technique been used with the aim of preserving the marginal arteries.

In summary, therefore, the authors have done no more than confirm that resection or constriction at the apex has a strictly cosmetic effect on ejection fraction and a negligible effect on global left ventricular function. There is no need to invoke any pivotal impact of the spiraling muscles presumed to be involved in ventricular filling so as to explain the lack of therapeutic benefit. Indeed, the authors have provided no evidence of a major impairment of the dynamics of the ventricular filling apart from a moderate increase in the filling pressure of the left ventricle and a decrease by one tenth in stroke volume. In our opinion, this latter finding is more likely due to the overcorrection of the volume of the ventricle by reducing its long axis. They should remember that clinical results have now provided ample evidence that when the left ventricle is dilated, resection of any part of its walls, including the septum, is tolerated without impairing left ventricular function.³⁻⁶ Indeed, a marked improvement in left ventricular function is achieved whenever the radius of the upper two thirds of the left ventricle is significantly reduced. The conclusions drawn by the authors, namely, that surgical intervention on the left ventricular apex is markedly detrimental, are not, in our opinion, supported by their experimental evidence.

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Reply to the Editor:

Savage and colleagues' study¹ demonstrated that volume reduction surgery (VRS) in a model of left ventricular (LV) aneurysm without global LV dysfunction plicated only LV apex. On the other hand, apex-sacrificing VRS in our series reduced the diameter of not only LV apex but also LV mid-portion in a model of global LV dysfunction. The effects of volume reduction in akinetic or dyskinetic area are different from hypokinetic area.² Drs Lunkenheimer and Anderson support reduction of the radius of the ventricle in its upper two thirds (ie, basal). We agree with them on that point, and reduction of the upper two thirds of left ventricle is the same as our apex-sparing VRS. In clinical series, however, no one has described or advocated reduction of the radius of the ventricle in its upper two thirds. The letter by Drs Lunkenheimer and Anderson focused on the issue in LV aneurysm or ischemic cardiomyopathy (raised by Savage and colleagues¹), but the disease that we studied via this animal model was dilated cardiomyopathy (ie, nonischemic). From this viewpoint, their criticism is interesting and important but misdirected.

Konertz and coworkers³ reported excellent results of VRS but they also did not described reduction of the upper two thirds of left ventricle, and a majority of their cases are ischemic cardiomyopathy. Again, our study was focused on the dilated cardiomyopathy (nonischemic cardiomyopathy, a different disease). It is difficult to adopt their results to our study.

Theoretically the therapeutic effect of reducing ventricular radius is at least in part dependent on the degree of preexisting ventricular dilation. In the clinical setting, however, operative mortality is high and LV redilatation and dysfunction are not rare after VRS.⁴⁻¹⁰ This suggests that the theoretical concept is not necessarily reproducible in the clinical situation and that the way to excise and repair the LV is also important. Although there are some limitations due to the model of acute heart failure induced by β -blocker, the heart rate before and after VRS was statistically no different between the 2 groups. LV contractility in apex-sparing VRS was shown to be superior to apex-sacrificing VRS, and LV end-diastolic pressure was lower in apex-sparing VRS. Those differences between the 2 groups were significant.

Batista's group described an incision made at the apex of the left ventricle. In our article,² we never stated that Batista and colleagues advocated the apex resection, but they did not try to preserve the apex as much as possible. In fact, Lunkenheimer and colleagues¹¹ use the term "oyster-shaped excision" in their case report, following the scheme of Batista's operation by widely resecting the LV apex. Throughout our article we tried to show the potential pitfall of the great operation described by Batista and colleagues and to improve it. There would be some ischemic damage extending beyond the plicated segment in VRS, and we have described plication effects in the adjacent area in our study limitation. It is unknown, however, whether the ischemic damage follows according to the position of resection or plication area. However, this potential concern of compromising the marginal arteries is equally applicable to conventional Batista procedure. Further investigation would be warranted.

We have already demonstrated these 2 types of VRS in a model of chronic dilated cardiomyopathy.¹² Elevation of LV end-diastolic pressure and LV redilatation were

remarkable from 1 week after apex-sacrificing VRS but not in apex-sparing VRS. Drs Lunkenheimer and Anderson described in their letter that resection of any part of LV wall, including the septum, is tolerated without impairing LV function when the left ventricle is dilated. However, septal anterior ventricular exclusion or pacoexy reduces the septal and anterior wall without amputating the LV apex and yields good clinical results.^{13,14} We think those results are compatible with our study.

In addition, results of some clinical cases that underwent the apex-sparing VRS in our unit have been excellent so far. Thus we believe that it is important to reduce the LV volume in dilated left ventricle with severe LV dysfunction. Apex-sparing VRS may improve the clinical results.

Although the hypothesis by Dr Torrent-Guasp and colleagues is very interesting,¹⁵ there is no scientific proof so far. Therefore the relationship between cause and result in the improved LV function after the apex-sparing VRS and preservation of single muscle band is still unknown, and further study will be required.

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Study of warm perfusion rather than cardioplegia

To the Editor:

I read the article by Mallidi and colleagues,¹ "The Short-term and Long-term Effects of Warm or Tepid Cardioplegia," and raise the following concern. The title is not descriptive of the protocols. Rather than isolated cold or warm cardioplegia, the article really describes cold and warm total-body and cardiac perfusion strategies. The article states, "In the warm or tepid blood cardioplegia group, the systemic

temperature was maintained at 33°C to 37°C, and the blood cardioplegia was delivered at a temperature of 37°C. In the tepid cardioplegia group, the systemic temperature was permitted to drift passively during the operation to 32°C to 34°C. The temperature of the cardioplegia was 28° to 30°C. In the cold cardioplegia group, the systemic temperature was actively cooled to 25°C to 32°C, and the blood cardioplegia was actively cooled to a temperature of 5°C to 8°C."

Other combinations may have similar results, for example a warm corporeal perfusion strategy (drifting without active cooling) and cold cardioplegia. The data do not preclude such a result.

This group has done a nice job scientifically studying and promoting warm perfusion and protection strategies. I think that describing their technique as "warm blood cardioplegia" does not describe the strategy adequately, and "warm perfusion strategy" might be more accurate.

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Reply to the Editor:

Dr Savage correctly points out that the article published in the March issue of the Journal by our group was not simply a comparison of cold versus warm or tepid cardioplegia, but rather a comparison of the strategy of warm or tepid cardioplegia versus cold cardioplegia. The three cardioplegic and systemic perfusion strategies used in patients undergoing isolated coronary artery grafting surgery at our institution were described in detail in the article. Other possible cardioplegic and systemic perfusion strategies (such as warm systemic perfusion with cold cardioplegia, tepid systemic perfusion with cold cardioplegia, systemic hypothermia with warm or tepid cardioplegia, and so on) were not used in our institution.