while the patient is walking and can maintain its pressure for 7 days [41]. However, Hafner [37] reported only a small pressure decrease when the patient is supine.

A compromise bandage system consists of a core of short-stretch bandages and an outer layer of cohesive medium-stretch bandage. This type of bandage decreases pressure (resting pressure) when the patient is supine, induces high-pressure amplitudes during exercise (working pressure), and sustains compression after an initial decrease of approximately 10 mmHg in the first 6 hours. Consequently, it can be applied with a slightly elevated initial pressure to reach the therapeutic range after a few hours [37]. The

standard Unna boot with a medium-stretch outer layer fits this description, as does a bandage system consisting of an inner layer of cotton/gauze and an outer layer of self adhesive medium-stretch bandage.

Using various combinations of compression techniques, properly applied bandages can be effective even in compromised limbs. Obviously, there are critical pressures of compression that will either enhance or impede arterial, venous, and lymphatic flow. Studies are now in progress to define the hemodynamics that appear to clinically permit such application, such as the effects of varying compression on ankle-brachial indices, transcutaneous oxygen measurement, and laser Doppler flow. However, in the absence of parameters that have yet to be defined, basic clinical tools can be used to safely monitor patients in need of compression bandaging (Box 7). In most patients with normal sensation and arterial perfusion (palpable distal pulses), the commonly used methods are safe. In all patients, regardless of the underlying conditions, patient feedback regarding pain, parasthesias (when possible), capillary return, and observable skin change are mandatory whenever such bandages are used. Reassessment—sometimes daily—may be required for limbs that are obviously at risk. With vigilant awareness, most patients in need of compression can be treated. Every caregiver entrusted to limb wrapping must be properly trained and monitored, because compression applied improperly, even with normal circulation, can result in serious limb damage [42].

The severity of swelling, diabetes, arterial and venous insufficiency, and local skin condition must be considered. Wound characteristics and patient mobility influence the choice. Awareness of cardiac function and the danger of congestive heart failure with increased fluid mobilization is critical in susceptible patients. In the authors' clinic, we have compressed the limbs of patients with a history of congestive heart failure to improve wound healing or to prevent skin breakdown. The following precautions should be observed in such cases:

1. There is no clinical evidence of active failure.
2. Knowledgeable caregivers are available to monitor the patient for any change in the patient status.
3. Compression wraps are placed at daily intervals on one leg at a time.

#### **Box 7. Compression safety guidelines**

- Pain
- Parasthesia
- Capillary return
- Visible skin trauma
- Reassessment

When the swelling has been reduced over time in this fashion, the patient can then be fit for knee-high compression garments.

Assessment and intervention guidelines are shown in Table 1. An important technical point in bandage technique is the use of a primary layer of cotton/gauze padding separating the short-stretch bandage from the skin. In addition to its absorptive capacity in the wound dressing, this layer affords a measure of protection from the applied pressure. It must also be stressed that when bandaging a limb for compression, be it an arm or a leg, the bandage must begin at the distal extremity, cover the entire limb (including the heel), and extend to the knee/elbow or hip/shoulder, depending on the location of the lesion. Likewise, when applying an Unna boot, placing a strip of ¼-inch synthetic foam from midfoot to the knee under the outer wrap provides excellent protection for fragile skin.

As is true for all methods of wound care, frequent reassessment is vital for adapting the treatment to the improvement or lack of improvement in the wound. Likewise, compression bandages must be altered as the conditions dictate. For instance, in a moderate to severely edematous limb, the initial bandage combination could likely contain a long-stretch bandage. Then, as the limb became edema free, short-stretch bandages would be safer and more appropriate. In general, in patients with peripheral artery disease or insensate neuropathy, the use of short-stretch bandages or Unna boots with inelastic zinc plaster bandages should be encouraged [43].

Traditional teaching has stated that compression bandages are contraindicated in the presence of acute cellulites; however, a review of the literature finds no substantive support for such a claim. Likewise, the alternative (ie, the positive effect of compression in the treatment of acute cellulites) has not as yet been supported by evidence-based studies. However, the authors have made compelling clinical observations that with strict guidelines, compression is almost always indicated in the treatment of the

Table 1  
Assessment and intervention compression guide

Assessment	Unna boot	Cotton Short-stretch	Cotton, Short-stretch, long-stretch coban	Cotton Coban
Venous hypertension	Yes	No	Yes	No
Normal arterial perfusion	Yes	Yes	Yes	Yes
Decreased arterial perfusion	Yes	Yes	No	Caution
Neuropathy	Yes	Yes	No	Caution
Ambulatory	Yes	Yes	Yes	Yes
Non ambulatory	No	Yes	No	Yes
Slight drainage	Yes	Yes	Yes	Yes
Heavy drainage	Yes	Yes	No	Yes
Lymphedema trace	Yes	Yes	No	Yes
Lymphedema 1 +–3 +	Yes	Yes	Yes	Yes
Lymphedema fibrosis	Yes	No	Yes	No
Cellulitis	Yes	Yes	Yes	Yes

edema/lymphedema associated with acute and chronic cellulitis. Two factors must be addressed: immediate systemic antibiotic cover and appropriate pain control. Diuretics usually have little effect in controlling swelling. Furthermore, as in the case of wound healing, the negative effect of persistent fluid stasis in the infected limb inhibits timely resolution. Hopefully, this clinical observation will soon be verified by evidence-based studies.

## Summary

As the science of wound healing has evolved over the past two decades, so has awareness of the “hidden epidemic” of lymphedema. Substantial information has been accumulated regarding the pathophysiology and therapy of lymphedema. Until recently, the relationship between wound healing and the negative effects of associated peri-wound lymphedema has received little attention. Identifying wound-related lymph stasis and safe mobilization of the fluid are fundamentals that must be addressed for proper therapy. Experience gained from the successful treatment of primary and secondary lymphedema has proven very useful in the applications to wound-related lymphedema. The mobilization of lymph fluid from the peri-wound area with the use of reasoned compression is essential for proper therapy of the open wound, as are appropriate bandage selection and safeguards for bandage application.

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