Heart and soul

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As cardiac surgeons, our primary goal is to make certain that the body of our patient remains connected to his or her soul. In their article in this issue of the *Journal*, Paparella and colleagues¹ propose the not-too-surprising hypothesis that increasing perioperative cardiomyocyte damage increases the likelihood of our patient's soul departing his or her body. The image of a completely innocuous myocardial preservation strategy has, like a mirage, haunted cardiac surgeons for decades.

Paparella and colleagues¹ serially measured cardiac troponin I (cTnI) in 965 patients after mitral and aortic valve surgery. They found higher cTnI after mitral valve surgery than after aortic valve surgery, and they concluded that inclusion of postoperative cTnI in a reference model that permitted stratification for New York Heart Association functional class, left ventricular ejection fraction, and duration of cardiopulmonary bypass improved the model's statistical relationship with postoperative morbidity and mortality at 3 months. Paparella and colleagues¹ refer to this increased cTnI as a "risk" and postulate that during myocardial revascularization procedures (such as coronary artery bypass grafting), "coronary disease extension, recent myocardial infarction, and incomplete revascularization" may lead to myocardial damage, whereas in valve surgery, the efficacy of cardioplegia is just as likely to be related to cellular necrosis. If indeed cTnI is elaborated when cardiomyocytes succumb and spill out this intracellular enzyme, then all cardiac surgery provokes at least some bump in cTnI when oxygen and substrate delivery does not meet oxygen and substrate demand. For aortic valve surgery, there is extraordinarily limited direct incisional damage to the myocardium; in mitral valve surgery, the atrial incision is unlikely to release much cTnI. A hypertrophic left ventricle as a result of aortic stenosis may predispose toward myocardial

ischemia, and a dilated left ventricle as a result of mitral insufficiency may also exacerbate the ventricular "tension," driving up oxygen demand by the rule of Laplace. Thus it seems that analysis of any increase in postoperative cTnI must always focus on the adequacy of cardioplegia delivery as an etiology.

Unfortunately, Paparella and colleagues¹ were not able to detect an association between cTnI increase and the type ($P = .26$), temperature ($P = .17$), or mode of delivery ($P = .32$) of the cardioplegic solution. Importantly, however, they do shine a bright light on the disappointing fact that we have not yet achieved "optimal" myocardial preservation practice—especially during mitral valve surgery.

To date, cardiac surgeons have focused on driving down myocardial oxygen and substrate use after the application of the crossclamp. The Fullerton laboratory in Colorado^{2,3} has identified an interleukin 1–nuclear factor κ B–heat shock protein 27 inflammatory pathway that is provoked by crossclamp-induced ischemia and reperfusion. Cardiac surgeons should glory in the recognition that we already deserve a favorable grade for protecting cardiomyocytes during surgery. If we dream of truly aspiring to honors status, however, perhaps we should add an anti-inflammatory agent to our cardioplegic solution.

It would seem that a "risk" is avoidable only when you haven't taken it yet. Once cardiomyocytes are dead, and the cTnI is detected, the dead ones become a handicap, not a risk. The message of this important study¹ therefore is that to prevent our patients' souls from crossing through the pearly gates, we must not just target oxygen and substrate imbalance in our myocardial preservation strategies but also, perhaps, add a "pinch" of anti-inflammatory sauce to our cardioplegic stew.

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