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COMMENTARY

ON TREATMENT OF PERIPHERAL LYMPHEDEMA

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Since 1995, many of us have suspected that peripheral lymphatic failure is often complicated by venous failure and by changes in the organs drained. Moreover, the epidermis and adipose tissue are not simply passive players but are likely factories of growth factors and mediators of inflammation.

We should add to lymphedema management recognition that measures which facilitate emptying the venous system are part of the care of the lymph vascular system. Although such maneuvers are probably unsuspectedly already practiced, phenomena such as proper breathing, exercise and limb elevation should be thought of as being maximally effective when aimed also at the venous system.

We should also add to lymphedema management all that has been learned by dermatologists concerning the huge reservoir of cytokines manufactured by the epidermis. These agents are probably responsible for the soft tissue overgrowth observed in lymphedema and perhaps are also responsible for recurrent inflammatory episodes. Bacterial and fungal contamination of the epidermis should be seen as generating an outpouring of such cytokines which also play a role in their own functions. It is well known that these microorganisms survive on an "irritated" epidermis more easily than on a normal epidermis. Soothing the epidermis by application of external emollients that are anti-inflammatory are, accordingly, important in the treatment of many skin conditions and should be looked upon as being pro-active in the management of lymphedema.

Like the epidermis, fat cells have long been regarded as bystanders rather than active participants in lymphedema. It is clear, however, that they too can generate cytokines and hormones and a catalogue of their activities increases on a monthly basis. Indirectly, obesity contributes to lymphedema by discouraging mobility and furthering weight gain.

A revised consensus document should give greater weight to these ideas.

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