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THE EFFECTS OF THE RATE OF REOXYGENATION ON THE RECOVERY OF HYPOXEMIC HEARTS

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The myocardial damage induced by the "reoxygenation phenomenon" of hypoxic hearts is well known from both experimental¹⁻⁴ and clinical⁵ reports. The surgical repair of cyanotic congenital heart defects in infants may be complicated by the problem of acute reoxygenation at the beginning of cardiopulmonary bypass (CPB).⁵ In fact, at the onset of CPB the whole body and, therefore, the myocardium are suddenly perfused from the arterial line with an oxygen saturation and an oxygen tension (PO₂) (generally >200 mm Hg) substantially higher than the baseline value because of the underlying cyanotic congenital heart defect. The main responsibility for the heart injury has been clinically associated with the burst of oxygen-derived free radicals when cyanotic hearts are suddenly exposed to high oxygen pressures.⁵

To test the hypothesis that the rate of reoxygenation may be critical in the observed pattern, we performed an experimental study in which isolated rat hearts were exposed to acute hypoxia and then underwent either "fast" or "slow" reoxygenation: the recovery of ventricular performance was then compared with the mode of the reoxygenation. Isolated rat hearts, perfused with Krebs-Henseleit buffer with a coronary flow of 15 ml/min,³ after a period of stabilization with 100% oxygen saturation, were exposed to 20 minutes of hypoxic perfusion (coronary flow of 15 ml/min, oxygen saturation 10% of baseline). Reoxygenation was either "fast" (PO₂ increase >200 mm Hg/sec) or "slow" (PO₂ increase 2 mm Hg/sec).

The results (Table I) indicate that both the systolic and diastolic ventricular functions were more depressed after "fast" reoxygenation than after "slow" reoxygenation. Although it is difficult to transfer results gathered in an isolated heart preparation to an in vivo situation, from this

Table I

	Fast	<i>P</i> Value	Slow
<i>n</i>	5		5
Rate of reoxygenation (mm Hg/sec)	200		2
Recovery (% of baseline)			
Developed pressure	46 ± 7	0.1	62 ± 5
+dP/dt	48 ± 5	0.005	70 ± 3
-dP/dt	48 ± 10	NS	61 ± 4
Oxygen uptake	57 ± 8	NS	68 ± 9
Change over baseline			
End-diastolic pressure (mm Hg)	40 ± 9	0.09	22 ± 3
Coronary pressure (mm Hg)	37 ± 18	NS	28 ± 7

dP/dt, Rate of pressure rise; NS, not significant.

experimental study it appears that (1) gradual reoxygenation after hypoxia may contribute to the reduction of myocardial injury in infants with cyanosis who undergo cardiac repair with the use of CPB; (2) the persisting myocardial dysfunction after reoxygenation underlines the presence of factors responsible for the reoxygenation damage other than oxygen free radicals³; (3) the bulk of the injury may be associated with events that occur not only during the reoxygenation, but most likely also during hypoxia, such as the high energy demand⁴; and (4) other experimental and clinical studies are required to learn how to prevent or reduce the reoxygenation phenomenon.

We are already aware of other centers of pediatric cardiac surgical care in which CPB in infants with cyanosis is routinely initiated with an arterial PO₂ (80 to 100 mm Hg) lower than that generally used and maintained at a relatively lower value (maximum 120 mm Hg) than that conventionally used (Pedro Del Nido, personal communication, 1994).

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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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