Reanimation and engraftment of the asystolic primate heart

To the Editor:

Dr. Gundry and colleagues¹ present convincing evidence regarding successful reanimation and engraftment of the asystolic primate heart.

Several studies have examined the metabolic and functional compromise after varying periods of warm ischemia. In a study of isolated perfused rat hearts, the left ventricular developed pressure after 20 and 45 minutes of ischemia was 75% and 46% of preischemic values, respectively.² In another study, Carr and associates³ measured age-related responses of rabbit hearts to normothermic ischemia using ³¹P nuclear magnetic resonance techniques. Parameters of functional recovery were similar to ones in the previous study. However, they demonstrated increased tolerance of the neonatal heart and increased susceptibility of the immature heart to unprotected normothermic ischemic injury relative to the adult hearts. They suggested maturational changes in regulation of myocardial intracellular pH for the observed age-related responses.

It is clear that the excellent results in this study stem from the resuscitative effects of controlled reperfusion. Amino acid supplementation of blood cardioplegic solution results in complete recovery of the immature myocardium.⁴ Reperfusion with leukocyte-depleted blood for 10 minutes and enriched blood cardioplegic solution for the first 3 minutes of reperfusion after prolonged hypothermic ischemia resulted in shorter duration of inotropic support and decreased biochemical evidence of ischemic injury.⁵ We⁶ have shown in an isolated rat heart model with ³¹P nuclear magnetic resonance that reperfusion with warm blood cardioplegic solution for 20 minutes after an ischemic interval associated with an undetectable pool of adenosine triphosphate resulted in an $81\% \pm 9\%$ recovery of rate of pressure rise (over preischemic values) versus $48\% \pm 9\%$ without warm blood cardioplegia. The recovery rates of adenosine triphosphate and creatine phosphate pools were also significantly faster.

The molecular and metabolic mechanisms of reanimation are yet to be defined. The precise role played by hypothermic preservation, reperfusion techniques, and state of the vascular bed in the immediate postoperative period and in long-term preservation of the graft needs to be elucidated by performing experimental studies with controlled extracardiac variables that influence postischemic cardiac function.

*Mukul Chandra, MD
Department of Medicine
Emory University
Atlanta, GA 30303*

REFERENCES

1. Gundry SR, Fukushima N, Eke CC, Hill AC, Zuppan C, Bailey LL. Successful survival of primates receiving transplantation with "dead" nonbeating donor hearts. *J THORAC CARDIOVASC SURG* 1995;109:1097-102.
2. Bolling SF, Olzanski DA, Childs KF, Gallagher KP, Ning X. Stunning, preconditioning and functional recovery after global myocardial ischemia. *Ann Thorac Surg* 1994;58:822-7.
3. Carr LJ, Vanderwerf QM, Anderson SE, Kost GJ. Age-related response of rabbit heart to normothermic ischemia: a ³¹P-MRS study. *Am J Physiol* 1992;262:H391-8.
4. Pearl JM, Hiramoto J, Laks H, Drinkwater DC, Chang PA. Fumarate-enriched blood cardioplegia results in complete functional recovery of the immature myocardium. *Ann Thorac Surg* 1994;57:1636-41.
5. Pearl J, Drinkwater DC, Laks H, Capouya ER, Gates RN. Leukocyte-depleted reperfusion of transplanted human hearts: a randomized, double-blind clinical trial. *J Heart Lung Transplant*. 1992;11:1082-92.
6. Przybylski R, Olivson A, Chandra M et al. Resuscitation of the ischemic heart by warm blood cardioplegia. *J Mol Cell Cardiol* 1993;25(Suppl 1):VI p4.

12/8/70536

[RESPONSE DECLINED]