Long-term results of compression treatment for lymphedema

Christos J. Pappas, MD, and Thomas F. O'Donnell, Jr., MD, FACS, Boston, Mass.

Although numerous operations have been devised for lymphedema, most surgeons manage this vexing condition by nonsurgical means. Previous studies by us showed that high-pressure (90 to 100 torr) sequential external pneumatic compression (SEP) reduced both limb girth and volume in a lymphedematous extremity. To assess the long-term effects of a program entailing (1) SEP, (2) elastic compression stockings to maintain the post-SEP girth, and (3) daily skin care, we reviewed the long-term courses of 49 patients managed by one surgeon. Limb girths measured at nine levels on the limb were obtained serially in follow-up (mean 25 months) by an independent observer to provide an objective response to therapy. The relative reduction in lymphedematous tissue was determined by the difference between the pretreatment, postacute treatment, and long-term treatment girths at nine points in the limb. In long-term follow-up, 26 of the patients maintained a full response (reduction at > 3 levels), whereas 10 maintained a partial response (reduction at ≤ 3 levels). At late follow-up, calf and ankle girths were reduced by an absolute value of 5.37 ± 1.01 and 4.63 ± 0.88 cm in the full-response group and 5.43 ± 1.58 and 3.98 ± 1.18 cm in the partial-response group over pretreatment measurements. The degree of subcutaneous fibrosis in relationship to the duration of the edema appeared to influence results greatly. The treatment of lymphedema with SEP and compression stockings is associated with long-term maintenance of reduced limb girth in 90% of patients. (J VASC SURG 1992;16:555-64.)

Lymphedema can be a frustrating disease for both the patient and treating physician. Delays in establishing the correct diagnosis are frequent and response to therapy can be disappointing. Treatment is directed toward reducing the limb girth and weight, as well as preventing infection. Although a variety of surgical procedures have been developed that either excise the bulky lymphedematous tissue or anastomose lymphatic vessel to lymphatic vessel or lymphatic vessel to vein, the results are variable and usually not documented objectively. Kinmonth et al. showed that a high proportion of patients regressed to their preoperative levels within 3 to 4 years after surgery. Surgery is usually reserved for the most extensive cases of lymphedema, so the majority of patients are treated by nonsurgical means. Excess lymphedematous tissue that produces a cosmetic deformity and limb heaviness, as well as infection, are the two key clinical features of lymphedema requiring therapy.

Simple elevation, custom-fitted elastic compression stockings, and external pneumatic compression are the usual approaches that are employed to reduce limb size. Elastic stockings generally maintain limb girth but do not uniformly reduce the lymphedematous tissue. Previously we showed that sequential high-pressure intermittent pneumatic compression (SIPC) by the Lymphapress device (Camp International, Inc., Jackson, Mich.) was superior to single-cell low-pressure external pneumatic compression in the acute reduction of lymphedema. No late follow-up data have been available, however, that present the long-term benefits of such therapy. To assess the impact of a program of SIPC, custom-fitted elastic stockings, and skin care on the long-term reduction of lymphedematous tissue and prevention of infection, 49 patients with lymphedema treated by us were studied.

METHODS

Protocol (Fig. 1). Patients with primary and secondary lymphedema of either the upper or lower extremity were hospitalized for a 2- to 3-day period to determine their acute response to SIPC (Lym-
phapress). We have found that the maximum response to therapy can be achieved in a controlled hospital environment rather than at home or serial outpatient treatments. The patient is restricted to complete bed rest, and defined periods of compression are achieved. Third-party insurers have concurred with this approach. Before initiation of treatment, a detailed history was obtained from the patient with an emphasis on duration and extent of lymphedema, prior surgery or radiation, and frequency of infection. In addition, previous treatment with elastic compression stockings or unicompartamental intermittent pneumatic pressure devices and the response to therapy were detailed. Abdominal and pelvic computed tomographic scanning was performed in the majority of patients, whereas lymphoscintigraphy and lymphangiography were obtained in selected patients by our previous criteria. After treatment with the SIPC device, the patients were measured for custom two-way stretch elastic stockings at their new postreduction girth by an experienced fitter. The majority of the patients had 40 mm Hg compression stockings. Patients were encouraged to wear their stockings daily by applying them in the morning before rising from bed. Antifungal powder or spray was applied daily to the interdigital spaces. Water-soluble skin lotion was recommended for twice-daily use on the affected limb.

Patients were seen at 4- to 6-month intervals in follow-up, at which time clinical evaluation was repeated and measurements of limb girth at specified points were obtained, with the templates used for girth sizing of elastic stockings. Special attention was paid to a history of infection or the development of skin lesions. Patients were recommended for repeat SIPC treatment depending on the reaccumulation of edema fluid in the interim period. Patients in whom reaccumulation of edema fluid was rapid were prescribed home use of SIPC, whereas patients whose reaccumulation of edema fluid was slow had SIPC treatment at 4- to 6-month intervals. During the interim period, the limb girth was maintained with elastic stockings. Therefore three patterns of response and need for SIPC treatment were observed: (1) acute SIPC treatment with elastic stockings only, (2) acute SIPC treatment with elastic stockings and repeat SIPC treatment at 4- to 6-month intervals on an outpatient basis, and (3) acute SIPC treatment with elastic stockings—daily SIPC treatment.

Lympaphress therapy. The multicompartamental sleeve SIPC (Lymphapress) was applied to the extremity. This sleeve contained 9 to 12 overlapping cells that can be fitted to the limb according to size. In treatment of the lower extremity an additional one-cell boot was applied to the foot area. Inflation of the cells was initiated from the most distal cell and progressed proximally. The cells were powered separately by a compressor through a distributor so that each successive inflation created a sequential milking mechanism. When the entire sleeve was filled with air,
the cells automatically and simultaneously deflated. The compression period lasted 20 seconds for the first, most distal cell and two seconds for the more proximal cell (thigh or upper arm). This short cycle permitted the application of high pressure to the limb (80 to 110 mm Hg) without producing pain. Pressures above systemic systolic measures were avoided so that maximum pressures were usually 80 to 90 mm Hg. Moreover the pressure could be adjusted easily by a nurse or the patient so that peak pressures could be altered to accommodate individual tolerance. After 6 to 8 hours of compression, the sleeve was removed and the limb was allowed to air dry. All patients were hospitalized for 48 to 72 hours in a controlled environment for the acute phase of treatment. The period during which compression was applied was uniform for the acute-treatment session (4 to 6 hours) but was more variable in those patients who used the device at home. Patients would schedule the home treatment programs to accommodate their life-style. In general, patients used the SIPC unit for a minimum of 4 hours a day, usually in the evening and at a pressure of 80 to 90 mm Hg.

Limb girth measurements. The percentage of reduction of lymphedematous tissue at each point in the limb was determined with a template, commonly employed for fitting elastic stockings. Circumferential measurements were obtained at nine sites in the leg and six sites in the arm. Measurements were expressed in absolute and relative terms. Pretreatment circumference – posttreatment circumference/pretreatment circumference × 100 provided the relative decrease in lymphedematous tissue, whereas pretreatment girth (in centimeters) – posttreatment girth yielded the absolute reduction. The latter expression was used to calculate any significant change between the acute-phase results and follow-up values. Paired t testing was used for the latter, whereas analysis of variance was employed to compare the differences between separate anatomic sites on the limb.

RESULTS

Patient characteristics. The 49 patients were comprised of 22 patients with primary lymphedema and 27 patients with secondary lymphedema. All patients with primary lymphedema had involvement of the lower extremity with a relatively comparable distribution between the right and left limbs (3:2 left/right ratio). Six patients with secondary lymphedema had edema of the upper extremity, all after breast surgery with node dissection. Women predominated in both groups (primary, 16 female to six male, versus secondary, 19 female to eight male patients). Patients with primary lymphedema (mean age 34 ± 6 years) were younger than those with secondary lymphedema (mean age 56 ± 8 years). Although four patients had recurrent infection as the cause of secondary lymphedema, the majority of the cases (n = 20) followed surgery for either cancer (n = 17) or trauma (n = 3). Five patients had an infection of the lymphedematous extremity before entry into the study. Forty of 49 patients were wearing elastic compression stockings or sleeves before entry into our study, whereas five patients were using a unicompartamental compression device before the initiation of the study. Two patients had undergone previous surgery with limb-reduction procedures at other institutions but had reaccumulation of the lymphedema.

Pretreatment girths. Table I compares the increase in limb girth over the contralateral normal limb girth for the three therapeutic response groups. Obviously, patients with bilateral edema were excluded from these groups. The greatest increases occurred at calf, distal calf, and ankle levels. No difference among the three groups was observed at any level. The degree of lymphedema, as assessed by comparison to the normal contralateral limb, was at least 2 inches greater in girth.

Response to SIPC therapy. Serial follow-up after initiation of a patient into the study averaged 25 ± 4 months, during which all patients wore their elastic stockings and followed the recommendation for skin care. Approximately one half of the patients used the SIPC device at home for daily or alternateday therapy. Four patients had infections in the follow-up period, three of whom required hospitalization for administration of intravenous antibiotics. One of these patients had an additional hospitalization for a second episode of infection. Two patients had evidence of tinea pedis as the possible site of entry, whereas two had no obvious skin lesions as an initiating site.

The patients were divided into two groups depending on their initial response to SIPC therapy during the acute phase (Fig. 2): those who had at least a 2 cm acute reduction in limb girth and those with no response. The group of patients who responded to the initial SIPC treatment (acute phase) were subdivided further into two groups: (1) those whose limb-girth reduction was maintained at least within 1 cm of the acute-phase girth measurement at more than three levels (full-response group) and (2) those whose measurements were only partially main-
Table I. Pretreatment lymphedematous tissue: Comparison of limb girths in three therapeutic
groups (abnormal limb-normal limb)*

<table>
<thead>
<tr>
<th>Level</th>
<th>Full responders (cm) (n = 26)</th>
<th>Partial responders (cm) (n = 10)</th>
<th>No response (cm) (n = 7)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gluteal fold</td>
<td>4.28 ± 1.79</td>
<td>4.77 ± 2.00</td>
<td>3.70 ± 2.45</td>
</tr>
<tr>
<td>Upper thigh</td>
<td>4.20 ± 1.76</td>
<td>4.30 ± 1.84</td>
<td>4.74 ± 2.59</td>
</tr>
<tr>
<td>Lower thigh</td>
<td>5.10 ± 1.75</td>
<td>5.67 ± 1.80</td>
<td>4.00 ± 2.82</td>
</tr>
<tr>
<td>Knee</td>
<td>5.47 ± 1.43</td>
<td>3.25 ± 1.16</td>
<td>2.64 ± 1.75</td>
</tr>
<tr>
<td>Calf</td>
<td>6.76 ± 1.54</td>
<td>4.95 ± 1.58</td>
<td>4.54 ± 0.88</td>
</tr>
<tr>
<td>Distal calf</td>
<td>7.24 ± 1.98</td>
<td>5.83 ± 3.30</td>
<td>4.45 ± 3.19</td>
</tr>
<tr>
<td>Ankle</td>
<td>6.28 ± 1.40</td>
<td>4.72 ± 2.14</td>
<td>4.48 ± 2.80</td>
</tr>
<tr>
<td>Instep</td>
<td>3.64 ± 0.83</td>
<td>3.77 ± 1.01</td>
<td>3.50 ± 1.77</td>
</tr>
<tr>
<td>Metatarsophalangeal joint</td>
<td>1.55 ± 0.56</td>
<td>1.53 ± 0.53</td>
<td>1.45 ± 0.41</td>
</tr>
</tbody>
</table>

Data are means ± SEM. *Excludes six limbs with upper-extremity edema.

The data are expressed as pretreatment girth at a specific level minus posttreatment girth. With initial treatment the greatest absolute reduction was achieved at the distal calf and ankle levels for both groups. The least change was noted at the metatarsophalangeal joint line (Table II). Reduction of the upper segment of the leg (gluteal fold was greater than lower thigh) was comparable for both groups at approximately 2.5 cm. The partial-response group, however, had less of an acute response than the full responders at the gluteal fold and upper thigh levels ($p < 0.05$); limb-girth reduction (acute) was also less for the partial response group at the knee level ($p < 0.05$). Although at late follow-up the full-response group had no increase in limb girth, no additional reduction in limb girth was noted with long-term therapy. By contrast, the partial-response group had some return of fluid (increased girth) at the upper thigh, lower thigh, calf, and instep levels. The degree of reduction at other levels, however, was maintained in this group.

Clinical characteristics were analyzed to deter-
Compression treatment for lymphedema

Table II. Absolute reduction of lymphedematous tissue: Comparison of changes in limb girths with acute compression and long-term treatment*

<table>
<thead>
<tr>
<th>Level</th>
<th>Full responders (cm) (n = 26)</th>
<th>Partial responders (cm) (n = 10)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Acute</td>
<td>Late</td>
</tr>
<tr>
<td>Gluteal fold</td>
<td>2.86 ± 0.52</td>
<td>2.39 ± 1.02</td>
</tr>
<tr>
<td>Upper thigh</td>
<td>2.42 ± 0.63</td>
<td>2.55 ± 1.01</td>
</tr>
<tr>
<td>Lower thigh</td>
<td>2.62 ± 0.59</td>
<td>3.11 ± 0.79</td>
</tr>
<tr>
<td>Knee</td>
<td>2.95 ± 0.76</td>
<td>2.86 ± 0.76</td>
</tr>
<tr>
<td>Calf</td>
<td>3.58 ± 0.78</td>
<td>4.38 ± 0.7</td>
</tr>
<tr>
<td>Distal calf</td>
<td>6.22 ± 1.30</td>
<td>5.37 ± 1.01</td>
</tr>
<tr>
<td>Ankle</td>
<td>4.27 ± 0.61</td>
<td>4.63 ± 0.88</td>
</tr>
<tr>
<td>Instep</td>
<td>1.88 ± 0.36</td>
<td>2.32 ± 0.70</td>
</tr>
<tr>
<td>Metatarsophalangeal joint</td>
<td>1.30 ± 0.26</td>
<td>1.50 ± 0.26</td>
</tr>
</tbody>
</table>

Data are means ± SEM.
*Excludes six limbs with upper-extremity edema.

mine whether any factors might predict which patients would achieve the best long-term results. Among the three subgroups (full, partial, and no response groups), the incidence of lymphedema type (primary or secondary), gender, extremity involved, or amount of lymphedematous tissue pretreatment did not predict the subsequent response to treatment. A comparable proportion of patients (50% to 60%) in both the full- and partial-response groups used the SIPC device at home. Indeed, all but one of the poor-response group used the SIPC device at home. Thus the use of the SIPC device alone did not appear to account for which limbs responded the best, but rather the condition of the subcutaneous tissue (less fibrosis and more compliant) and possibly the degree of collateral lymphatic flow governed the response to compression therapy. There was no difference in the length of follow-up among the three groups. The duration of the lymphedema before the treatment program started did appear to correlate with the response. Although 50% of the full-response group had had lymphedema for more than 10 years (mean 13.6 ± 2.3 years), 80% of both the partial (mean 17.5 ± 3.8 years) and nonresponding groups (mean 18.2 ± 4.1 years) had lymphedema for more than 10 years, which suggests a potential adverse role of lymphedema fluid on the subcutaneous tissue. Lymphedema results from insufficient transport of both water and protein from the skin and subcutaneous tissue as a result of either insufficient development of lymphatic vessels or their destruction. The resultant accumulation of fluid produces symptoms of limb heaviness and the characteristic cosmetic deformity of an enlarged limb. Because of altered host defense mechanisms in the limb, approximately 20% of these patients also have recurrent infections, of which some are life-threatening. A number of surgical procedures have been developed to decrease limb size, and these are classified as either excisional, whereby segments of the skin and subcutaneous tissue are removed from the limb, or physiologic, whereby, lymphatic-to-lymphatic or lymphatic-to-venous drainage is promoted. A review of the results of these procedures, however, shows a surprising lack of objective data to judge the efficacy of the procedure. Kinmonth et al. retrospectively assessed their patients who had undergone a series of buried dermal flap (Thompson12) procedures. They observed that fewer than one third of their patients had what could be judged qualitatively as "good results." The best results appeared to be achieved in those patients with the greatest amount of lymphedematous tissue. In addition, a significant proportion of patients had reaccumulation of the lymphedema by 3 to 4 years after surgery. With the long skin incisions and extensive dissection of subcutaneous tissue required by this procedure, nearly one quarter of the patients had major skin necrosis that lengthened both hospital stay and time lost from work. Given these results, most physicians treat lymphedema by nonsurgical means. The chief goals of this therapy are (1) to reduce lymphedematous tissue and (2) to prevent skin infections. In addition to elevation of the limb at night and
custom-fitted elastic compression stockings, external pneumatic compression devices have been employed in the treatment of lymphedema. There are several variables influencing the degree of reduction achieved by external pneumatic compression treatment and are related to not only the device itself but also the condition of the limb. The absolute peak pressure, compression cycle, and sequence and distribution of compression constitute the mechanical factors related to reduction of lymphedema. By contrast, one of us has shown that the degree of subcutaneous fibrosis or tissue compliance greatly influences the extent to which a limb is reduced in size. Unicompartmental pressure devices compress the entire segment under the sleeve at the same time, so pressure is distributed both centripetally and centrifugally—somewhat similar to squeezing a toothpaste tube in the middle. Fluid is compressed toward both the foot or hand and the trunk. Although some unicellular devices do use a short pressure cycle, the peak compression developed by the unit is usually low (60 mm Hg).

We observed previously that 30% of patients with lymphedema failed to achieve a significant decrease in limb girth under treatment with a unicell device and the proximal portion of the limb (thigh or arm) responded poorly to the low-pressure unicompartmental device. Zelikovski et al. developed a multicompartmental high-pressure pneumatic compression device that uses a short pressure cycle and distal-to-proximal "milking" action. By applying multiple cells at high pressures (100 to 110 mm Hg), large volumes of fluid can be removed from the limbs. In one of the few objective evaluations of the physiologic basis for external pneumatic compression therapy, Partsch et al. examined limb volume measurements, isotope lymphography, and the disappearance of subcutaneously injected I-131-I albumin and compared a unicellular device to an SIPC unit. They demonstrated that the average limb volume decreased approximately 5% per session and, like our experience, noted that the most susceptible area to reduction was the lower leg rather than the thigh. As observed in our acute series, there was an increase in urinary output with SIPC therapy. When the unicellular device (Jobst) was compared with the SIPC device (Lymphapress), the latter was associated with a more rapid transit and greater intensity of labeled tracer at the inguinal lymph node than the unicellular device, which indicated improved lymph transport. The studies of Partsch et al. also suggested that the SIPC device mobilized free water to a greater degree than it did protein within the extremity, so the efficacy of treatment was related to the degree of water loss from the limb.

In his original presentation of the SIPC device, Zelikovsky et al. reported 20 patients with both primary and secondary lymphedema. Although only one girth measurement on the limb (at the midcalf) was obtained, the mean acute reduction of edema was 14.5 cm with a 2.0 to 70.0 cm range. No report of the long-term results of these patients was provided. In a subsequent report they assessed the acute response of 25 patients with postmastectomy edema to Lymphapress treatment. They reported a 36% to 70% reduction in lymphedema in 80% of the patients. In addition, they noted improved clearance of radioactive albumin from the tissue, similar to the results of Partsch et al. Girth measurements at multiple levels and long-term results were not provided.

We carried out a controlled in-hospital trial in which the SIPC device was applied to the limbs of 25 patients to assess the acute response to compression and showed a significant reduction in limb girths at all levels, as well as in limb volume for the majority of limbs. Serial measurements of muscle enzymes demonstrated no evidence of tissue damage with this high-pressure device. In this study the absolute reduction in lymphedematous tissue in both our full and partial responders during the acute phase was similar in degree to results achieved in our earlier series. Both our earlier and present studies showed that the maximum sites of girth reduction were achieved at the ankle and calf levels. Our present group of patients, however, had a greater reduction acutely at the thigh level than had patients in our previous study. Like our previous study, the majority of patients appeared to achieve some reduction at all levels.

The long-term results in maintenance of reduced limb girths were equally gratifying. Approximately 60% of the patients maintained the reduced limb girth at all nine levels, whereas an additional 20% maintained reduction at the distal calf through toe levels. Nineteen percent of the patients had minimal reduction in limb girth.

Our study is not a randomized study comparing SIPC to no-compression therapy (elevation of limb alone) or to elastic compression alone. Although it can be argued that some reduction in limb girth can occur with prolonged rest, our clinical experience suggests that this is much less than with SIPC. The classification of our patients was by their long-term response to therapy and therefore is a sequential follow-up study. The absolute long-term reduction in...
limb girth was used to classify patients so that other variables such as years with lymphedema or frequency of SIPC treatments could be analyzed as predictors of response to SIPC treatment.

McLeod et al. compared the efficacy of three multicellular external pneumatic compression devices in a small number of children with congenital primary lymphedema. In one of the few cross-comparative objective trials with long-term results, they showed that the decrease in total limb volume was superior with the SIPC (Lymphapress) device versus the three-celled Hemoflow II unit (Camp International, Inc., Jackson, Mich.). In the other device compared (the three-celled Wright-Linear pump; Wright-Linear Pump, Inc., Imperial, Pa.) the distal cell must be adjusted so that the more proximal cells rise to a lower peak pressure. They pointed out that malfunction of one of the three cells could create a tourniquet effect. Long-term results in their Lymphapress group were similar to ours, with a continued decrease in limb size over time.

We have shown previously that the degree of subcutaneous fibrosis is related directly to the degree of reduction achieved by a compression device. More than 80% of patients in both the poor- and partial-response groups had lymphedema for greater than 10 years before initiation of our protocol. Qualitative examination of these limbs showed that the degree of subcutaneous fibrosis was greater than in the full-response group. In our previous study, xeroradiographic assessment of subcutaneous tissue fibrosis was employed. We are now assessing the role of B-mode ultrasonography in characterizing the condition of the subcutaneous tissue, and preliminary results show that this morphologic assessment correlates well with our clinical impression of the degree of subcutaneous fibrosis. The deleterious effect of subcutaneous fibrosis on the long-term results argues for earlier treatment of lymphedema. Casley-Smith et al. have shown that the proteinaceous lymphedema fluid induced an inflammatory reaction in the subcutaneous tissue. Efforts to decrease lymphedema fluid within the extremity should therefore reduce the inflammatory response that leads to subcutaneous fibrosis. Similar conclusions were reached by Partsch et al., who also recommended early compression treatment.

The home use of SIPC is modified to the patient’s schedule. Patients use the compression device for 4 to 6 hours, usually in the evening while watching television or reading, whereas a small percentage can use the device during sleeping. Peak systolic compression pressure is adjusted to patient tolerance. We believe that this pressure should be above the patient’s systemic diastolic pressure but should not exceed systolic pressure. Generally pressures of 80 to 90 mm Hg are used. Bastien et al. described a 14-year-old girl with congenital lymphedema in whom acute treatment had to be abandoned because of leg pain. They theorized that the compression pressure was above systolic pressure and produced ischemic pain. In our experience, if a patient has pain during SIPC treatments it is usually over the foot area and a result of the difficulty of conforming the compression cell to the contour of the foot. A lightweight cotton full-length sock should be applied to the limb to absorb moisture. Diuretics are not employed routinely in our patients because with chronic use these agents become ineffective. Strict attention should be paid to the condition of the elastic compression stocking. A two-way stretch stocking is employed because of excellent patient acceptance and durability. Once the stocking loses its elasticity it should be renewed. In general, two pairs of stockings should last 4 to 6 months.

During this study, four patients had infections; three required hospitalization. All were in the poor-response group. No relationship to therapy was noted. Reduction in the incidence of infections cannot be linked directly to the use of the SIPC device and is usually related to skin care. Indeed, prevention of folliculitis is imperative in patients using SIPC or elastic compression stockings. Daily washing of the affected limb with mild soap, as well as inspection for early signs of infection, is important. Assiduous protection against fungal infections with topical antifungal cream or spray and the use of oral antifungal agents when tinea pedis is discovered prevent a portal of entry for pyogenic bacteria. The twice-daily use of water-soluble skin lotion softens the skin and removes the eczematous scaling, which also can be a site of entry for bacteria. With a treatment program of compression and skin care, patients with lymphedema are able to enjoy a near-normal life-style.

Michael A. Belkin, MD, provided statistical analysis for this work.

REFERENCES

DISCUSSION

Dr. Robert L. Kistner (Honolulu, Hawaii). I would like to point out that you do not come recently to this field. You reach well back into your training experience when you worked in London with Professor Kinmouth and Professor Browse, and there they count their experience in this area. However, I have the suspicion that some of these cases were resistant and represent a rather more advanced degree of lymphedema. The 49 patients in this study are evenly divided between primary and secondary lymphedema. Most of them were in the lower extremity. Almost one third had malignant disease.

This distribution indicates that the sequential pneumatic compression is widely applicable across the breadth of causes of lymphedema. It brings up my first question, which is about diagnosis. Although most of us can look at a leg and determine whether lymphedema is present, we really cannot tell the cause of the lymphedema.

Causes may be related to hyperplasia of lymphatics, obstruction at lymph nodes, or severe reflux in the lymphatics. These make some difference. Also, there is a possibility of severe chronic venous disease.

Do you rely on lymphoscintigraphy, which lacks specificity in its diagnostic ability but is safe, or do you still use lymphangiography to identify the cause of the patient's problem and extent of the disease?

Relative to chronic venous disease, I do have something to add and that is in several cases in which we have looked at patients with lymphedema combined with refluxive disease, we repaired the chronic venous insufficiencies as far as the reflux is concerned and found essentially no change in the clinical course. I would be interested to know what your experience has been in this area.

Another question that I have relates to the selection of patients for this series. We know that the majority of patients with lymphedema can ultimately be treated with compression therapy in the form of elastic support. If your cases are unselected simple cases, perhaps your experience of a greater than 20% reduction in 80% of patients is easy to understand. However, I have the suspicion that some of these cases were resistant and represent a rather more advanced degree of lymphedema.

Your results state that patients did as well with two to three treatments per year as they did with daily treatments at home. To me this is also a surprise. You said it surprised you, and it makes one question what the compression therapy is really doing. It sounds like we are just ringing water from the extremity and then holding it down with the elastic stockings.
Is there some reason to have the patient purchase a pump for use at home or should we just treat the patients intermittently in a clinic or hospital setting? The pumps are not cheap, and actually in your article you refer multiple times to the use of the Lymphapress. Since there are multiple pumps on the market, is there something specific about the Lymphapress pump that is unique and would make us look more toward the Lymphapress pump than some of the other choices? I believe the Lymphapress pump is more expensive than most of the others.

In the nonsurgical treatment of lymphedema, we have compression with elastic stockings, pneumatic compression, and also massage therapy. Fuldi in Germany describes complex therapeutic massage with rather amazing results, and they are consistent with the percentage results that you described.

Do you use massage therapy and specifically complex massage therapy or do you have an opinion about whether we should become knowledgeable about this approach?

In your long-standing cases you apparently found the least help from compression. The proximal edema was more resistant, as was the edema in the distal foot. What do you do with these problems? Is there a place for surgery?

Relative to the various choices for surgery, do you still use these? Specifically, there are rare patients who have reflux disease who would seem most unlikely to respond to compression therapy, and they are capable of being helped by surgery.

I think we would all be glad to be rid of the debulking procedures. Do you still employ surgical procedures in the very resistant cases?

**Dr. Thomas F. O'Donnell.** Regarding the diagnosis of lymphedema, I believe that the diagnosis can be made in the majority of patients by a clinical evaluation on the basis of the characteristic historic and physical findings of lymphedema. If there is any question whether the edema is lymphatic, or to pick up that rare patient who may have reflux, we use lymphoscintigraphy. This radionuclide technique is minimally invasive, provides pretty good objective data, and allows one to classify the patients with primary lymphedema into obliterator, the commonest type, and reflux, which is unusual. A contrast computed tomographic (CT) scan of the abdomen and pelvis, including the inguinal areas, also can be helpful in the diagnosis, because in primary lymphedema it can show the absence or a diminished number of lymph nodes, consistent with the hypoplastic, or obliterator, form. A CT scan with contrast material can demonstrate large lymphatic vessels in those patients with reflux and select these rare patients who might benefit by ligation of the lymphatic trunks. I also believe that CT scan evaluation is essential in patients with secondary lymphedema after an oncologic procedure. Here it is used to rule out recurrence of tumor.

As far as the combination of chronic venous insufficiency in lymphedema, there have been several studies in which lymphangiography has been performed in patients with chronic venous insufficiency: the first, by Negus and Associates at St. Thomas' Hospital, London, and the most recent one by Leonel Villavincenzio at Walter Reed. Both suggested obliteration of the lymphatic trunks by chronic infection that is so common in patients with chronic venous insufficiency.

Dr. Kisner rightfully points out that one has to be very wary of the response to limb-reduction measures in patients with a combination of chronic venous insufficiency and lymphedema. The girth of the limb may not decrease after a venous procedure. Indeed, in my personal series of deep venous reconstructions, there have been a few patients in whom the ulcer healed but there was a worsening of the edema. One such patient is actually included in this particular study. The worsening of the edema may be related to inadvertent damage of the lymphatic trunks at venous reconstruction surgery. Conversely, patients who undergo intermittent pneumatic compression with venous obstruction were shown in a previous study by us to have poorer results than those with lymphatic disease alone. As far as patient selection, the group presented today does represent a portion of the patients I treat. I divide the approximately 175 to 200 patients with lymphedema in my practice into three groups: those who require only custom-fitted elastic support (very mild edema), those who require Lymphapress treatment, as has been presented today (those with more extensive degrees of edema), and, finally, a small percentage with massive edema unresponsive to conservative therapy (about 10%) who will require reduction surgery.

There were questions regarding the unique features of the Lymphapress. It is multicellular, like some other compression devices, but has nine to 12 cells versus the two to three cells of other devices (McLeod A, Brooks D, Hale J, et al. Physiother Can 1991;43:28-31). A recent study in Toronto compared the Lymphapress device with the Wright-Linear pump and the Hemaflow device in a small number of pediatric patients. This study showed that the degree of edema reduction in the same patients was greater with Lymphapress, which I believe is a result of the greater number of cells and its unique massaging effect. As far as massage therapy, do I practice it? I think that the multicellular Lymphapress device is providing massage therapy. The late John Kinmonth recommended massage therapy in his practice but wished that there was a mechanical device to simulate massage. With its constant distal to proximal milking action, the Lymphapress device, in effect, massages the limb.

**Dr. Eugene F. Bernstein** (La Jolla, Calif.). I would like to add support with an essentially equal experience in numbers of patients and duration of follow-up with the same device. It is my sense that the only diagnosis that matters in a new patient, which would mean that I would treat the patient with something other than the Lymphapress first, is if they have proximal venous obstructive disease that might be repaired surgically. How do you feel about that? That is the only thing I attempt to define before a trial of this treatment.
My second question is the issue of how to deal with a patient who requires long-term Lymphapress therapy. Does the insurance company get billed three times a week for life, or do you try to negotiate on behalf of the patient with a third-party payer to buy a device for the patient?

Dr. O'Donnell. First, you are right to point out that it is important to identify patients who have proximal venous obstruction. In a previous study by us with a single-cell compression device employed for the treatment of postmastectomy lymphedema, the patients with lymphatic damage and venous occlusion had the least benefit. Therefore some type of noninvasive screening for venous obstruction, particularly in patients who have undergone previous oncologic surgery, is important to incorporate into your pretreatment diagnostic studies.

As far as who pays, I met with several of the third-party payers in Massachusetts and, with them, developed some guidelines that would qualify a patient to purchase a compression device with support of the insurance. In general the criteria are a 3 to 4 cm difference at various levels of the leg or arm and a positive response to therapy, which has been demonstrated in a controlled environment (i.e., the patient has shown a satisfactory acute response to compression). In general, Lymphapress devices are restricted to those patients with massive edema or those who show rapid reaccumulation of moderate edema despite elastic compression stocking treatment.

Dr. Bernstein. What percentage of your patients end up buying a machine?

Dr. O'Donnell. Approximately 50% of the full-response group had a machine at home because they wanted it; 80% of the partial-response group and 90% of the marginal-response group had machines for home use. Thus in this series, more than 65% of the patients had their own machines at home.

Dr. John J. Bergan (La Jolla, Calif.). Our numbers are similar to those of Dr. Bernstein, and our conclusions are similar and yet different. The similarities would be that proximal lymphedema is very difficult to control, and this correlates with lymphedema tarda. Distal lymphedema is easier to control.

The Lymphapress is clearly the superior device. The differences are that our workup includes only duplex studies to rule out venous reflux, air plethysmography to rule out obstruction, and magnetic resonance to evaluate venous flow, and all this can be done on an outpatient basis so that the patients never enter the hospital, and the majority of our patients actually have, by third-party payers, purchased the device.

We have come to the point that four of our patients now are refractory enough and still disabled that two have been operated on by debulking procedures and two are scheduled.

I would like to know your feelings about surgery because it is clear that, although this is a program that is very useful, we still have to fall back on some surgical techniques.

Dr. O'Donnell. The role of surgery in patients with lymphedema is based on both the physician's recommendation and the patient's dissatisfaction with an enlarged limb. Patients with a markedly enlarged bulky limb may not be satisfied even with Lymphapress therapy. They will therefore opt for surgery. The indications for surgery, in my mind, are either failure to respond satisfactorily to conservative compression therapy with Lymphapress or the patient with a heavy, cosmetically displeasing limb desires a surgical procedure to reduce the size of that limb. We find that the Lymphapress is important in preparing the patients for surgery. The patient is admitted to the hospital for a 4-day treatment period with the Lymphapress before surgery. The leg is reduced to a tremendous degree so that the redundant skin can be pinched by the surgeon to determine the amount of tissue that can be removed. Three patients in this series (about 10% of our patients) eventually underwent reduction surgery, and the Lymphapress was helpful in managing them both before and after surgery.

Dr. Herbert I. Machleder (Los Angeles, Calif.). In managing a group of patients with a Lymphapress, rather than the proximal edema, I found that edema of the forefoot is very difficult to handle chronically. Do you have any recommendations in that regard?

Dr. O'Donnell. The foot can be difficult because of the conforming of the foot portion to the contour of the leg. It is very difficult sometimes to achieve compression of the foot.

In addition, if the patient is going to complain of pain during the treatment, it is usually in the foot area because of the way the cell conforms. We have tried putting elastic on underneath the compression device to help promote the reduction, but it can be an area that is resistant. I think the major problem with the foot is that patients usually complain of pain with therapy. It just does not fit well.

Dr. Machleder. Do you have any experience with the wrapping and heat therapy that the Chinese have reported with great success?

Dr. O'Donnell. No, I do not.