example, shocks may contribute to myocardial injury, and backup ventricular pacing may impair left ventricular function. An alternative strategy would be to address the substrate for arrhythmias directly during SVR. Dor and colleagues⁴ reported excellent results after nonguided subtotal endocardectomy and cryoablations in combination with SVR in 1994. Since then, other centers have performed various techniques for aneurysm repair, including cryoablation or endocardial resection with a low incidence of postoperative VT,^{5,6} and some centers used intraoperative mapping.⁵ Therefore, it was somewhat surprising that Babokin and colleagues¹ stated that they did not find any studies that used a similar approach to theirs regarding VT treatment in patients with post-infarction changes in the heart. Moreover, they stated that known surgical approaches in this patient group are incomplete and leave VT sources in the heart, and they supported this statement by referencing the Surgical Treatment for Ischemic Heart Failure trial (hypothesis 2)⁷ and the study by Harken and colleagues⁸ in 1980. The Surgical Treatment for Ischemic Heart Failure trial did not investigate VT procedures or results, and the article by Harken and colleagues was published more than a decade before Dor and colleagues⁴ presented their comprehensive strategy in this patient group. Last, the statement that the use of radiofrequency ablation-induced markings prevented excessive myocardial resection and therefore reduced complications is speculative and does not have support in the data.

In addition to the previous comments, some questions arise when reading the article by Babokin and colleagues.¹ What was the detailed stimulation protocol during the electrophysiologic study, and what was the definition of VT inducibility? These are important questions because an aggressive stimulation protocol will result in more patients with inducible VT and vice versa.

Did the surgical procedure include some sort of ablation or only

endocardectomy? Only 12 patients received an ICD postoperatively. How many patients had an ICD before surgery? What were the indications for ICD treatment at the authors' institution during the study period? Because ICD use was an end point in the study,⁴ it is valuable for the reader to know whether the indications for ICD use were liberal or restrictive.

The authors are to be commended for their effort in improving a surgical procedure and for demonstrating good results in complicated cases. There is support in the literature for a preventive effect of endocardectomy on recurrence of VT. However, whether guided endocardectomy is better than nonguided endocardectomy during SVR is still an open question.

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Reply to the Editor:

Our study began with making a decision to refer patients with left ventricular aneurysm for a preoperative electrophysiologic study (EPS) with CARTO (Biosense Webster, Diamond Bar, Calif) mapping. During one of our first EPS cases, a stable ventricular tachycardia (VT) was induced in the myocardial area with the reduced potential (0.5 to 1.5 mV). This VT was abolished by radiofrequency ablation (RFA) by using the ablation electrode at power output of 45 W for 40 to 50 seconds with saline irrigation of 12 mL/min. Later, during heart surgery that involved endocardectomy, a surgeon noticed a myocardial lesion at the scarred area of the interventricular septum in that patient. The lesion, which had resulted from the RFA performed for VT treatment 5 days previously, macroscopically looked like a 3- to 7-day-old myocardial infarction. That case was a turning point in our decision to use RFA during EPS to produce visible markings of the electrophysiologically abnormal substrates to facilitate guided endocardectomy. Our article provided description of the results of the endocardectomy along RFA-induced markings. We did not compare our results with the results of either unguided subtotal endocardectomy¹ or guided subendocardial resection.² The purpose of our study was specifically to provide evidence of benefits of endocardectomy for VT treatment in patients with postinfarction left ventricular aneurysm when more advanced methods of preoperative examination, such as EPS with electroanatomic left ventricular CARTO reconstruction, were performed.

In answer to the question of Dr Sartipy about automatic implantable

cardioverter defibrillator placement, in the postoperative period, 11 patients from the group of surgical ventricular restoration (SVR) without endocardectomy received automatic implantable cardioverter defibrillators because of spontaneous VT (5 cases) or EPS-induced stable VT (7 cases). Only 1 patient in the group of SVR with endocardectomy had indications for and received an automatic implantable cardioverter defibrillator because of EPS-induced VT in the postoperative period. The EPS protocol was as follows: right ventricular stimulation was performed from the apical and septal positions; programed stimulation continued until frequency reached 220 to 230 impulses per minute; and the programed stimulation protocol included single, double, and triple right ventricular extrastimuli, burst stimulation with 10 stimuli with decremental decrease by 10 ms, and ramp pacing (cardiac pacing in which stimuli are delivered at a rapid but continually altering rate) with 5 to 10 stimuli. The result of EPS were considered as positive when the stimulation resulted in induction of at least 2 events of VT consisting of at least 10 monomorphic ventricular extrasystoles. We diagnosed stable EPS-induced VT when the electrical stimulation resulted in VT lasting for at least 30 seconds.

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PARADOXICAL STITCHES AIM TO DISCIPLINE THE ANTERIOR LEAFLET TO AVOID POSTPLASTY SYSTOLIC ANTERIOR MOTION

To the Editor:

I read with interest the article by Varghese and colleagues.¹ The systolic anterior motion (SAM) associated with

mitral valve repair is related to many factors, both anatomic and technical. The latter could be eliminated, or at least reduced, by a good repair technique. The anatomic factors are considered a hard challenge. The techniques for the treatment or prevention of SAM can be divided into 2 main strategies: resect versus respect rather

FIGURE 1. A, Illustration represents intraoperative transesophageal echocardiographic imaging in the longitudinal axis: *a-b* line indicates the level between the anterior annulus and the tip of the papillary muscle during the late diastole; *c-d* is the distance between the highest point of the interventricular septum and the previous level. The position of the papillary muscle determines the distance *c-d*. B, Illustration represents the A2 of the anterior leaflet. The risk triangle (*abd*) is determined by the borders of A2 on the margin of anterior leaflet and its tip. An increase in the height of this triangle (*cd*) causes an increase in the risk of postoperative systolic anterior motion. The presence of myxomatous tissue in the *abd* triangle is an additional risk factor for systolic anterior motion.