greater than say that of a patient with idiopathic pulmonary fibrosis in their 60s or 70s. In the end, however, the message is clear: respect idiopathic pulmonary hypertension or urine trouble.

See Article page 524.

Commentary: The perfidious left ventricle in pulmonary hypertension

Max K. H. Wong, MBBS, and
Michael K. Hsin, MD, FRCS CTh

Early postoperative left ventricle (LV) dysfunction following bilateral lung transplant (LTx) in patients with pulmonary arterial hypertension (PAH) is a well-recognized and complex challenge.¹ It has been shown to occur in up to one third of LTx recipients for PAH, causes early morbidity, and contributes to the development of primary graft dysfunction.^{2,3}

In this issue of the *Journal*, Benazzo and colleagues⁴ describe the Vienna experience, spanning 18 years with restrictive fluid management after double LTx in patients with idiopathic PAH, namely a liberal policy of early implementation of continuous renal-replacement therapy (CRRT) when a negative fluid balance could not be achieved despite maximal medical therapy. This is irrespective of levels of creatinine, blood urea nitrogen, or glomerular filtration rate. The authors concluded early CRRT did not impair long-term kidney function, and postoperative CRRT was not a risk factor for mortality.

In patients with pulmonary hypertension, the elevated pulmonary vascular resistance represents an increased afterload to the right ventricle (RV) and causes remodeling,

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Check for updates

Max K. H. Wong, MBBS, and Michael K. Hsin, MD, FRCS CTh

CENTRAL MESSAGE

Aggressive management of the volume status with renal-replacement therapy should be considered in the management of LV dysfunction after lung transplant in pulmonary arterial hypertension patients.

initially with hypertrophy and eventually dilatation. This results in a spherical-shaped RV, increased RV wall stress, impaired contractility, and progressive tricuspid regurgitation. Together, these further reduce the effective cardiac output of the RV.⁵

In patients with PAH, magnetic resonance imaging studies have shown reduced LV pump function. LV dysfunction in PAH is caused by (1) direct ventricular interaction, where the interventricular septum is bowing leftward, thus limiting LV diastolic filling; and (2) hemodynamic effects from a decrease in RV output, causing reduced LV filling. Human cardiac biopsy samples taken from heart/lung transplant recipients revealed that LV cardiomyocytes in patients with PAH have smaller cross-sectional area, indicating atrophy; reduced force-generating capacity, which is partially caused by the loss of major contractile protein myosin; and reduced phosphorylation levels of key sarcomeric proteins.⁶ Biopsy samples

From the Department of Cardiothoracic Surgery, Queen Mary Hospital, Hong Kong SAR, China.

Disclosures: The authors reported no conflicts of interest.

The *Journal* policy requires editors and reviewers to disclose conflicts of interest and to decline handling or reviewing manuscripts for which they may have a conflict of interest. The editors and reviewers of this article have no conflicts of interest.

Received for publication May 31, 2021; revisions received May 31, 2021; accepted for publication June 1, 2021; available ahead of print June 25, 2021.

Address for reprints: Michael K. Hsin, MD, FRCS CTh, Department of Cardiothoracic Surgery, Queen Mary Hospital, Room 308, New Clinical Building, Pok Fu Lam, Hong Kong, Hong Kong SAR, China (E-mail: mkhhsin@hotmail.com).

J Thorac Cardiovasc Surg 2022;163:537-8

0022-5223/\$36.00

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<https://doi.org/10.1016/j.jtcvs.2021.06.001>

from the same patients with PAH showed that RV cardiomyocytes, when compared with LV myocytes, demonstrate features of overloading.⁷ After LTx, because the lung graft has a much lower pulmonary vascular resistance than before, the LV preload is normalized, and the deconditioned LV is unable to cope with a sudden increase in LV filling and is at high risk of failure.⁸

In addition to impaired systolic LV function, patients with PAH also have impairment of LV diastolic function and LV relaxation.^{9,10} Restoration of LV preload to a stiff LV following allograft reperfusion may increase pulmonary venous pressure and exacerbate ischemia–reperfusion injury-induced noncardiogenic edema, and LV diastolic function contributes to the development of primary graft dysfunction. The LV appears to undergo reverse remodeling following restoration of preload over time, and abnormal filling may persist up to 1 year.¹¹

The Vienna group previously reported their strategy of prophylactic prolongation of VA extracorporeal membrane oxygenation in the early post-LTx period in patients with primary pulmonary hypertension to allow the deconditioned LV to adapt to new hemodynamics after LTx and demonstrated excellent survival outcomes.^{12,13} Together with this report on aggressive fluid restriction, the Vienna experience provides a useful roadmap in the management of this complex group after LTx.

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