Letter to the Editor

Recovery of hypoxic neonatal hearts after cardioplegic arrest

Sir:—With regard to the article by Feldbaum et al.¹ and Dr Baker’s comments,² we believe that a few critical points were missed.

First, in the authors’ protocol, hearts from hypoxic animals (presumed P<sub>O₂</sub> < 4.16 kPa, 32 mm Hg) were acutely exposed to a medium containing 95% O<sub>2</sub> (P<sub>O₂</sub> = 89 kPa, 686 mm Hg) at the onset of the in vitro perfusion. This may have led to significant reoxygenation injury before baseline evaluation. This possibility, consistent with Dr Baker’s consideration that these preparations were functionally compromised at baseline evaluation,³ may considerably weaken the authors’ conclusion that hypoxic immature rabbit hearts can recover from an ischaemic insult as well as matched controls.

Furthermore, the cited references do not support the authors’ view that “cyanotic infants recover as well as non-cyanotic infants after heart surgery”. For instance, Rizzoli et al.⁴ and Sunderland et al.⁵ consider cyanotic patients only, while Jonas et al.⁶ and Friedli et al.⁷ make no comparisons between cyanotic and non-cyanotic groups. Therefore, the general consensus now is that injury in cyanotic hearts is more severe than in non-cyanotic hearts.⁸

We believe that other crucial data should be made available to allow a better understanding of importance of the authors’ work:

(1) Blood gases in the living animals, because the calculation of P<sub>O₂</sub> from F<sub>O₂</sub> critically depends on ventilation, alkalis, and acidosis. Assessing if animals were adapted to hypoxia is critical because acidosis implies alterations of plasma (and thus intracellular) pH⁹,¹⁰ and of bioenergetic patterns.¹¹,¹²

(2) P<sub>O₂</sub> and/or lactate output, to evaluate the bioenergetic pattern and to understand whether different degrees of anaerobic metabolism have protected against ischaemic reperfusion, especially in view of the different ages of the animals and the known age related differences of myocardial metabolism.¹³,¹⁴

(3) Some evaluation of the diastolic function, as for example the pressure-volume curve, to understand postischaemic ventricular dysfunction in control and hypoxemic/reoxygenated hearts.¹⁵

Finally, the Authors have reported that “some hearts no longer functioned at the postischaemic measurement time”, and that they “analysed values of 0 for all variables in such hearts”. Thus a quantitative variable (number of deaths) was transformed into a qualitative one (value = 0). We wonder whether statistical evaluation may have been severely compromised since n ranged from 6 to 11 in the various groups.

MICHELE SAMAJA
Department of Biomedical Science and Technology, University of Milan, Italy

ANTONIO CORNO
Hospital San Donato, Milan, Italy