Authors have nothing to disclose with regard to commercial support.

a rate of 17%. We believe that any concerns about calcification of a pericardial patch should be balanced against the high mortality associated with rerupture and the unlikelihood of such a patient requiring (or being a candidate for) a redo sternotomy. We also note a recent systematic review of left ventricular free wall rupture repair showing no difference in mortality between 96 patients repaired with a sutureless technique and 116 patients in whom sutures were used to close the ruptured myocardium or secure a patch on the epicardium.⁴

Both groups report the use of venoarterial extracorporeal membrane oxygenation both preoperatively and perioperatively in a proportion of patients. Cardiogenic shock as a result of acute myocardial infarction, even in the absence of a mechanical complication of acute myocardial infarction, has a poor prognosis.⁵ Venoarterial extracorporeal membrane oxygenation is an excellent short-term support for the patient in cardiogenic shock, allowing rapid restoration of end-organ perfusion. Its main limitations, however, are increased afterload and inability to unload the left ventricle.⁶ Increased afterload in the context of both left ventricular free wall rupture and acute myocardial infarction is unfavorable and should be avoided. Routine use of an intra-aortic balloon pump has been suggested for this purpose.⁷ We speculate whether a strategy of unloading the left ventricle after surgery with a left ventricular assist device, such as the Impella 5.0 (Abiomed, Danvers, Mass), inserted through the axillary artery may assist left ventricular recovery. Use of such a device as a bridge to surgery has been reported in the management of ischemic ventricular septal defect.⁸ We await the availability of some data to support such a strategy.

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**REPLY: OPTIMAL
MANAGEMENT
OF LEFT
VENTRICULAR FREE
WALL RUPTURE
CONSIDERING THE
PROBABILITY OF**

RERUPTURE

Reply to the Editor:

I read with great interest the letter to the Editor by Formica and colleagues regarding their excellent experience with the “patch and glue” technique and their comments regarding our study. The optimal surgical strategy for left ventricular free wall rupture (LVFWR) remains controversial because of the lack of large-scale studies describing this subject and the severity of the patients’ condition. Formica and colleagues¹ have described the application of the pericardial patch and glue with cardiopulmonary bypass and cardioplegic arrest or on a beating heart. The pericardial patch is sutured to the myocardium with polypropylene sutures. Combined applications of collagen sponges and subsequent fixation with an autologous pericardial patch more effectively reinforces the fragile infarcted myocardium and can reduce the incidence of rerupture of the repaired site. Notably, Formica and colleagues¹ have reported that the surgical revision rate for bleeding was as high as 25.7%. Their study included both the “patch-and-glue” technique and the direct suture techniques. Furthermore, in my view, patients undergoing surgical revision for bleeding might include those who presented with rerupture of the repaired site and those with bleeding from noncardiac areas, such as the sternum. I expect Formica and colleagues to report the outcomes of the patch-and-glue technique after studies

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involving a greater number of patients undergoing this treatment.

The technique of fixation of the autologous pericardial patch determines tissue durability and resistance to late degeneration and calcification after implantation. It is unclear whether a glutaraldehyde-treated autologous pericardial patch was used for the patch-and-glue technique. Notably, the autologous pericardial patch shown in Figure 1 in the letter to the Editor appears pliable and untreated. A few researchers have reported postoperative calcification and degradation of the autologous pericardial patch over time. Holubec and associates² reported that autologous pericardium or xenopericardium usually undergoes calcification and degradation with time. Ross and Olsen³ used untreated autologous pericardium for mitral valve repair and reported prominent degeneration with thickening and fibrotic changes in the harvested pericardial patches during redo surgery.³ Fukunaga and coworkers⁴ reported that glutaraldehyde-treated autologous pericardium was severely calcified 5 years after mitral valve repair.

I wish to highlight the following concerns regarding the routine use of autologous pericardium in patients undergoing surgery for LVFWR: (1) Autologous pericardium (glutaraldehyde treated or untreated) shows possible late calcification, with consequent diastolic dysfunction of the repaired myocardium. (2) Suture placement for fixation of the pericardial patch can cause tearing of the infarcted myocardium with subsequent bleeding. In cases of LVFWR after acute myocardial infarction, distinguishing between the infarcted and the intact myocardium is difficult and suturing the normal myocardium is challenging, particularly in cases of acute myocardial infarction associated with proximal occlusion of the coronary artery. (3) Harvesting a large part of the anterior pericardium is associated with significant drawbacks for future reoperations. Considering these concerns, I recommend that the patch-and-glue technique be reserved for patients with a high probability of rerupture, as reported in my group's study.⁵ This category of patients would include those with blowout type LVFWR, those with oozing of blood from multiple epicardial sites, and those with bulging of the infarcted myocardium and other such conditions.

I am in complete agreement with Formica and colleagues regarding the utility of extracorporeal membrane oxygenation (ECMO) as a valid and immediate tool for hemodynamic stabilization in patients with cardiac tamponade, cardiogenic shock, or cardiac arrest. The rate of ECMO use was lower in our series than in the series

FIGURE 1. Kaplan-Meier curves of overall survival with 95% confidence intervals among patients who survived surgery.

reported by Formica and colleagues¹ (11.4% vs 34.3%), which could be attributed to the fact that the rate of blowout type LVFWR was significantly lower in our cohort (6% vs 46%). Notably, rapid hemodynamic deterioration can occur in patients with blowout type LVFWR, necessitating the preoperative use of ECMO. This finding could also be attributed to the fact that their study perhaps included patients in whom ECMO therapy had already been initiated at the referring centers before being transferred for surgery. In our series, ECMO was initiated in all 4 patients who received this therapy, after arrival at our center.

Overall survival among patients who survived the initial surgery is shown in Figure 1. The 5-year survival was 82.8% in our study, comparable with 74.7% in the study by Formica and colleagues.¹

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