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The Lymphedema Chaos: A Lancet

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Pathophysiology of lymphedema is not an enigma. It is caused by a low-output failure of the lymph vascular system in combination with an inadequate scavenging of stagnating plasma protein by macrophages. Axillary venous diseases alone never cause chronic postmastectomy edema. In the diagnosis of lymphedema, invasive methods (i.e., direct lymphography and venography) are not only unnecessary but are potentially harmful and do not give any information of therapeutic relevance. Lymphedema of the limbs without reflux of lymph or chyle is not a surgical disease. It can be treated successfully by the skillful application of specific physiotherapeutic measures free of any side effect. The results of this therapy can be maintained if the patient's compliance is good.

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In this article we discuss some recently published conflicting opinions concerning the pathophysiology and therapy of lymphedema and present a plea for the conservative treatment of this "perplexing and poorly understood clinical entity" [21].

Physiology and Pathophysiology of the Lymph Vascular System

To understand lymphedema, the concept of the insufficiency of the lymph vascular system [13] has to be defined. The lymph vascular system is insufficient if its transport capacity is lower than the lymphatic load. The expression "transport capacity" is defined by the highest possible lymph flow per unit of time. We have proposed to distinguish three forms of lymph vascular insufficiency. Not conceiving or misunderstanding these three forms is a prime source of lymphedema chaos.

One form of lymph vascular insufficiency is called dynamic insufficiency. It arises if the lymphatic load exceeds the lymphatic transport capacity. In this case, the lymphatics are anatomically and functionally normal but overwhelmed. The main propulsive force of the lymph is generated by the contractions of the lymphangions (segments of lymphatics bordered by a distal and a proximal valve); stretch-sensitive pacemakers situated in each lymphangion cause lymph flow to increase automatically if input (i.e., intralymphatic pressure) increases. In addition, initial lymphatics increase the formation of lymph if water content and pressure in the interstitium increase. The relation of normal, resting lymph flow to lymphatic transport capacity is approximately 1 to 10 [23].

An increase in blood capillary pressure or a decrease of plasma colloid-osmotic pressure, by disturbing Starling's equilibrium (with a consecutive increase in net capillary ultrafiltrate) and, alternatively, a lesion of blood capillaries, causing their permeability to plasma proteins to increase, result in a compensatory increase of lymph flow.

A normal, healthy lymph vascular system is able, for some time, to handle an increase of the lymphatic protein and water load, preventing the appearance of edema. This is the "lymphatic safety factor" against edema. As long as the lymphatic load remains lower than the lymphatic transport capacity, lymphatic compensation is successful. If, in spite of the extremely elevated lymph flow, lymphatic load becomes higher than the lymphatic transport capacity, edema will appear. It has to be stressed that the type

of edema that is due to this high lymph flow failure has nothing to do with lymphedema.

A second form of insufficiency of the lymph vascular system is caused by a reduction, by some pathological process, of the lymphatic transport capacity below the level of a normal lymphatic protein load. According to the basic law of lymphology, the main task of the lymph vascular system is the clearance and the recycling to the blood vascular system of the protein molecules, which are continuously escaping from the blood capillaries [10]. This reduction of the lymphatic transport capacity results in a low lymph flow failure called "mechanical insufficiency." The consequence is a protein-rich edema: lymphedema.

If lymphatics with a reduced transport capacity are also overwhelmed by an increased lymphatic load, a third form of lymph vascular insufficiency arises, designated by us as "safety valve insufficiency." In the most severe stage of this insufficiency, lymphatic transport capacity is so low that, with a normal lymphatic load, lymphedema would arise. In addition, the lymphatic load is so high that a dynamic insufficiency of a healthy lymph vascular system would develop. In such an extreme case, protein-rich edema with diffuse necrosis of cells lying in the edematous area results.

Based on these pathophysiological aspects of the lymph vascular system, we discuss, in light of some recent publications, the present chaos in the concepts of lymphedema concerning the definition of the disease, its classification, its diagnosis, and its treatment.

Lymphedema: The Chaos of Concepts and of Diagnostic Measures

The deplorable state of affairs concerning lymphedema in the medical profession is highlighted by the publication of the 17th edition of Cecil's Textbook of Medicine [8]. There, Kontos devotes half a page on this subject. According to this author, "lymphedema refers to edema from accumulation of lymph. . ." There is an essential difference between lymph and tissue fluid [1, 12]; tissue fluid, not lymph, accumulates.

Witte and Witte [28] regarded the pathophysiological alteration which arises "when microvascular filtration rises to the point of overwhelming the rapidly draining but nonetheless inadequate lymphatic apparatus" causing "tissues also to swell" as an "other form of lymphedema." A "dynamic insufficiency," a high lymph flow failure, is, contrary to the view of these authors, not lymphedema. Not only is lymphedema an abbreviation of lymphostatic edema, but also the consequences arising in the tissues if proteins stagnate (proliferation of connective tissue) are totally different from those in which low-protein edema fluid streams rapidly through them.

A further example of the present chaos in the field of lymphedema is the fact that in the *Textbook of Dermatology and Venerology* by Braun-Falco and colleagues [4]—again confusing lymphedema (localized high-protein edema) with generalized low-protein edema—among the causes for secondary lymphedema, cardiovascular and renal insufficiency and protein carency are mentioned. A figure with the legend "Trophedema type Meige" shows a patient with a typical lipedema.

According to Miller [21], the fact that after the same type of surgery and irradiation, postmastectomy lymphedema arises in some patients and not in others "is unfortunately not clear" and makes "lymphedema a perplexing and poorly understood clinical entity." Miller's question can easily be answered: Axillary lymphadenectomy necessarily reduces the transport capacity of the entire tributary lymph vascular system (i.e., that of the arm and of the ipsilateral upper quadrant of the trunk) (Fig 1). The body now endeavors to overcome this by the simultaneous activation of the following mechanisms to compensate for the reduced lymphatic transport capacity:

1. Lymphatics spared by surgery and by irradiation start their "safety valve function."
2. Collateral lymphatic flow through axilloaxillary and axilloinguinal anastomoses becomes activated. The tributary area of the axillary lymph nodes comprises not only the arm but also the ipsilateral upper quadrant of the trunk. Interaxillary and axilloinguinal anastomoses are present, the extent of which is variable from individual to individual. Even more variable is the anatomy of the cephalic lymph vessels. The Table shows various types and how these are linked to the chance to avoid or to develop lymphedema.
3. Through connective tissue channels and through superficial dermal lymphatic plexus void of valves, protein-rich tissue fluid trickles out of the lymphostatic area into the bordering trunk quadrants possessing healthy lymphatics.
4. Lympholymphatic anastomoses develop at the axillary resection site.
5. Peripheral lymphovenous anastomoses may establish new connections between veins and lymphatics.

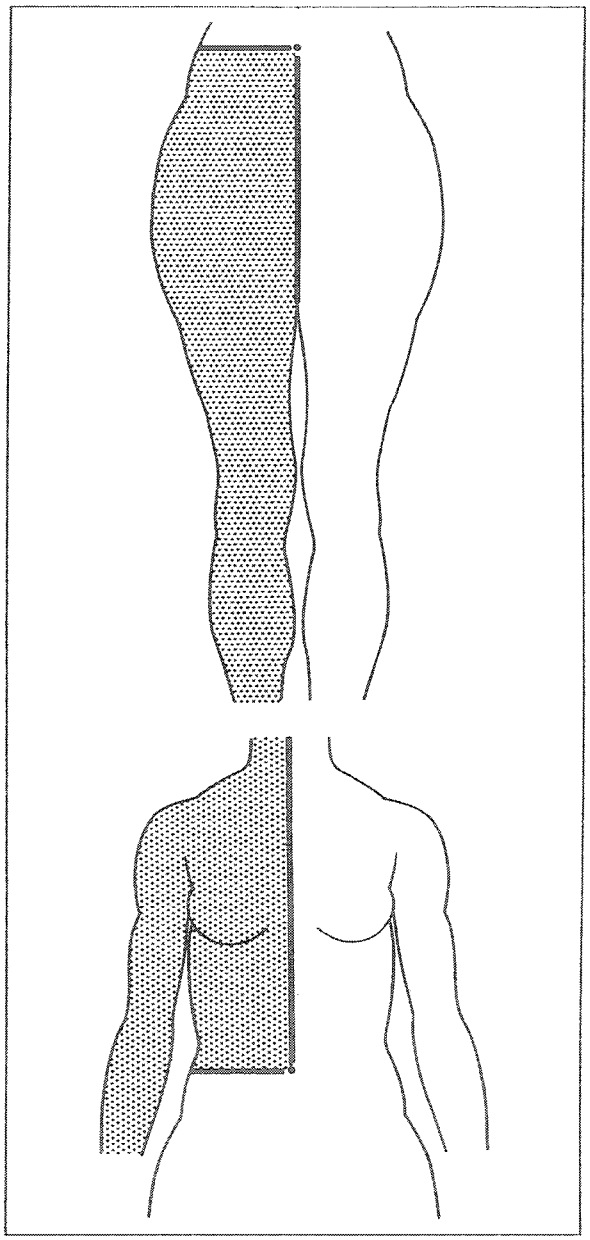


Fig 1. The tributary area of the axillary and the inguinal lymph nodes. Axillary lymphadenectomy may result in secondary lymphedema not only of the arm but of the ipsilateral upper quadrant of the trunk. Removal of the inguinal lymph nodes or their fibrosis may result in secondary lymphedema not only of the leg, but of the trunk.

Anatomical Variations of Cephalic Lymph Vessels and Consequences for Adequate Collateral Lymph Flow after Axillary Lymphadenectomy

Type of Cephalic Lymph Vessel	Risk of Collateral Lymph Flow after Axillary Lymphadenectomy
Long,[a] regional lymph node supraclavicular	Excellent
Long, regional lymph node infraclavicular	None
Short,[b] regional lymph node supraclavicular	Moderate
Short, regional lymph node infraclavicular	None

[a]The system is linked to lymphatics draining the lower arm.
[b]The system starts at the olecranon.

6. Monocytes, escaping in increased numbers from the blood capillaries in the lymphostatic region, transformed into macrophages, start proteolytic breakdown of stagnating plasma proteins ("extralymphatic mastering of plasma proteins") [13].

The regeneration of interrupted lymphatics by lympholymphatic anastomoses is known to be highly sensitive to scar formation. Postoperative seromas, irradiation, and unskilled early physiotherapy involving the shoulder induce this and even hamper the protein-rich tissue fluid from being removed from the lymphostatic area through tissue channels and the superficial lymphatic plexus.

The mentioned false concept of pathophysiology of lymphedema is also reflected in the monograph "Cancer—Principles and Practice of Oncology" [11]: "It has never been explained why some mastectomy patients develop lymphedema and some do not. Some may go several years with no lymphedema, only to develop massive edema." Lymphedema is a quantitative problem between the lymphatic load and the lymphatic transport capacity. If the compensatory mechanisms as outlined previously are in the short or long run insufficient, the equilibrium between lymphatic protein load and lymphatic transport capacity will become overthrown; if lymphatic transport capacity decreases below the level of the normal lymphatic protein load, lymphedema will immediately arise.

The chaos in the literature also involves the role of

venous hemodynamics in the pathogenesis of lymphedema. Recently, several authors [11] expressed the following view: "To help determine the pathology and aid treatment, it is important to know whether the cause of lymphedema is a disease of the lymphatic or venous system. Venograms and lymphangiography may aid in making this differential diagnosis." This view contradicts experimental data and clinical experience.

In the experimental animal, chronic edema will never arise as a consequence of the ligature of a large vein as, for example, the axillary. On the other hand, by meticulously blocking the lymphatics of a limb, if regeneration is prevented by an adequate method, chronic irreversible lymphedema can readily be produced. In these animals, there is no concomitant venous pathology [19]. By ligating a large vein, in addition to a lymph block, the extent of the lymphedema will of course increase.

Thrombosis of the axillary vein (Paget-von-Schroetter syndrome) or ligation of the axillary vein is accompanied by an acute edema. This edema subsides after two weeks even if the axillary vein remains definitively occluded as a collateral venous circulation develops. Chronic edema of the arm, comparable to post-mastectomy swelling, never arises. It has been shown, moreover, that the resection of an entire segment of the axillary vein, if performed in addition to Halsted's operation, does not increase the rate or severity of postmastectomy lymphedema [20]. Hence, chronic postmastectomy swelling of the arm is always due to a disease of the lymphatic and never to that of the venous system. The advice given [11] to perform venograms is rejected because, first, venography may aggravate lymphedema. Second, venography is not without hazards [19]. Third, the venogram has no therapeutic consequence. Fourth, venous disease can readily be assessed by inspection (cyanosis, dilated veins, collateral veins) and by various noninvasive diagnostic tools of phlebology. The presence of visible symptoms of venostasis has to arouse the suspicion that the cause of postmastectomy lymphedema is the malignancy itself (that is, the blockage of lymphatic and venous flow by an axillary metastasis). Again it is not phlebography but the clinical history, palpation, computed tomography and/or magnetic resonance imaging, and biopsy that has to establish etiological diagnosis of lymphedema (benign or malignant).

The concept that some cases diagnosed clinically as lymphedema are caused by venous disease goes back to Calnan [6], who assumed that the cause of primary lymphedema of the left leg may be a "crossing syndrome," caused by the compression of the left iliac vein by the iliac artery. This false view, which culminated in the paper "Lymphedema—a Case for Doubt" has led to unnecessary operations with the aim of liberating the vein from the compression by the artery. At best, for the patient, there is no postoperative increase of lymphedema. In postmastectomy lymphedema, too, venolysis, the liberation of the axillary vein from scar tissue, is unreasonable; there are no statistical reports of any success in reducing the swelling.

To dissect the vein, lymphatics still in function will be severed, small veins will be cut, and the removed scar tissue sooner or later reforms. The advice [11] to perform arm lymphangiography to "aid in making . . . differential diagnosis whether the cause of the lymphedema is a disease of the lymphatic or venous system" is rejected: Lymphangiography may aggravate the swelling and has a mortality rate [26]. Moreover, only as an exception will lymphangiographic data influence therapy.

An unusual idea has recently been described by Bland and co-workers [3]: "The pathogenesis of ipsilateral arm lymphedema following mastectomy is the ablation of the lymphatic system (nodes and channels) within the en bloc resection of the primary mammary tumor. The prevention of the subsequent increase in plasma hydrostatic pressure that follows the removal of these conduits may follow the surgical procedure, irradiation . . ." How could the removal of lymph nodes from the lymphatic system lead to an increase in pressure of the blood vascular system?

A rather uncommon concept is expressed by Watts [27]: Lymphedema does not exist at all, "since total lymphatic obstruction of lymphatic does not cause lymphedema." It has been experimentally demonstrated that a total and irreversible blockage of the lymphatics of one extremity, with the venous outflow undisturbed, will result in a lethal lymphedema: The skin ruptures and the animal loses enormous amounts of protein-rich fluid resulting in shock [9]. Watts' question, "Why does lymphedema differ from ordinary pitting edema?" proves that this author may not have observed lymphedema patients in the first pitting stage and that he is unaware of the fact that protein-rich edemas, after a first pitting stage, regularly develop into the second, nonpitting brawny stage.

Lymphedema: The Therapy Chaos

The range of approaches to lymphedema in the medical profession involves, at one end of the spectrum, therapeutic nihilism ("keep up with it!") and at the other a multitude of operations. Between these two extremes lie different conservative treatment methods, some of which are in contradiction to basic lymphology. Decongestive physiotherapy, which we advocate, will be discussed later.

Should Lymphedema Be Treated at All?

Lymphedema is a high-protein edema. This has important pathological and clinical consequences. If lymphedema arises, for example, as a consequence of a surgical intervention, reducing lymphatic transport capacity definitely below the level of the normal lymphatic protein load, tissues will become inundated by a protein-rich edema fluid. In this first stage, stage I or reversible lymphedema [17], edema is pitting and the swelling may become temporarily reduced by simple elevation of the affected limb. If protein-rich edema persists, proliferation of connective tissue combined with the deposition of some adipose tissue as well will sooner or later result in a progressive hardening of the affected area. The pitting character of the swelling diminishes continuously, and finally it becomes impossible to induce a finger imprint. Elevation of the limb will not reduce the swelling any more. This stage II is called spontaneously irreversible. It has to be stressed that in spite of the brawny character of the involved tissues as a consequence of fibrosclerosis protein-rich fluid still remains in the lymphostatic area. This fact, which holds true even for stage III of lymphostatic elephantiasis, has important therapeutic consequences.

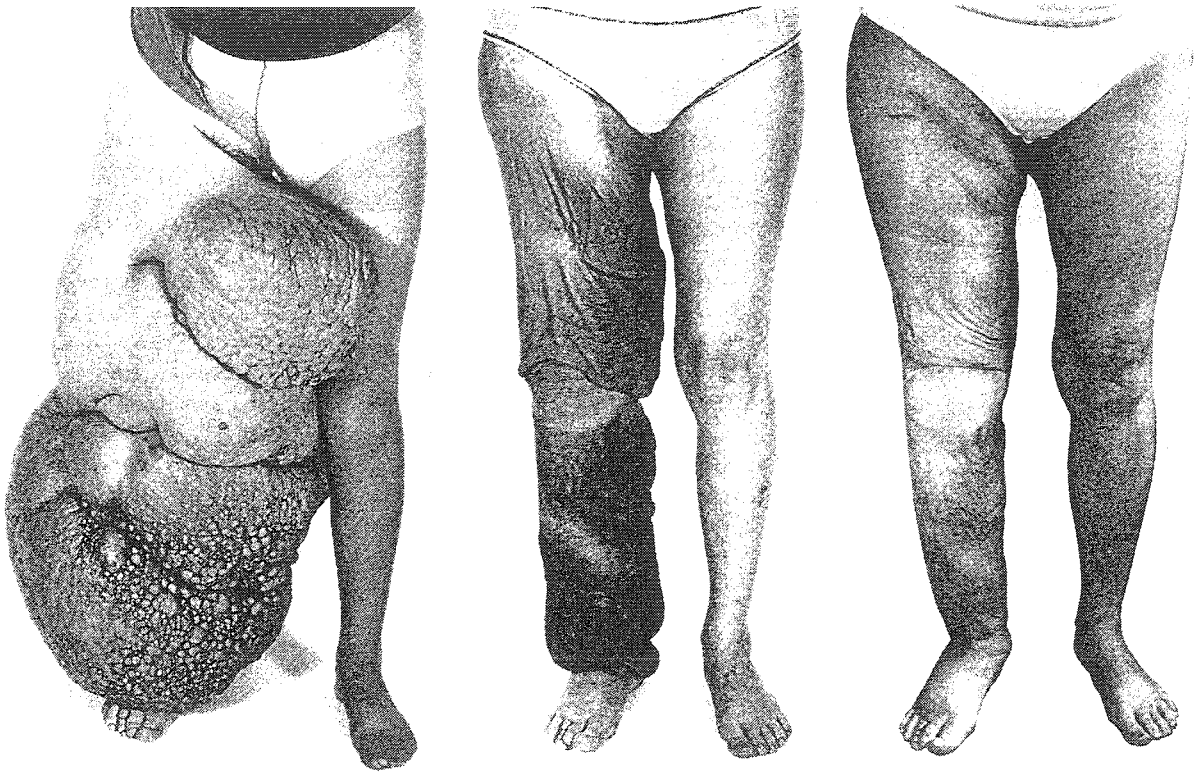
Lymphostatic elephantiasis is characterized by: (1) a tremendous increase in volume, leading to the association with the limb of an elephant; (2) a cartilage-like hardening of the dermal tissues (pachyderma) (again the elephant is designated by zoologists as a pachydermic animal); (3) papillomatous outgrowths, sometimes the formation of enormous lobuli (Figs 2A, 3A). Elephantiasis is not only a debilitating condition, it also threatens life: Stewart-Treves syndrome (angiosarcoma) may arise. Hence, lymphedema has to be treated, and treatment has to be initiated as early as possible.

False Conservative Treatment Methods

For the treatment of primary lymphedema, Kontos [8] advocated "frequent elevation of the limb, the use of elastic stockings," and he regards the administration of diuretics as "potentially useful." (There is no mention of the treatment of secondary ones—in the United States and in Euope, the most frequent form of lymphedema is secondary lymphedema.) With regard to elevation of the limb, this procedure is somewhat impractical and of temporary help even in the first pitting stage. Only in the early stage of edema of venous origin of the lower limbs will elevation be followed by a rapid evacuation of low-protein edema. It is a common misconception to order elastic stockings for the treatment of lymphedema. Elastic stockings have to be used to conserve a state free of edema achieved by successful therapy and not to evacuate edema (see later discussion).

The recommendation of diuretics contradicts pathophysiology of lymphedema and the pharmacological action of diuretics. Diuretics may be indicated for generalized low-protein edemas accompanied by an elevated sodium content of the body. Lymphedema, however, is not due to sodium retention. Diuretics are able to draw away, by hemoconcentration, water held by the colloid-osmotic pressure of the stagnating protein molecules; this is the underlying pathology, not sodium retention. However, the proteins themselves, the primary cause of the swelling, will remain in the tissues. Moreover, their concentration in the edema fluid will increase, so in the long term the involved tissues will become even more fibrotic, harder than they would have been without diuretics.

According to Kontos [8], "pneumatic compression (pneumomassage) can be helpful in increasing the flow of fluid out of the limb." In postmastectomy lymphedema, this type of treatment disregards the anatomic fact that the ipsilateral quadrant of the trunk is also involved in lymphostasis (see Fig 1). By pushing the protein-rich edema fluid from the arm into the trunk, there will be an increase of edema in the trunk quadrant, from which lymph should be drained away by the normal lymphatics situated in the bordering quadrants. Consequently, in the long run, the lymphatic drainage from the arm will be reduced. The same considerations hold true for lymphedemas involving the leg and the ipsilateral lower quadrant of the trunk as well as the genitalia.



A B C

Fig 2. (A) Lymphostatic elephantiasis in a 38-year-old woman (primary lymphedema) before treatment. (B) After decongestive physiotherapy. (Later some large empty skin-folds had been removed by plastic surgery.) (C) Five years after treatment was initiated.

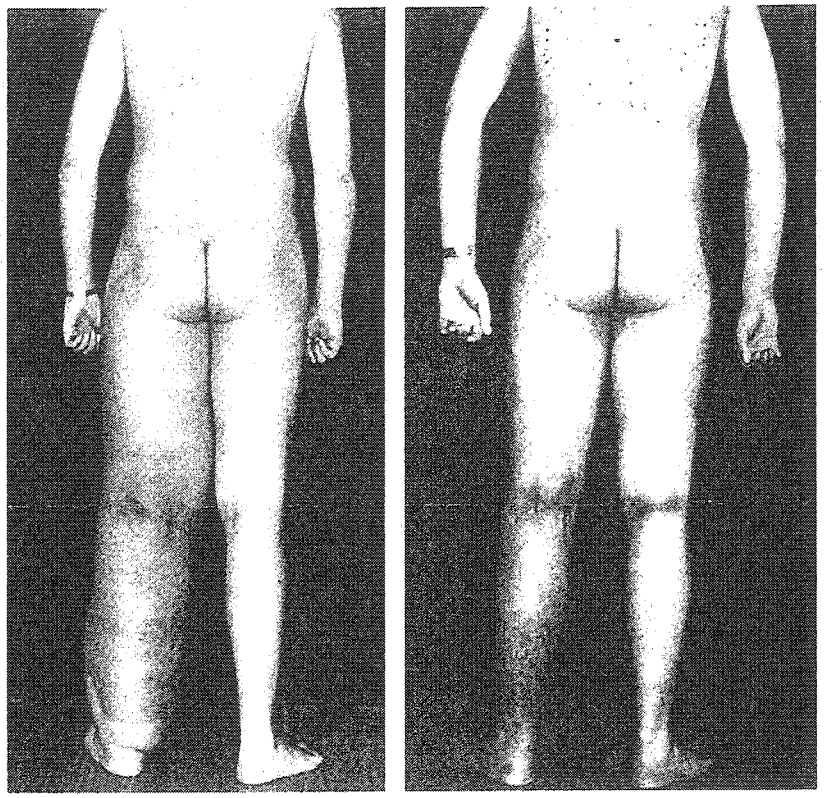


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Fig 3. (A) Lymphostatic elephantiasis in a 20-year-old man (primary lymphedema) before treatment. Seven years before a lymphovenous shunt operation had been performed, which resulted in a deterioration. (B) The result of complex decongestive physiotherapy.

Surgery of Lymphedema

From the pathophysiological point of view, surgery of lymphedema should either increase the lymphatic transport capacity or reduce the normal lymphatic load to permanently achieve its goal: a definitely stabilized balance between the lymphatic load and the lymphatic transport capacity. All lymphedema surgeons agree that a true surgical cure of lymphedema—a restitutio ad integrum—is a rare exception. All patients will have some residual edema [21]. Surgery "may be tried in advanced cases, to remove subcutaneous tissue and induce new lymph vessel formation" [8]. The authors are unaware of any research maintaining that "new lymph vessel formation" could be induced by resective surgery. Kontos [8] stated that "anastomoses of small lymphatic vessels with veins by microsurgery has been reported" to give good results "in some cases." For the individual surgeon and for the patient, it is essential to know in which particular case a good result may be obtained and what is a "good result" in surgery of lymphedema.

In 1985 Hurst and associates [17] published their paper, "Long Term Results of the Enteromesenteric Bridge Operation in the Treatment of Primary Lymphedema." The operation consists of the isolation and opening of a segment of the ileum with its mesentery. The segment is opened at its antimesenteric border. The mucosa is stripped off. The inguinal nodes are bisected or deroofed, and the bridge is sutured down over them. Altogether 8 patients were operated on in this manner. One patient developed a small bowel obstruction, which of course necessitated surgical intervention. Six patients (75%) showed sustained clinical improvement. Four of these 6 patients (66%) "wear stockings and adhere to a programme of massage and limb care." By these results, the authors "are encouraged . . . and now feel justified in offering this operation to carefully selected patients . . ." This conclusion, in our opinion, is open to debate. To select suitable cases for enteromesenteric bridge oper-

ation, direct lymphography with an oily contrast medium must be performed. This may aggravate lymphedema and, as already pointed out, is affected by a mortality rate. The danger of ileus cannot be overlooked. Moreover, general anesthesia is necessary for the operation.

Because every lymphedema of the benign type (not caused by malignancy) can be treated with great success by decongestive physiotherapy, the indication for surgery is a matter of great caution. The use of prosthetic material (e.g., nylon threads) with hopes of reestablishing lymph flow within scar tunnels simply disregards the fact that the propulsive force of the lymph flow is furnished by the pulsations of the lymphangions. The same holds true for implanted tubes. A blood vessel may readily be replaced by a tube because the heart pumps the blood through it, but there is no force that could propel lymph through an artificial valveless tube. Strangely enough, Handley's [15] 1908 paper advocating the implantation of threads is referred to by those who still employ these techniques, but the same author's revocation published two years later is regularly missing from the list of references [16].

The buried dermal flap of Thompson, which consists of the creation of a flap of deepithelized dermis introduced into the deep muscle compartment in the hope that the flap will drain lymph from the superficial compartment into the noninvolved muscle, disregards the anatomical fact that the deep lymph compartment, after axillary lymphadenectomy, is not less affected by lymphostasis than the superficial.

Concerning surgery of lymphedema, we cite Goldsmith and De Los Santos [14]: "The large number of operations devised for improving lymphatic drainage from a chronically lymphedematous limb indicates the lack of a surgical procedure which is consistently effective. Because of the relative rarity of chronic lymphedema, only a few surgeons have been able to develop a sizable clinical experience with the problem. This has meant that the majority of surgeons who see only an occasional patient with chronic lymphedema usually must rely upon the reports of others in deciding the best operation to offer their patients." They continue, "Unfortunately, even surgeons with a relatively large experience with chronic lymphedema do not have enough patients or the inclination to carry out a controlled clinical experiment. This results in the surgeon championing the operation found most effective in his hands without his ever developing sufficient experience with other procedures to test their clinical effectiveness or to learn the operative techniques necessary to minimize their postoperative complications. The author has performed only omental transpositions for chronic lymphedema and therefore must accept this criticism. A carefully controlled clinical study, which allows objective evaluation by unbiased observers of long-term results of various surgical procedures for chronic lymphedema, is truly needed."

The physiological operation conceived by Baumeister [2] consists of lymph vessel transplantation. This operation does not solve the problem of the quantitative relation between the lymphatic load and the lymphatic transport capacity either: Lymph scintigraphic controls performed by Baumeister have shown an improvement, but not the necessary normalization of lymph flow. According to Jungbluth [18], the number of axillary lymph vessels varies between one and ten; 51.6% of women have four to ten axillary lymphatics. This means that in these patients the transplantation of two lymph vessels taken from the leg will be unable to restore lymphatic transport capacity to the preoperative state. Moreover, these transplanted lymphatics have to pump lymph continuously at the level of their transport capacity (i.e., the highest possible lymph flow). This fact will sooner or later cause the transplanted lymphatics to stop functioning: If intralymphatic pressure is elevated, lymph moves into the wall of the vessel, and this protein-rich fluid induces fibrosis. Pulsation of lymphangions will come to a standstill [9].

After successful treatment of an elephantiastic limb by decongestive physiotherapy, large, empty skinfolds replace the huge lobuli. Their removal by a skilled plastic surgeon—on an outpatient basis—is a relative indication for surgery, because these skinfolds make the wearing of compressive stockings and sleeves difficult.

One of the most unwanted consequences of advocating false or insufficient methods of conservative lymphedema treatment is that it provides false arguments for those surgeons who are determined to operate. After a false unsuccessful conservative treatment, the surgeon may plead for an operation because of the progressively debilitating condition. Elevation of the arm, even if combined with the continuous administration of diuretics, the use of which is unfortunately advocated [11], will always be unsuccessful; hence, every patient treated in this manner becomes a candidate for surgery.

Complex Decongestive Physiotherapy

The aim of the conservative treatment of lymphedema must be the permanent restoration of the disturbed equilibrium between lymphatic protein load and lymph vascular transport capacity (i.e., of the protein content of interstitial fluid to normal). The only physiological way to achieve this goal is to cause lymphatics to remove the excess plasma proteins from the tissue. This can be accomplished in both primary and secondary lymphedemas by the skillful application of various physiotherapeutical measures. Treatment consists of five parts of equal importance. First, hygienic measures and the eradication of fungal affections are mandatory. In most cases, this will suffice to abolish attacks of erysipelas. If not, antibacterial treatment, eventually lifelong, must be started.

Second, manual lymph drainage must be undertaken. The first goal of this special massage technique, commenced over the contralateral quadrant of the trunk free of lymphostasis, is to increase lymphocinetic activity in these normal lymphatics (Figs 1, 3). It is known that lymphangions increase their output if subjected to mild mechanical stimuli [18]. These lymphatics now start to drain the lymphostatic quadrant across the lymphatic watershed. The decongestion of the lymphostatic trunk quadrant will then allow edema fluid to pass through dilated tissue channels (and, if present, lymphatics) from the limb, first into the ipsilateral, then into the contralateral trunk quadrant. This will, of course, start centrally, the decongestion finally arriving in the tip of the fingers and toes. Pecking and associates [24] have shown that the speed of the lymphatic transport of a tracer injected into the swollen hand of a patient suffering from postmastectomy lymphedema increases immediately if the contralateral normal quadrant is treated by manual lymph drainage. The second step consists of pushing edema fluid gently from the lymphostatic quadrant into the normal one, the healthy lymphatics of which have previously been stimulated (i.e., prepared for additional work). The upper arm (thigh) will be evacuated if the ipsilateral quadrant of the trunk is already free of edema; treatment of the lower arm (leg) comes later; the latest step consists of the treatment of the hand (foot).

Third, from the beginning of the treatment, bandages are applied over the lymphedematous limb. One of the first pathological consequences of lymphedema is a destruction of elastic fibers; therefore, elastic insufficiency of the connective tissue of the subcutis will add to the effect of the decrease of tissue pressure brought about by the evacuation of edema fluid by manual lymph drainage. If tissue pressure decreases, effective ultrafiltrating pressure increases. Without bandaging, the edema fluid would reaccumulate. Bandages not only counterbalance elastic insufficiency but increase tissue pressure. It has been shown that there is a positive correlation between tissue pressure and lymph flow.

Fourth, remedial exercises, performed while wearing the bandages, enable muscle and joint pumps to exert their lymphocinetic effects. Fifth, at the end of the treatment, which lasts approximately four weeks, an elastic support, as strong as can be tolerated by the patient, made to measure, must be prescribed. In Figure 3B the result of complex decongestive physiotherapy in lymphostatic elephantiasis of the leg is demonstrated.

In the benign form of postmastectomy lymphedema of the arm, volume reduction could be achieved in 95% of the patients. In the malignant forms, in which the transport capacity of the lymphatic system has been reduced by the blockage of the lymphatics by tumor cells, volume reduction could be achieved in 51%. Three-year follow-up has shown that 54% of the patients belonging to the benign group had fully maintained the result of therapy. Of the patients in this group 35% have shown some partial relapse, but the original pretreatment volume did not return. A full relapse occurred in 10%, and this was due to the patients' failure to comply, abandoning compressive bandages. In the group of malignant lymphedemas, the result of therapy could be maintained in only 14%, and a total relapse occurred in 52%.

These statistics are based on 399 patients with a benign lymphedema and 119 patients with a malignant lymphedema concerning the immediate effect of complex decongestive physiotherapy. The statistics concerning the long-term results are based on 177 patients with a benign type and 21 patients with a malignant type (Figs 4, 5). Needless to say, most concomitant diseases (diabetes, obesity, cardiac failure, idiopathic edema, lipedema, and so on) aggravate lymphedema and must be treated adequately. If lymphedema is caused by a malignancy blocking lymphatics, oncological treatment must be performed, eventually in combination with physiotherapy.

Lymphedema of the limbs, if not complicated by a reflux of lymph or chyle, can be treated effectively by conservative methods free of mortality, of general an-

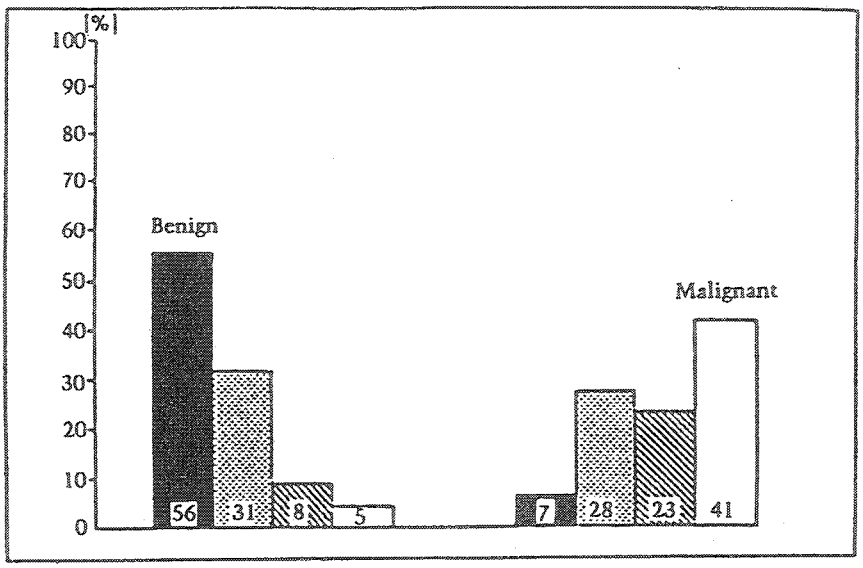

Fig 4. Immediate results of complex decongestive physiotherapy in patients suffering from postmastectomy lymphedema of the arm. (□ = no change in respiratory increase; ▨ = reduction = 1–24%; ▩ = reduction = 25–49%; ■ = reduction = > 50%.)

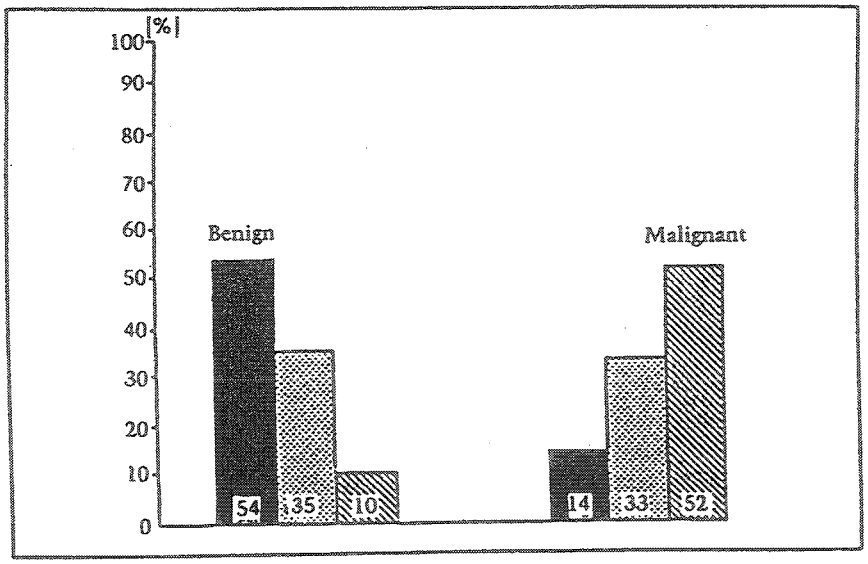

Fig 5. Long-term results of complex decongestive physiotherapy in patients suffering from postmastectomy lymphedema of the arm. (▨ = result not maintained; ▩ = insignificant increase; ■ = result maintained.)

esthesia, and the possible morbidity of surgical interventions. Neither excisional nor physiological types of operations nor amputations to handle postmastectomy arm lymphedema in advanced cases should be performed [11].

Benzopyrones

There is ample evidence that benzopyrones exert their moderate therapeutic effect by stimulating macrophages to scavenger the stagnating plasma proteins. Two problems arise, however. First, even a high dosage will reduce lymphedema in the course of five years to the extent decongestive physiotherapy can readily achieve in the course of four weeks [18]. In other clinical trials [7], a volume reduction of approximately 20% could be achieved in the course of six months by benzopyrones. Treatment by decongestive physiotherapy is not only much shorter, but the reduction in volume is also much higher.

Second, the lifelong administration of benzopyrones in any lymphedema involving a major part of the body (e.g., a limb) as a sole therapeutic measure means that protein catabolism increases as a consequence of the continuous degradation of protein molecules, which escape from the microcirculation. Careful studies have to elucidate whether this increased protein catabolism will not result in some side effects if the diet of the patient is not adequate both qualitatively and quantitatively. Most of the patients suffering from lymphedema caused by filariasis are, for example, undernourished.

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