

Editorial

Single lung transplantation and pulmonary hypertension

There are few successful treatments for pulmonary hypertension. Long-term domiciliary oxygen therapy prolongs survival in patients with secondary pulmonary hypertension resulting from chronic hypoxic lung disease.¹ There is some evidence that the quality of life is also improved.² For primary pulmonary hypertension, which is unexplained by cardiac or pulmonary disease, no established medical treatment, other than anticoagulant therapy increases survival.³ Vasodilatation treatment can improve the quality of life⁴ and early evidence from the use of long-term intravenous infusion of prostacyclin in the most severe forms of the disease suggests that survival can be increased.⁵

Heart-lung transplantation has emerged as a successful treatment for a wide range of chronic end stage cardiopulmonary diseases, including pulmonary hypertension.⁶ It improves the survival of patients with cystic fibrosis and respiratory failure.⁷ In patients with pulmonary hypertension, heart-lung transplantation restores pulmonary vascular resistance to normal.⁸ However, the limited availability of donors with both lungs suitable for transplant surgery,⁹ impedes the wider use of this treatment. Indeed patients often wait for more than seven months and over a quarter die on the waiting list.

For some diseases, including lung fibrosis and emphysema, the development of single lung transplantation offers certain advantages over heart-lung transplantation.¹⁰ It requires only one suitable lung, which may theoretically increase the supply of donor organs. Because recipients retain their own hearts, they will not experience graft related coronary occlusive disease which limits the long-term success of cardiac transplantation.¹¹ Actuarial survival after single lung transplantation is approaching that after heart-lung transplantation⁹ so it is with special interest we learn of the haemodynamic effects of single lung transplantation on the treatment of pulmonary hypertension. Doig *et al* showed that haemodynamic function improved considerably after single lung transplantation for moderately severe pulmonary hypertension.¹²

This experience encourages the wider use of single lung transplantation perhaps limiting heart-lung transplantation to patients with cystic fibrosis. It also offers the interesting prospect of the use of single lobes of lung for

transplant surgery. This has already been described in three cases of childhood pulmonary hypertension by Vaughan Starnes in Stanford, California, and could lead to the use of organs from living donors for lung transplantation. The partial correction of the physiological abnormality would have to be sufficient to justify such a development. The debate on the ethics of live donation has recently been joined¹³ and there is an implicit need to balance risks to the donor against the benefits to the recipient. At this early stage when results are very limited, our judgement perhaps should be guarded. However, if the use of single lung transplants grew a further increase in donors would be possible, just as single organ transplants affected the prospects of renal transplantation.⁹

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