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

Mackenzie—then chief of surgery). What distinguished Dr Lewis from the outset was his confidence. He never had second doubts; he never wavered when faced with a life threatening emergency. Although very cautious in terms of patient selection, he was audacious as a technical surgeon. On my first official rotation with him as a second-year resident, I knew that I would always be a thoracic surgeon. We had what would be called today multidisciplinary conferences, which usually revolved around whether a computed tomographic scan should be performed or whether a tissue diagnosis by computed tomographic guidance or bronchoscopy was appropriate. He was a firm believer in routine mediastinoscopy and taught me the concept of “med on Monday and thoracotomy on Wednesday (or the following Monday!). He was, with his partner Dr James Mackenzie, one of the first proponents of routine thoracoscopy, then using a standard mediastinoscope for pleural disease. This early exposure to “rigid thoracoscopy”, obviously later led to my interest in utilizing newer technology in helping to develop video-assisted thoracoscopic surgery once in practice. He firmly believed that any surgery for mesothelioma should be done only for palliation, because there was no way to cure these patients surgically. He arrived at this conclusion after personally doing dozens of autopsies on these patients and demonstrating microscopic invasion into the endothoracic fascia and chest wall. Although the concept of combined modality therapy had not yet gained acceptance, he at least used routine surgical staging of mesothelioma and non-small cell lung cancer to refer patients for definitive nonoperative therapy in cases of positive findings.

My first transforming experience with him was when I was second-year resident, assisting on a transhiatal esophagectomy. The chief resident cut the left hepatic vein flush with the in-

ferior vena cava, and the patient began to exsanguinate. I immediately put my hand on the cava, and asked for a sternal saw. He looked at me, somewhat bewildered, and said, “Can you do this?” to which I promptly replied, “Yes.” After he said to go ahead, and I controlled the avulsed cava intrapericardially and repaired the left hepatic vein–caval junction below (with 3-0 silk!), I was a constant presence on his major cases for the next 4 years of my training. I got to know his sense of humor, practical approach to problem solving, and personal contact with each of his patients firsthand. From placing the arterial line and double-lumen tube preoperatively to intensive care unit and ventilator management postoperatively, Dr Lewis was a constant presence in his patients’ postoperative care. I vividly remember him teaching me how to do chest physiotherapy with percussion and incentive spirometry, so that I could teach the nurses and therapists when the need arose.

I have a very personal debt to Dr Lewis, who introduced me to my next mentor, Dr F. Henry (Bunky) Ellis, Jr. I am convinced that if not for his belief that I would make a successful academic thoracic surgeon, I would never have gotten into the prestigious New England Deaconess program in 1988.

After completing my training at Harvard, I was fortunate to get to know Ralph, as he now insisted on being called, on a much more personal basis. We met each other frequently and debated with each other constantly on the different aspects of thoracoscopy developed as we both tried to push the field in our own directions. Ralph believed that smaller operations would allow “other therapies” to complement the operation and achieve good long-term results, while I was committed to a conservative, standard oncologic approach with smaller incisions. Even this competition was fruitful, as I later found out that he single-handedly

helped all the Young Turks of the thoracoscopy era (Landraneau, Hazelrigg, Naunheim, Mack, Krasna, and so on) to get published in the *Annals of Thoracic Surgery* (under the careful eye of Dr Tom Ferguson).

Ralph’s contributions to the field of thoracic surgery include articles and presentations at national meetings. Most importantly, he was the dominant force in thoracic surgery for New Jersey for more than 3 decades and trained numerous future cardiac and thoracic surgeons in the field of general thoracic surgery at a time when there were few American surgeons who had dedicated themselves to our subspecialty. His humor, warmth, and frank, outspoken nature will be missed by us all.

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GENTLE GIANT: WILFORD NEPTUNE, MD

To the Editor:

Wilford B. Neptune passed away on April 3, 2010.

My favorite vignette of Dr Neptune is definitely my last case operating with him and his senior partner, Dr Richard Overholt, at the New England Deaconess Hospital in Boston in the spring of 1990. Imagine the scene. The patient is in the prone position,