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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 experimental[1-4] and clinical[5] 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 (Po_2) (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" (Po_2 increase >200 mm Hg/sec) or "slow" (Po_2 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 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 radicals[3]; (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 demand[4]; 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 Po_2 (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).

Table I

	Fast	*p Value*	*Slow*
n	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.

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