After endoventricular linear patch plasty, the decrease in EDV became linearly proportional to SV in both groups of patients (Figure 1). The SV either decreased from baseline or showed no significant change from baseline. None of the patients demonstrated an increase in SV after SVR. For this same reason, the ejection fraction is a poor indicator of left ventricular pump function after SVR.

The EDV and SV have been documented to be linearly proportional in the normal heart under various loading conditions.⁵ This linearity signifies a normal homeostatic relationship in an ellipsoid ventricle.

Therefore, left ventricular pump function and clinical outcome in SVR are dependent on the anatomic substrate of the surgically restored ventricle, which should demonstrate the normal physiologic linearity between EDV and SV.

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Reply to the Editor:

Since the original contributions by Dor and colleagues,¹ surgical ventricular restoration (SVR) for post-infarction dilated cardiomyopathy has consistently shown favorable changes in left ventricular (LV) geometry (reduction in volumes and increase in ejection fraction) and circulatory equilibrium (increase in ventricular elastance and reduction in arterial elastance), associated with clinical improvement.² However, these favorable changes often were in contrast with the frequent occurrence of postoperative stroke volume (SV) reduction at rest after SVR.³ The main and universally accepted notion is that the reduction of cardiac output is the most important pathogenetic mechanism of heart failure, but after SVR, stroke volume index (SVI) reduction is accompanied by a net clinical improvement. The apparent paradox of heart failure with reduced ejection fraction presenting with a baseline SVI higher than normal that decreases after SVR deserves some comments.

One hypothesis to explain SVI reduction is diastolic dysfunction induced

by SVR,⁴ but we did not find significant abnormalities in the diastolic filling pattern in the majority of patients. A previous study from our group, exploring regional contraction synchrony through segmental pressure/length loops,⁵ demonstrated that SVR induces a mechanical resynchronization through the improvement in shape and orientation of the single regional P/L loops with normalization of both contraction and relaxation isometric phases, thus improving global wall contraction. That study also analyzed diastolic function and found that SVR induces an early improvement in filling pressure with a significant decrease in minimal and end-diastolic pressure.

A second hypothesis to interpret SVI decrease is the reduction of mitral regurgitation induced by SVR. Because SV is calculated by the difference between end-diastolic and end-systolic volumes, a reduction in mitral regurgitation might cause an apparent reduction in total SVI, without a real reduction in antegrade flow. This hypothesis is unlikely, because in our recent study (commented on by Adhyapak and Parachuri⁶ in this

FIGURE 1. Stroke volume (SV) changes after surgical ventricular restoration. Patients who decrease stroke volume after surgery (red squares) have a higher value at baseline in respect to patients who increase stroke volume (blue circles). A, Stroke volume index (SVI) values in mL/m²; B, percent change in SVI. At follow-up, no differences are detected between the 2 groups. **P* < .01.

issue of the *Journal*), we intentionally excluded patients with mitral regurgitation, but SV decreased in the majority of patients after SVR.

Finally, the hypothesis that intrinsic contractility is reduced or at least not improved by the intervention, despite the improvement in LV geometry in terms of volumes and ejection fraction, seems unlikely in view of the well-demonstrated clinical improvement after surgery. One possible interpretation is that after SVR the ventricle regains its preload reserve that allows SV to increase during exercise, independently of the baseline value of resting SV. Unfortunately, data on exercise after SVR are not available yet.

The linear and significant correlation between SV and end-diastolic volume also was confirmed in the study by Adhyapak and Parachuri.⁶ We found that the majority of patients (almost all in New York Heart Association class III/IV) had normal or even increased resting SV (normal volume, 27 ± 7 mL/m²). Our patients presented with distinct hemodynamic patterns that seemed to influence the response to SVR: Patients with greater than normal preoperative SVI showed SVI reduction after SVR, whereas patients with lower preoperative SVI showed SVI improvement. At 1 year, control average resting SVI was within normal limits in both groups (Figure 1).

These 2 different SVI responses to SVR correspond to 2 distinct baseline geometric patterns: one characterized by the increase in LV internal diameter and volume, either as absolute or relative values of wall thickness and LV mass ("eccentric remodeling"), and one characterized by a relatively smaller increase of chamber size in respect to parietal thickness and mass ("concentric remodeling"), present in a small number of patients. Ejection fraction is progressively reduced in the first geometric pattern, whereas it is relatively preserved in the second pattern.

SV increase proportional to end-diastolic volume increase most likely reflects the increase in venous return

that, in the absence of an increase in peripheral needs, depends on an increase in circulatory mass, which in turn depends on water and sodium retention. In other words, eccentric remodeling identifies a "backward heart failure" secondary to the increase in filling pressure with pulmonary congestion. Dilated ventricles have a greater effective preload than small ventricles (at the same end-diastolic intracavitary pressure). This factor is rarely taken into account when P/V loops of ventricles with different geometry are compared. The postoperative increase in SVI of patients with concentric remodeling pattern and reduced baseline SVI might be the result of ventricular afterload reduction. We are aware that further analysis is needed to understand the origin of such different geometric patterns after myocardial infarction and the differences in cardiocirculatory equilibrium.

CONCLUSIONS

Data on the analysis of geometric patterns of patients undergoing SVR after myocardial infarction raise interesting questions on the clinical pathology and physiopathology of heart failure in ischemic cardiomyopathy. The solution might have an important impact not only on the patient selection for surgical treatment but also on the medical approach to different remodeling patterns.

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RALPH LEWIS, MD: A PIONEER IN THORACIC SURGERY

To the Editor:

Ralph Lewis passed away at age 77 on February 15, 2010. He was a graduate of New York Hospital–Cornell Medical School and did his residency there.

I first met Dr Lewis when I was an intern at University of Medicine and Dentistry of New Jersey–Robert Wood Johnson Medical School. He was chief of thoracic surgery at St Peters Hospital and the lead thoracic surgeon of his group, which included Glen Sisler (and previously James