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IMPACT OF NITRIC OXIDE INHALATION ON RIGHT VENTRICULAR FAILURE AFTER IMPLANTATION OF NOVACOR LEFT VENTRICULAR ASSIST SYSTEM

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The left ventricular assist system (LVAS) (Baxter Healthcare Corporation, Novacor Division, Oakland, Calif.) used as a bridge in the expectation of transplantation has been shown to effectively improve cardiac output even in patients with biventricular failure.¹ Nevertheless, many patients still require some sort of inotropic support and/or pulmonary vasodilator therapy to optimize right ventricular (RV) performance. Consequently, appropriate selection of potential recipients calls for careful evaluation of preimplantation pulmonary artery pressure and vascular resistance, which should remain in a suitable range to ensure successful heart transplantation. However, in certain cases, high pulmonary resistance necessitating concomitant RV support has been observed. We report here one such case in which an early rise in pulmonary artery pressure led to rapid RV failure, and the use of inhaled nitric oxide allowed us to forgo the need for mechanical RV support.

A 54-year-old man with dilated idiopathic cardiomyopathy received an LVAS because of repeated episodes of left ventricular failure necessitating increasing doses of sympathomimetic drugs. This patient was regarded eligible for heart transplantation. The preoperative pulmonary vascular resistance was 4 Wood units. After he was weaned from cardiopulmonary bypass (CPB), a rapid rise in mean pulmonary arterial pressure was observed from 40 to 65 mm Hg, associated with a decrease in RV ejection fraction from 17% to 6% and persistent low pump output below 3 L/min. This reduced pump output was related to difficulties in pump filling as witnessed by the reduction in LVAS peak filling volume, which precisely monitors this phenomenon. This change occurred despite the use of isoproterenol (0.1 µg/kg per minute) and epinephrine (0.2 µg/kg per minute) therapy, concomitant with administra-

tion of prostacyclin at an increasing rate of 5 to 20 ng/kg per minute. After an hour, in the absence of a satisfactory hemodynamic response, CPB was reinstituted. Nitric oxide was then administered as a specific pulmonary vasodilator therapy in a premixed concentration of 900 ppm nitric oxide in nitrogen (CFPO, Paris, France). Nitric oxide was introduced into the breathing system circuit near the endotracheal tube via a separate flowmeter. The nitric oxide flow rate was adjusted to reach a concentration of 50 ppm. Almost 30 minutes later, a progressive increase in peak LVAS filling volume was noted. CPB was then progressively interrupted. At the same time, LVAS pump output rapidly increased to 5 L/min, RV function improved (ejection fraction 17%), and mean pulmonary arterial pressure dropped to 35 mm Hg. The day after, mean pulmonary arterial pressure further decreased to 27 mm Hg while RV ejection fraction rose to 23%. Inotropic support and nitric oxide inhalation were then progressively decreased and the patient was extubated after 5 days.

This observation underscores the possibility afforded by nitric oxide inhalation in the reduction of acutely increased pulmonary vascular resistance after CPB during LVAS implantation.^{2,3} In our opinion, its feasibility should be considered before any decision is made to institute RV support in cases of early inadequate LVAS system filling.

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J THORAC CARDIOVASC SURG 1995;109:1251

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