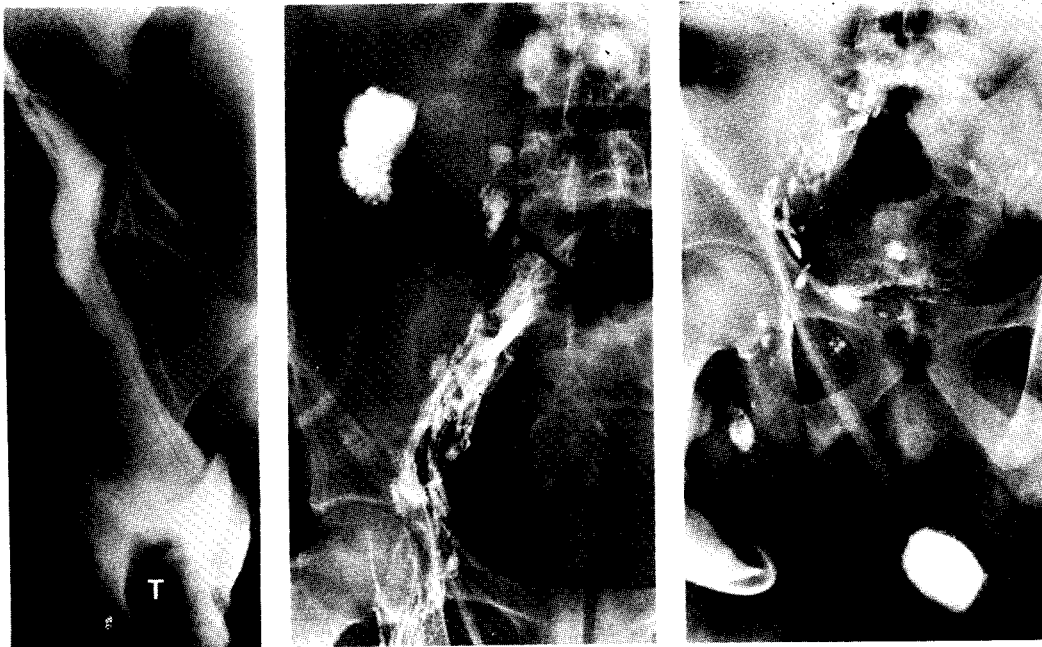


## LYMPHOGRAPHIA

**SCROTAL LYMPHOCELE COMPLICATING  
RENAL TRANSPLANTATION****J. V. Kaude and R. A. Moore**

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*Fig. 1: Fistulogram (left) demonstrating communication with the scrotum and surrounding the testicle (T). After injection of Ethiodol into lymphatics of the right foot there is extravasation of contrast medium from the common iliac lymphatics (small arrow) with localized accumulation (large arrows) (middle). Eight hours later (right) contrast medium has puddled in the pelvis and scrotum (arrows).*

**CASE HISTORY**

A 37-year-old man received a cadaveric kidney transplant on 27 October, 1983. There was no sign of rejection although edema was noted in the pelvis during the postoperative period. Two months later bilateral scrotal swelling developed and insertion of a Jackson-Pratt drain yielded chylous fluid. Because of continued output of approximately 300 ml of fluid a day the patient underwent a fistulogram and dorsal pedal lymphangiography which disclosed a large pelvic-scrotal lymphocele (see Fig. 1). The patient thereafter underwent re-exploration through the original incision and was found to have approximately one liter of chylous fluid about the transplanted kidney. The lymphocele extended via the internal inguinal ring into the scrotum. The defect in the internal inguinal ring was closed and the lymphocele was internally drained by marsupialization into the peritoneal cavity. The patient was discharged without reaccumulating fluid.

## COMMENT

After renal transplantation, lymphocele may enlarge to considerable size and cause edema of the lower extremity by venous compression (1-3). In this patient, the chief clinical manifestation was scrotal swelling from direct extension of a pelvic lymphocele. Although ultrasonography facilitates determination of presence and size of post-transplantation lymphocele, lymphography further delineates the site and extent of extravasation and lymphatic damage (4).

## CORRESPONDENCE

Based on our clinical experience, I am doubtful of the explanation of Stewart et al (LYMPHOLOGY 17, 23: 1984) for fibrogenesis in chronic lymphedema. According to these authors, disturbed fibrinolysis is the *primum novens* for pachydermia (dermatosclerosis) in lymphedema rather than chronic inflammation from stagnated (altered) plasma proteins in the interstitium. The problem is of more than theoretical interest, because Stewart and his colleagues advocate administration of anabolic steroids to ameliorate the condition. But, by complex physical decongestive therapy only, without drug treatment, we have been able also to improve advanced trophic changes in lymphedematous skin (Fig. 1). This observation tends to contradict the theory proposed by Stewart et al and favors more the "protein-stagnation" concept. It would, of course, be interesting to determine whether fibrinolytic activity (presumably decreased before physiotherapy) increases after manual decongestion alone.

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Fig. 1: Amelioration of advanced lymphedema of the right leg by physical manipulation. The only operation was excision of flaccid, redundant skin (Dr. Leo Clodius) after reduction of edema.

*Editor's Comment:*  
Aside from theoretical implications regarding pathogenesis of fibrinolysis, the remarkable improvement in this grotesque extremity almost entirely by manual manipulation borders on the miraculous. CLW.