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Lymphedema Surgery: Patient Selection and an Overview of Surgical Techniques

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Evaluation of the lymphedema patients with appropriate staging is fundamental for further treatment. Treatment includes compressive decongestive therapy for stage 0 and 1 patients, lymphovenous anastomosis for stage 1 and 2 patients, vascularized lymph node transfer for stage 2 and above patients. Wedge resection, liposuction, and the Charles procedure are alternatives or additions to physiological procedures. The selection of donor lymph node flap and recipient site depends on the patient's lymphedema status and surgeon's expertise.
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LYMPHEDEMA: DEFINITION AND PATHOPHYSIOLOGY

The lymphatic system provides several functions: prevention and clearance of edema, regulation of interstitial fluid homeostasis, immune system transportation and surveillance, and absorption in the gastrointestinal tract [1]. To these effects, the lymphatic system is an essential element of both the circulatory and immune systems.

It is estimated that there are between 600–700 lymph nodes in the body, with main concentrations found in the axilla, groin, mediastinum, and gastrointestinal tract [2]. Lymph fluid is transported into and out of lymph nodes via afferent and efferent lymphatic channels. Recent evidence has also demonstrated lymphaticovenous connections that provide drainage of surrounding tissues into the lymph node as well as out of the node back into the local venous network [3]. When these processes are disrupted, the result is lymphedema.

Up to 250 million people worldwide suffer from lymphedema, which can be divided into two major types [4]. Primary lymphedema results from genetic or developmental anomalies, while secondary lymphedema results from postnatal causes including trauma, infection, malignancy, or radiation to the lymphatic system. Primary lymphedema is further classified based on time of onset into congenital lymphedema, lymphedema praecox, and lymphedema tarda. Congenital lymphedema presents at birth or within the first 2 years of life, lymphedema praecox typically presents around puberty and before the age of 35. In contrast, lymphedema tarda presents in patients over the age of 35 [4]. For the basis of this review, we will focus on secondary lymphedema unless otherwise specified.

Worldwide, the leading cause of lymphedema is filiriasis, a parasitic infection caused by the roundworm *Wuchereria bancrofti*, which mostly affects developing countries [5]. These roundworms infiltrate the lymphatic system causing secondary lymphedema via obstruction. In developed countries, however, the leading cause of lymphedema is the consequence of oncologic therapies [6]. Breast cancer treatment in the forms of lymph node dissection and radiotherapy is the classic precursor of secondary lymphedema, but it is also observed in patients undergoing treatment of solid tumors elsewhere in the body. Whatever the cause, obstruction of or injury to the lymphatic system results in a specific series of events that leads to lymphedema. Fortunately, all patients undergoing these cancer therapies do not develop lymphedema. For example, between 29–49% of patients that undergo axillary lymph node dissection will develop lymphedema, although only 5–7% of patients that undergo sentinel lymph node dissection will suffer the same fate [7,8]. And for the patients that do develop lymphedema, the onset of disease is highly variable and infrequently immediate. On average, these patients develop lymphedema within 8 months of surgery, with 75% developing signs of lymphedema within the first 3 years [9]. This variability in incidence, onset and progression has revealed several independent risk factors for the development of lymphedema: obesity, radiation, infection, and genetics [7,9]. Not only is the timing of lymphedema variable, but the progression of disease also differs widely among patients. To understand this progression, knowledge of the pathophysiology of the disease process is necessary.

Although incompletely understood, the natural progression of lymphedema proceeds from a buildup of protein-rich fluid in the interstitial space, resulting in the early symptoms of soft, pitting edema in the affected extremity [10]. This initial event has been shown to cause inflammation of tissues and stimulation of fibrosis via a number of mechanisms [11,12]. With time, worsening lymphatic function results in adipose deposition in the subcutaneous tissues [13]. All factors feed back in a positive fashion to worsen the symptoms of lymphedema which progresses to a thick, fibrotic,

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