Should peripheral lymphedema be treated by non-operative means, drugs, or by operation? From Avicenna, who recommended long skin incisions with a knife and covered them with honey (1) to modern times, there have been innumerable operations that have been advocated for treating lymphedema. Fortunately, we have progressed from the misguided ideas of Carnochan who advocated ligation of the femoral artery in 1851 (2) or Morton who recommended transection of the sciatic nerve in 1878 to improve leg lymphedema (1).

Historically, recall that Handley (1908) suggested silk threads and Ransohoff in 1945 proposed polyethylene tubing with perforations to substitute for lymphatics, an idea resurrected by the Brazilian surgeon Degni in 1975 (3). Lanz in 1911 buried fascia near muscles to promote lymph drainage, an idea modified by Thompson in 1966. Kondoleon, Sistrunk and Auchinloss in the first half of the 20th century proposed a variety of excisional therapies with or without supplemental skin grafts, whereas Kimura (1925), Gillies (1935), Martorel (1958), and Azpurua (1966) favored various pedicle grafts to enhance lymph transport (4).

Other resectional procedures were advocated by Servelle (1975), Josías-Mayall (1969), Di Gaetano (1928), Clodius (1977), whereas Murphy (1906), Reinoff (1937), Treves (1952), Peck and White (1992) advocated transplantation of muscles to help drain stagnant lymph. Other surgeons who tackled the problematic swelling of lymphatic dysfunction include Lexer (1919), Mowlen (1948), McDonald (1948), Opel (1912), Overton (1935), Gibson-Tough (1954), Jantet (1961), Barinka (1977), Bunchman (1977), Smith and Conway (1968), Kaufman (1977), Clodius (1977), Kinmonth (1982), and many more.


Unfortunately, despite the plethora of operations described, the overall results have been less than overwhelming for a chronic illness that afflicts millions worldwide. Some surgical groups continue to present excessively optimistic statistics (8,9).

In my personal experience, however, with operations for lymphedema—both resective procedures (Josías-Mayall, Servelle, Thompson) and “physiologic” operations (lympho-venous shunts and pedicle
A) Most publications that advocate operations for lymphedema still incorporate non-operative methods to reduce limb swelling including manual drainage, pressotherapy, diuretic drugs, corticosteroids, elastic compression, heating, and bandaging. Some surgeons favor that an operation is indicated when non-operative treatment fails and yet they continue to use these “failed” methods in the post-operative period to complement the operation (9,11,13).

B) “Lymphatic” surgeons emphasize that a “healthy” lymphatic must be present to construct a successful lymphatic-venous shunt (13). Lymphatic trauma or neoplastic invasion of the lymph node with secondary lymphedema renders it unlikely that residual collectors are non-dysfunctional. This is even more true when lymphedema is secondary to repeated lymphangitis and cellulitis (erysipelas).

C) Lymphatic transfer is recommended to stimulate lymphatic “neoformation,” but I suspect lymphangiogenesis is an ongoing process without a “transfer operation” (14-16).

D) The principle of a lymphatic-venous shunt is to drive hypertensive lymph into a venous tributary of much lower pressure. Yet, once the lymphatic and venous pressures reach equilibrium and are balanced, does lymph flow continue indefinitely?

E) Casley-Smith published an adaptation of Földi on the organic causes of lymphedema. These include: 1) decreased tissue channels, 2) decreased initial lymphatics, 3) damage to initial lymphatic membrane, 4) torn anchoring filaments, 5) decreased collecting lymphatics, 6) incompetent intralymphatic valves, and 7) gaps in the collecting lymphatic membrane. As these lesions occur in the initial lymphatics or collectors distal to proposed lymphatic-venous shunts, I doubt benefit can accrue from an operation proximal to these uncorrected derangements (17).

F) Although a lymphatic-venous shunt is a relatively minor operation (albeit tedious), a surgeon must first do no harm. For example, in post-mastectomy lymphedema after axillary dissection, perhaps only 2 or 3 “healthy” lymphatic collectors remain. Accordingly, one can run the risk of making a shunt with these few residual lymphatic vessels knowing that its failure may lose the critical threshold preventing lymphedema.

Whereas my 10 years of dedicated efforts to surgically improve lymphatics has largely been a failure, I do believe that there are clinical circumstances that still favor operative management. For example,

A) In patients with chylous reflux, ligation, and obliteration of retroperitoneal lymphatics with an intraabdominal lymphatic-venous shunt is reasonable (18).

B) In advanced lymphedema of the scrotum, penis or vulva, “debulking” even if self-limited is still the best option (19).

C) In chronic lymphedema, with a grotesque increase in the mass of the subcutaneous tissue that is disabling due to the weight and deformity of the limb, “debulking” to diminish the exuberant soft tissue is worthwhile but not with the pretense of curing the condition (20,21).

D) In unusual circumstances of lymphedema where lymphatics appear healthy, demonstrate good contraction in the absence of structural defects and incompetent valves, it may be justified to attempt a lymphatic-venous or lymphnodal-venous shunt (22).

Whereas some may agree and others may disagree with these thoughts, we still need to know the intricate mysteries of the lymph-vascular system including its microelements and factors that govern lymphatic growth and development (lymphangiogenesis) (14-16) if we are to find the optimal answer to the vexing global problem of lymphedema.

REFERENCES


Dr. Raul Beltramino
Service of Phlebology and Lymphology
Sanatorio Parque
Saavedra Lamas 450
4107 — Yerba Buena
Tucuman, Argentina

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