important “delta” was recorded in that study in terms of systemic arterial pressure and dp/dt_{MAX} (variables not collected in our study and not considered as end points of the trial), but a less important “delta” (although statistically significant) was recorded for cardiac index and cardiac cycle efficiency (magnitude ~ 0.24 L/min for cardiac index and 0.018 units for cardiac cycle efficiency, respectively).³ However, these “deltas” were registered in an animal model during systemic hypertensive conditions (mean values of systolic blood pressure ranging from 160 to 174 mm Hg),³ a hemodynamic pattern distant from that of a vasodilated patient in stable hemodynamic condition, as in our hemodynamically oriented IABP-weaning trial. We strongly believe that the magnitude of these “deltas” in patients with an overall good hemodynamic performance, especially when confirmed by other hemodynamic, biochemical, and clinical data, should be considered negligible from a clinical perspective.

Finally, we assume that the measurement of any biological process is truly a difficult task, and that the trend over time of a hemodynamic index, rather than its absolute value at a single time, is important in clinical practice, as in the case when evaluating 2 different IABP-weaning strategies. Accordingly, considering that most of the mentioned mechanisms responsible for a “high” cardiovascular stiffness are unavoidable and constant in a given patient, provided that a good transduction setting is used and that preload and afterload have been adequately corrected, we still consider that the traditional PRAM method (in the absence of ad hoc manufactured transducers) can be reliable in clinical practice, as long as a proficient human validation phase of the PRAM-derived indices is provided.

We are pleased that our study confirmed, for the first time in humans, the important findings found by Gelsomino and colleagues⁶ in a swine model of acute ischemia, in which hemodynamic indices and cardiac contractile efficiency parameters were similarly derived by conventional PRAM methodology, showing the same inadequacy of 1:2 and 1:3 IABP assisting rates on hemodynamic recovery.

We hope that these endeavors, taken together, can open the way to a new paradigm when weaning patients from IABP support.

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References

1. Onorati F, Santini F, Amoncelli E, Campanella F, Chiominto B, Faggian G, et al. How should I wean my next intra-aortic balloon pump? Differences between progressive volume weaning and rate weaning. *J Thorac Cardiovasc Surg.* April 19, 2012 [Epub ahead of print].
2. Gelsomino S, Romano SM. Cardiac cycle efficiency during counterpulsation. *J Thorac Cardiovasc Surg.* 2013;145:889-90.
3. Romagnoli S, Romano SM, Bevilacqua S, Lazzeri C, Gensini GF, Pratesi C, et al. Dynamic response of fluid-filled catheter systems for measurement of blood pressure: precision of measurements and reliability of the Pressure Recording Analytical Method with different disposable systems. *J Crit Care.* 2011;26:415-22.
4. Gardner RM. Direct blood pressure measurement—dynamic response requirements. *Anesthesiology.* 1981;54:227-36.
5. Zangrillo A, Maj G, Monaco F, Scandroglio AM, Nuzzi M, Plumari V, et al. Cardiac index validation using the pressure recording analytic method in unstable patients. *J Cardiothorac Vasc Anesth.* 2010; 24:265-9.
6. Gelsomino S, Renzulli A, Rubino AS, Romano SM, Lucà F, Valente S, et al. Effects of 1:1, 1:2 or 1:3 intra-aortic balloon counterpulsation/heart support on coronary haemodynamics and cardiac contractile efficiency in an animal model of myocardial ischemia/reperfusion. *Eur J Cardiothorac Surg.* 2012;42:325-32.

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GUIDED OR NONGUIDED ENDOCARDECTOMY DURING SURGICAL VENTRICULAR RECONSTRUCTION?

To the Editor:

In a recent article in the *Journal*, Babokin and colleagues¹ presented results after surgical ventricular reconstruction (SVR) together with endocardectomy along radiofrequency ablation-induced markings. The objective was to evaluate the efficacy of a new approach for endocardectomy during SVR in patients with post-infarction left antero-septal ventricular aneurysms. The extent of the endocardectomy was guided by preoperative electroanatomic mapping and markings produced by radiofrequency ablations. The design was a retrospective single-center study, and 168 patients were included and categorized in 2 groups: 74 patients who underwent SVR including endocardectomy and 94 patients who underwent SVR. Whether or not endocardectomy should have been added to SVR was decided by the surgeon. All patients also underwent coronary artery bypass grafting. In the SVR + endocardectomy group and SVR only group, early mortality was 1% and 6%, respectively, and 1-year mortality was 5% and 13%, respectively. Cardiac function and volumes were similar between the groups both before and after surgery. In a subgroup of patients who underwent both pre- and postoperative electrophysiologic studies, Babokin and colleagues¹ found a lower incidence of spontaneous and induced ventricular tachycardia (VT) after surgery in patients who underwent SVR + endocardectomy compared with SVR only. There were also fewer implantable cardioverter-defibrillators (ICDs) in the SVR + endocardectomy group.

Patients are at risk for malignant arrhythmias after SVR; however, whether the risk is high² or low³ is unclear. One strategy would be to use ICDs routinely after SVR. However, there are downsides to ICD use, for

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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References

1. Babokin V, Shipulin V, Batalov R, Popov S. Surgical ventricular reconstruction with endocardectomy along radiofrequency ablation-induced markings. *J Thorac Cardiovasc Surg.* October 12, 2012 [Epub ahead of print].
2. O'Neill JO, Starling RC, Khaykin Y, et al. Residual high incidence of ventricular arrhythmias after left ventricular reconstructive surgery. *J Thorac Cardiovasc Surg.* 2005;130:1250-6.
3. Baravelli M, Cattaneo P, Rossi A, et al. Low-risk profile for malignant ventricular arrhythmias and sudden cardiac death after surgical ventricular reconstruction. *Pacing Clin Electrophysiol.* 2010;33:1054-62.
4. Dor V, Sabatier M, Montiglio F, Rossi P, Toso A, Di Donato M. Results of nonguided subtotal endocardectomy associated with left ventricular reconstruction in patients with ischemic ventricular arrhythmias. *J Thorac Cardiovasc Surg.* 1994;107:1301-7.
5. Rastegar H, Link MS, Foote CB, Wang PJ, Manolis AS, Estes NA III. Perioperative and long-term results with mapping-guided subendocardial resection and left ventricular endoaneurysmorrhaphy. *Circulation.* 1996;94:1041-8.
6. Sartipy U, Albåge A, Strååt E, Insulander P, Lindblom D. Surgery for ventricular tachycardia in patients undergoing left ventricular reconstruction by the Dor procedure. *Ann Thorac Surg.* 2006;81:65-71.
7. Jones RH, Velazquez EJ, Michler RE, et al. Coronary bypass surgery with or without surgical ventricular reconstruction. *N Engl J Med.* 2009;360:1705-17.

8. Harken AH, Horowitz LN, Josephson ME. Comparison of standard aneurysmectomy and aneurysmectomy with directed endocardial resection for the treatment of recurrent sustained ventricular tachycardia. *J Thorac Cardiovasc Surg.* 1980;80:527-34.

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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