Fifty-one PTFE grafts (GORETEXR, IMPRAXR), 5 bovine grafts, 5 human umbilical cords and 35 homologous veins were implanted in 91 patients. Narrowing at the venous anastomoses, infection of and around the grafts, septicaemia, and secondary thrombosis were the main reasons for operative revisions during which specimens of the implants were taken for microscopic examination.

The following observations were made: the intimal lining was consistently smooth and homogeneous in grafts of biological origin. The two brands of PTFE grafts reacted differently. The structure of the IMPRAXR graft allowed free ingrowth of connective tissue cells leading to a cellular neointimal lining. With GORETEXR, however, the ingrowth of fibroblasts was less pronounced, yielding a non-homogeneous and almost cell-free pseudointima. A cellular intima was found only at the sites of the anastomoses.

In our experience these differences have an influence on complications resulting from puncturing, occlusion rate, and infection.

GORETEXR grafts are, as a rule, more resistant to puncture. The force of the needle insertion can lead to boring and destruction of the rear wall of the graft.

The tendency of the puncture holes to retract was also greatly dependant on the PTFE structure: the ingrowth of fibroblasts led to better ‘healing’ in IMPRAXR grafts. Repeated puncture of the same fistula area should be avoided since it leads to sequestration and intraluminal dislocation of the graft material (Figure 1a).

Improper puncture technique, particularly non-oblique insertion of the needle and the use of fistula needles which core, may even lead to implantation of an epidermal core. Figure 1b demonstrates a viable epidermal sequester with dermal papillae punched into the lumen of a PTFE graft.

This graft had to be removed because the patient suffered from recurrent septicaemia due to staphylococcus aureus. Since the patient was asymptomatic after removal of the graft, we assume that septicaemia originated from contaminated skin punched into the fistula.
Figure 1a. Sequestration and intraluminal dislocation of graft material.

Figure 1b. Epidermal sequester (arrow) punched into the Lumen of a PTFE graft.

An additional interesting histological finding was the calcification of the inner portion of a PTFE graft. The majority of revisions was necessitated by narrowing at the venous anastomosis due to massive intimal hyperplasia and fibrosis which finally caused complete outflow obstruction (Figure 2a and 2b).
Figure 2a. Massive intimal hyperplasia and fibrosis at the venous anastomosis.

Figure 2b. Outflow obstruction due to intimal hyperplasia at the venous anastomosis (arrow).

From our histological studies we draw the following conclusions: in the event of infection, biological grafts will be destroyed by a productive inflammatory process involving the complete prosthesis which therefore must be removed. In prosthetic grafts however, inflammation is mainly restricted to periprosthetic tissue as fibroblast ingrowth into the PTFE itself is limited. Infected allografts can be saved by proper surgical drainage, antibiotic treat-
ment, and/or partial resection and by-passing. After local skin disinfection, choice of fistula needles and puncture technique should be carefully considered to avoid bacterial contamination of the fistula, boring of skin and implant, and traumatizing the fistula's rear wall. In this context a special fistula needle has proven advantageous [1]. In case of venous outflow obstruction secondary to hyperplasia and fibrosis at the venous anastomosis, re-anastomosis rather than balloon disobliteration is indicated.

References

1 Magasi, J, Asbach, HW, Möhring, K and Schuler, HW (1973) Dialysis, Transplantation, Nephrology, 10, 553