LEFT VENTRICULAR DYSFUNCTION AFTER MITRAL VALVE REPAIR: PREDETERMINED OR CAUSED?

To the Editor:

Early mitral valve repair is being done to avoid the delayed left ventricular (LV) damage that happens when repair is performed in larger hearts, as described in the recent article by Quintana and colleagues.¹ Suri, one of the coauthors of the study of Quintana and colleagues, reported the Mayo Clinic experience showing that LV functional recovery is impaired if LV end-systolic diameter is greater than 36 mm.² The recent report of Quintana and colleagues¹ analyzed 1705 Mayo Clinic patients with LV ejection fraction (EF) greater than 60% and showed that at 4 postoperative days EF was less than 50% in 1 in 5 patients. LV impairment persisted, with EF returning to a value greater than 60% in only one-third of patients, versus two-thirds of those with a postoperative EF greater than 50%. Moreover patients with a postoperative EF less than 40% had an increased LV end-systolic diameter and a higher hazard of late death. The postoperative LV dysfunction title of Quintana and colleagues¹ addresses the fallacy of “normal” preoperative myocardial function.

The interpretation of Quintana and colleagues¹ is that this LV functional finding was predetermined, reflecting a myocardial predisposition toward

LV deterioration; they could not impugn surgical interventions, which were similar in the groups with and without dysfunction. An alternate hypothesis is that these changes were caused by the surgical intervention of myocardial protection, and that these adverse changes may have been preventable. Quintana and colleagues’ report¹ does not address the methods of protection, and it should.

The capacity of echocardiography to gauge LV end-systolic diameter to guide early intervention was beautifully defined by Suri,² but it also measures how our interventions affect LV size, shape and performance, the nidus of this report. Moreover, another marker of intraoperative damage is septal function, which accounts for half of the LV wall weight.³ Reynolds and associates⁴ studied almost 3300 postoperative patients with normal preoperative septal motion and showed that septum paradox (bulging) developed in about 40%; however, they did not report other accepted yardsticks of septal dysfunction, such as mild or severe hypokinesia or akinesia. Septal bulging was thought to be related to surgical ischemia duration.

In contrast, septal damage can be avoided by use of the integrated method of myocardial protection that includes cold antegrade blood cardioplegia, retrograde blood cardioplegia, and a warm reperfusate containing glutamate and aspartate.

The period of clamping in the study of Quintana and colleagues¹ was only 60 minutes, but our own study in 119 patients showed that septal damage was offset despite ischemia extending to 157 minutes.³ Our recent study addressed the septum by means of speckle tracking imaging and showed that septal damage followed only antegrade blood cardioplegia, whereas improved septal function followed the integrated approach⁵ that avoided adverse changes in radial, circumferential, and longitudinal strain.

Quintana and colleagues¹ should report septal changes.

Findings of LV damage after mitral repair may become problematic in the future, because studies are being planned to compare the mitral clip to surgical correction that addresses the entire mitral apparatus. Catheter-based technology would avoid myocardial damage to paradoxically favor its use, since magnetic resonance imaging studies show no LV dysfunction.⁶ Conversely, LV dysfunction implies problems in myocardial protection that are avoidable with the integrated method or any alternate technique that avoids septal injury.

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