Letters to the Editor

A new technique for repair of mitral paravalvular leak?

To the Editor:

I read with interest a recent report of Mangi and Torchiana.[1] They reported successful repair of anterior mitral periprosthetic leaks after mitral valve replacement in 2 patients and made the following statement: "Here we propose a simple technique for repair of selected mitral paravalvular leaks that incorporates healthy, full-thickness autologous tissue into the repair and has promising durability."

This technique, however, is not novel. It seems appropriate to point out to Mangi and Torchiana that this technique had already been proposed 7 years ago, when my colleagues and I reported it.[2] It is amazing to see that the technique described by Mangi and Torchiana is exactly the same as the one that was first used by us more than 10 years ago (Figure 1, *A*).

Repair of periprosthetic leaks can indeed be very difficult and is often frustrating, as often there is a lack of strong tissue in the periprosthetic area to place direct sutures securely. For this reason, we decided to repair periprosthetic leaks with full-thickness autologous tissue. Further, and more importantly, we applied this con-

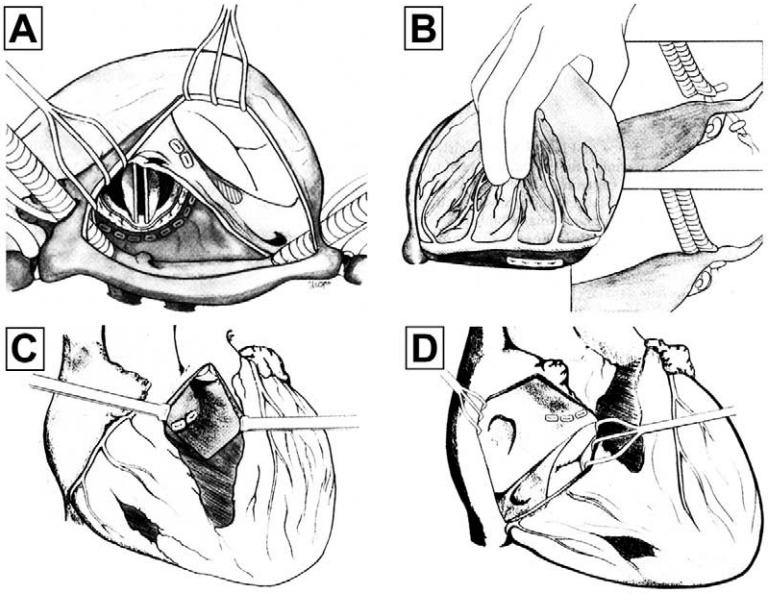

Figure 1. A, To close anterior mitral periprosthetic leak, sutures are applied through atrial septum adjacent to sewing ring of prosthesis. Ligament of Todaro and location of atrioventricular node are identified before suture placement. B, Posterior mitral periprosthetic leak is repaired by sutures passed through posterior left atrial wall. Care is taken to avoid damage to coronary sinus and left circumflex coronary artery. C, Aortic periprosthetic leak is repaired through incision in right ventricular outflow tract by passing sutures through ventricular septum. D, Aortic periprosthetic leak is repaired through right atrium. Distinct anatomic landmarks, such as triangle of Koch, membranous septum, and conus aorticus, are identified before suture placing. Sutures are then placed through right atrial wall and passed through lowest part of the conus aorticus above membranous septum. (Reproduced with permission from Konstantinov IE, Franzen SF, Olin CL. Periprosthetic Leaks and Valve Dehiscence: Alternative Methods of Repair. *J Heart Valve Dis.* 1997;6:281-7.)

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cept not only to anterior but also posterior mitral (Figure 1, *B*) and aortic (Figure 1, *C* and *D*) periprosthetic leaks.[2] During the last decade, we have maintained that these alternative methods of repair with healthy autologous tissues are superior to direct suture closure of the leak. It is gratifying to see that others have started to use this technique with good results.

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Reply to the Editor:

We appreciate Dr Konstantinov for bringing his report to our attention. A MEDLINE search on the keywords prosthetic valve and paravalvular leak yielded 119 articles since 1966 but did not include his reference from the *Journal of Heart Valve Disease.* We apologize for overlooking it.

In heart surgery, as in life, there is indeed nothing new under the sun.

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Acute decrease of left ventricular mechanical dyssynchrony and improvement of contractile state and energy efficiency after left ventricular restoration

To the Editor:

We congratulate Dr Schreuder and colleagues[1] on their study of intraoperative left ventricular (LV) performance after endoventricular patch aneurysmectomy. In this series of 9 consecutive patients, the study demonstrates significant reduction in end-diastolic volume and increase in ejection fraction and LV energy efficiency.

However, we would counsel caution in this interpretation due to what we believe are three inherent flaws in the study methods.

First, the hemodynamic data highlight a significant difference in the heart rate before and after aneurysmectomy. It is widely established that an increase in heart rate proportionately alters LV systolic function and ejection fraction by virtue of the force-frequency relationship.[2]

Second, 7 of 9 patients underwent coronary artery bypass grafting, which may contribute to the increase in LV performance by reversal of hibernation. The authors discuss the potential independent effects of bypass grafting, but not the critical issue of the contribution of hibernating myocardium to LV function. We suspect that this factor may be responsible for augmented LV systolic function and ejection fraction.[3]

Third, there is no information regarding the severity of mitral regurgitation. This can cause spurious recording of increased LV performance due to inappropriate offloading of the left ventricle.[4]

Notwithstanding these issues, we believe that this is a valuable study contributing required evidence to support objective improvement in LV function with reverse remodelling surgery.

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doi:10.1016/j.jtcvs.2005.02.043

Reply to the Editor:

We thank Dr Balacumaraswami and colleagues for their interest in our study on the acute effects of left ventricular (LV) restoration on cardiac performance. We prefer to answer in reverse order.

The reason for not giving any information on mitral regurgitation (MR) was the absence of significant perioperative MR in our patient group, all of whom underwent routine echocardiographic examinations. Concerning MR, McCarthy[1] referred to Di Donato and colleagues,[2] revealing that 10% of their patients had preoperative MR and 38% of their patients had had MR develop by 1 year after LV restoration, whereas immediately after surgery the patients were free from MR. When present, MR may offload the left ventricle; however, offloading will not change contractile state. Recently we[3] demonstrated that acute decrease in LV afterload by intra-aortic balloon pumping resulted in instantaneous increases in stroke volume but not in an increase of contractile state.

The second comment suggested that a significant improvement of LV systolic function and ejection fraction may have been attributable to recovery of hibernating myocardium in patients with additional coronary artery bypass grafting. Bax and colleagues[4] demonstrated in patients with ischemic cardiomyopathy undergoing CABG a reduction in wall motion abnormalities, based on hibernation, in 30% of the segments 3 months after bypass grafting. This suggests that in our acute LV restoration study, recovery from hibernation might have occurred; however, its contribution to an increase in contractile state could be doubted. Moreover, LV contractile state is commonly impaired or unchanged immediately after cardiopulmonary bypass.[5,6] In Table 3 of the LV restoration manuscript, we showed that LV mechanical dyssynchrony decreased significantly in the apical and midventricular segments, the areas of the LV restoration, whereas the dyssynchrony of the basal segments was unchanged.[7] The major finding of our study, the marked relationship between LV mechanical dyssynchrony and contractile state of the heart, however, is