IMPACT OF SURGICAL VENTRICULAR RESTORATION ON STROKE VOLUME: SURGICAL FINE-TUNING OF THE RELATIONSHIP BETWEEN END-DIASTOLIC VOLUME AND STROKE VOLUME

To the Editor:

We appreciate the elegant study by Di Donato and colleagues,¹ "Impact of surgical ventricular restoration on stroke volume in patients with ischemic cardiomyopathy," which elucidates that left ventricular pump function after surgical ventricular res-

toration (SVR) does not rely on stroke volume (SV).

The benefits of SVR in patients with ischemic cardiomyopathy and left ventricular aneurysms occur largely by decreases in the end-diastolic volume (EDV), leading to improvements in left ventricular pump function. The left ventricular pump function improvements are evident by increases in contractility measured by the E max and decreases in arterial elastance Ae.² Increases in SV as an index of improved ventricular function do not occur after SVR, as documented by various registry data.

The SV is a load-dependent index. Klein and colleagues³ have explained the phenomenon of changes in SV in ischemic cardiomyopathy. When the noncontractile or aneurysmal portion of the myocardium exceeds 20% to 25% of the ventricular surface area, the remaining contractile myocardium has to dilate to maintain a forward SV. This increase in EDV need not be proportional to SV.

However, we would like to highlight an interesting observation in our pa-

tients. We have studied 101 patients with post-infarction left ventricular aneurysms subjected to a modified technique of SVR: endoventricular linear patch plasty.⁴ We modified the endoventricular circular patch plasty by doing away with the Fontan suture and suturing a rectangular linear patch to the border zone, which achieved near total exclusion of the infarcted muscle. This linear endoventricular patch was sutured to exclude the infarcted myocardium from the left ventricular base to apex. At baseline, in most patients the end-diastolic volume indexed to body surface area was linearly proportional to SV indexed to body surface area ($r = 0.38$, $P < .001$, Figure 1), as seen in the patients in the study by Di Donato and colleagues.¹ The SV in these patients increased in proportion to increases in EDV (marker = 0). These increases in SV may be related to increases in preload. However, in some patients (marker = 1, Figure 1), SV did not increase in proportion to increases in EDV. Here, the SV may be more influenced by increases in afterload.

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Relationship of EDVI and SVI before and after ppt

FIGURE 1. Relationship of EDV index and SV index at baseline and after endoventricular linear patch plasty. Marker 0 (clear circles) = patients who demonstrated increase in SV with increases in EDV at baseline. Marker 1 (black circles) = patients who did not demonstrate an increase in SV at baseline with increases in EDV. SVI, Stroke volume index; EDVI, end-diastolic volume index.

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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doi:10.1016/j.jtcvs.2010.11.053

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.