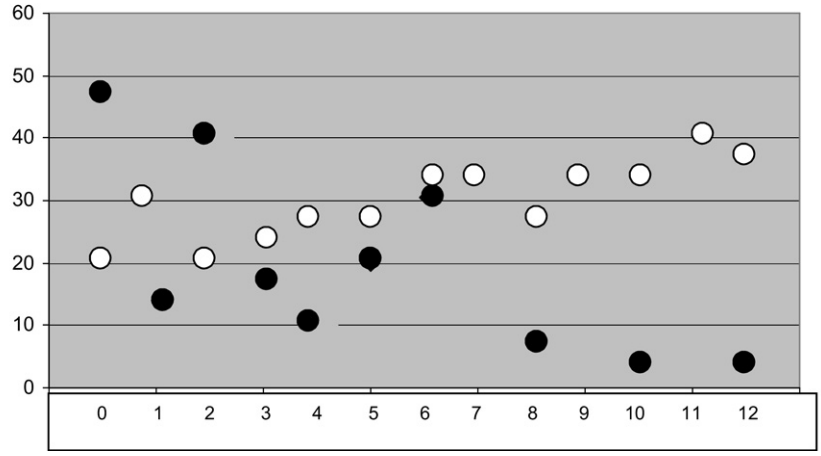

Figure 1. Random fecal A1AT levels in milligrams per gram dry weight (open circles) and serum albumin concentration in milligrams per liter (filled circles) within 12 weeks after heart transplantation (surgery at Time 0).

tion and had a predictably difficult post-operative course with moderate right ventricular dysfunction and oliguric renal failure. Dialysis was commenced on Day 4 and continued until Day 12 post-transplant. He required low-dose inotropic therapy for 6 days until right ventricular function recovery was noted at Day 9 post-transplant. He continued to have weekly albumin infusions until 6 weeks post-transplant with gradual improvement in serum albumin level and a corresponding gradual decrease in stool A1AT (Figure 1). At 12 weeks post-transplant his serum creatinine was stable at 139 μmol/liter, albumin level was 34 g/liter, fecal A1AT was 3.6 mg/g, and he was clinically euvolemic.

There have been no reports on reversal of PLE after heart transplantation in adult patients with cardiomyopathy and end-stage heart failure. In particular, reports on patients with right ventricular (RV) cardiomyopathy, who appear to be particularly at risk of developing PLE, have been limited. Available literature contains only one report of a fatal case of RV cardiomyopathy with severe PLE and congenital anomaly of the lymph ducts.[3] Our case illustrates that PLE, which occasionally complicates an end-stage cardiomyopathy, need not be considered a contraindication to transplantation. Given the existence of multiple causes of PLE, a careful pre-transplant evaluation that excludes the known extracardiac causes of PLE is warranted.

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THE CHALLENGE OF HOMOGRAFT TISSUE BANKS: THE VIABILITY OF CRYOPRESERVED VALVULAR HOMOGRAFTS

To the Editor:

The duration of the cryopreserved valvular homograft remains its main limitation, as the mechanisms of degeneration are largely unknown. The viability of the homograft is considered to have an important role in this issue, but it is not clear whether it leads to earlier degeneration or prolongs valvular life.

Since the 1980s, the rate of vitality of the valve after cryopreservation has been linked directly to better outcomes in long-term follow-up.[1] Valvular cells are responsible for valve renewal, even if all their functions are not completely revealed, and guarantee a continuous high turnover of the extracellular matrix components, such as collagen and glycosaminoglycans. Hence, their preservation after cryopreservation had been one of the goals of tissue banks and every effort has been

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made to guarantee their viability. Histologic analyses of homografts after degeneration have shown that donor cells remain present inside the leaflets at a higher percentage.[2] This tissue chimerism is not constant and, in our experience, has been shown to vary from 50% to 100%. Donor cells not only survive even years after implantation, but there may also be a sub-population with a capacity to proliferate. Even the early failure of decellularized allografts seems to confirm the role of vital cells in maintaining valvular homeostasis.[3]

Nevertheless, homograft implantation is performed without considering HLA matching and no immunosuppressive treatment is initiated. Viable homografts have been demonstrated to elicit both a specific cellular and humoral immune response against HLA determinants of the donor.[4] It is a sub-clinical long-lasting response that leads to early and late valvular degeneration.

One of the main aims of research in homograft tissue banking should be to understand the role of viability on both duration and degeneration of valvular allografts. Several aspects should be highlighted. Presently we do not know if there is an optimal percentage of viable cells that would permit longer duration and lower degeneration. The viability of the aortic valve is still unknown and there is no consensus, even with cellular types of aortic valves and their functions. Our preliminary evaluations have shown that donor cells can replicate after cryopreservation[5] and can maintain a low proliferation rate even years after implant. A more specific knowledge of homograft viability is the key to understand why the homograft degenerates earlier than the biologic prosthesis and how to evaluate new methods for prevention of degeneration. Moreover, it is the basis for research programs for bioengineering homografts, seeding decellularized scaffolds with patients' cells.

Homografts are transplanted organs that are incorrectly considered prostheses. Few data are available on the role of viability in allograft duration and degeneration. A complete understanding of the complex physiology and pathophysiology of homograft cells may lead to a renewed interest in use of human valvular allografts.

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