

LETTER TO THE EDITOR

In his letter [Lymphology 35 (2002), 130], Arnfinn Engeset raised an important point dealing with the vulnerability of subepidermal lymphatics to mechanical trauma. Indeed, one would think the dermal network and collecting lymphatics in skin and subcutis should undergo traumatic changes resulting in local lymph stasis – especially since small veins and arteries become damaged as evidenced by edema and hematoma. The problem of injury of lymphatics has been our interest since we started our studies on the pathomechanism of posttraumatic edema in humans with fractures and injury of soft tissues of the lower limbs. This type of protracted swelling can last for months or years. Its mechanism is unclear, and most attention is directed at veins but not lymphatics. Interestingly, only around 30% of patients have venous thrombosis diagnosed on ultrasonography (1,2). What is then the cause of prolonged edema in the remaining 70% of patients? The first thought would be damage to lymphatics. In experiments on mice, we applied trauma to the leg, defined as 50% of the minimal energy needed for tibia fracture. Intravenously injected fluorescein isothiocyanate- dextran 150 kDa and rhodamine-6G were used for intravital fluorescence microscopy of blood vessels below, at, and above the site of injury. Lymphatics were stained with FITC-dextran 150 injected into the footpad. Early changes in response to trauma were characterized by reduction in functional blood capillary density, increased extravasation index and maintenance of lymphatic vessel continuity with increased lymph flow rate (3). The EM pictures are still under evaluation. In another

study on dogs, we investigated factors presumably responsible for development of edema. We found dilatation of afferent lymphatics and enlargement of lymph nodes after subcutaneous injection of bone marrow cells (extravasates from fractured bone) (4). Finally, in lymphoscintigraphic studies of the lymphatic system of lower limbs in patients with fracture of tibia, joint dislocation and direct external trauma to calf muscles, we observed dilatation of lymphatics, sluggish lymph flow and enlargement of inguinal lymph nodes. No case with posttraumatic discontinuity of lymphatics was seen (1). Interestingly, these changes were seen as long as local inflammatory changes (edema, tenderness) persisted, that is for months. Taken together, these observations suggest that lymphatics are resistant to mechanical trauma. The initial lymphatics may avoid the traumatic discharge of kinetic energy by being empty (most initial and collecting lymphatics contain only small volumes of fluid in some segments – lymphangions, others remain empty) and cushioned by the mechanically resistant skin fiber structures and blood vessels filled with blood. Larger lymphatics are surrounded by fat tissue and stretchable, as is seen during surgery. Moreover, lymphatics can stand intraluminal pressures as high as 300-400 mmHg, observed in our own and others' studies (5,6). The question arises: had the lymphatics been severed and became non-conducting in a limb with hundreds of damaged small veins, blood extravasation, thrombosis, and venous stasis with subsequently increased tissue fluid and lymph formation, would that be compatible with tissue survival? Certainly not.

Lymphatics act here as a safety valve draining the excess of everything that has accumulated in the tissue space. They most likely developed the mechanical resistance to mechanical forces during evolution.

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Waldemar L. Olszewski, MD
Grzegorz Szczesny
Dept. of Surgical Research
& Transplantology,
Medical Research Center
Polish Academy of Sciences
02 116 Warsaw
Pawinski Str.5, Poland
e-mail: wlo@cmdik.pan.pl