Time course of regression of left ventricular hypertrophy after aortic valve replacement

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ABSTRACT To assess the time course and extent of regression of myocardial hypertrophy after removal of the inciting hemodynamic stress, 21 patients with either aortic stenosis or aortic insufficiency were studied preoperatively, after an intermediate period (1.6 ± 0.5 years), and late (8.1 ± 2.9 years) after aortic valve replacement, and results were compared with those in 11 control patients. After aortic valve replacement there was significant hemodynamic improvement, with a fall in the left ventricular end-diastolic volume index (164 ± 73 to 105 ± 35 ml/m², p < .01), a fall in left heart filling pressure (19 ± 9 to 12 ± 5 mm Hg, p < .01), and maintenance of the cardiac index (3.3 ± 0.8 to 3.5 ± 0.8 liters/min/m², NS) and left ventricular ejection fraction (60 ± 13% to 64 ± 10%, NS). By the late study the cardiac index (4.0 ± 0.6 liters/min/m², p < .01) and left ventricular ejection fraction (66 ± 15%, p < .05) had further increased and were significantly greater than before surgery. For the group as a whole, the left ventricular muscle mass index fell 31% after surgery by the time of the intermediate postoperative study (174 ± 38 vs 120 ± 29 g/m², p < .01), and a further 13% from the intermediate to the late study (105 ± 32 g/m², p < .05). At the preoperative study left ventricular muscle mass index was greatest in those patients with aortic insufficiency (191 ± 36 g/m²), and greater in those with aortic stenosis (158 ± 33 g/m²) than in control subjects (85 ± 9 g/m², p < .05). At the intermediate postoperative study left ventricular muscle mass index remained significantly higher in both those with preoperative aortic insufficiency (128 ± 29 g/m²) and those with stenosis (114 ± 27 g/m²) than in the control subjects (p < .01). By the time of the late postoperative study there were no longer any significant differences in left ventricular muscle mass index. Thus, the regression of myocardial hypertrophy is a process that occurs over many years after correction of the primary hemodynamic abnormality. As this process of myocardial remodeling occurs, continued improvement in cardiac function may occur, and the improvement occurring between the intermediate and late postoperative studies at a slight but constant afterload excess (inherent in the relative stenosis of the aortic prosthesis) suggests that the hypertrophied myocardium is operating at a reduced level compared with normal myocardium. Circulation 77, No. 6, 1345–1355, 1988.

LEFT VENTRICULAR pressure and volume overloads in response to aortic valve stenosis and aortic valve insufficiency lead to a marked hypertrophic response of the myocardium, likely as an adaptive response to normalize the increased wall stress accompanying these states.1 While this allows for maintenance of cardiac performance, it may lead to progressive myocardial failure.2 Aortic valve replacement has produced a dramatic change in the “natural” history of these disorders. There has been reported improvement in survival,3 New York Heart Association class,4,5 and resting hemodynamics.6-11 This improvement occurs even though the prosthetic valve itself constitutes a relative and persistent, although not progressive, stenotic obstruction to left ventricular ejection.12,13 The extent to which the myocardium itself may recover or deteriorate as a result of the initial hemodynamic insult in the years after such successful surgery and the regression of the attendant hypertrophy have not been defined.

In this study, we investigated the time course of regression of myocardial hypertrophy in 21 patients...
studied preoperatively, after an intermediate period, and late after successful aortic valve replacement.

Methods

Twenty-one ambulatory patients who had had prior aortic valve replacement (for predominant aortic stenosis in 11, and for predominant aortic insufficiency in 10) were studied preoperatively, after an intermediate period (1.6 ± 0.5 years), and late (8.1 ± 2.9 years) after surgery (table 1). Patients selected for study were from those followed in this hospital who had had the original (i.e., preoperative) cardiac catheterization at this institution, and who had no coronary artery disease at the time of preoperative or intermediate studies. Informed consent was obtained from all patients under a protocol approved by the human studies committee of University Hospital. No complications occurred in the performance of this study.

All but one patient at the preoperative study and all patients at the intermediate postoperative studies underwent tip-micromanometer left ventricular pressure measurements and contrast left ventriculography. At the late study 12 patients underwent tip-micromanometer left ventricular pressure measurement and contrast ventriculography. The other nine patients at late study underwent right heart catheterization and digital-subtraction left ventriculography (described below). In all patients with left ventricular tip-micromanometer studies the catheter was introduced via the transseptal route. At the late study arterial pressure was measured with an arm sphygmomanometer.

In nine subjects left heart filling pressures at the late study were assessed by right heart catheterization (with a No. 7F pulmonary artery balloon flotation catheter with a tip-micromanometer in seven patients and with a fluid-filled balloon flotation catheter in two patients; in those patients in whom a tip-micromanometer catheter was used, calibration of the pressure was performed with the pulmonary artery pressure). In these patients the mean pulmonary capillary wedge pressure was measured and used for analysis as the left heart filling pressure instead of the left ventricular end-diastolic pressure.

Left ventriculography was performed in the 30 degree right anterior oblique projection. In those nine patients studied by right heart catheterization, left ventriculography was performed by digital-subtraction angiography by a method previously described and validated for the determination of left ventricular end-diastolic volume, end-systolic volume, and ejection fraction, as well as for the determination of left ventricular end-diastolic wall thickness and muscle mass. With this technique, injection of radiographic contrast (sodium and meglumine ioxaglate [Hexabrix 320]) was into the pulmonary artery. Cineangiography (direct and subtraction) was recorded on 35 mm cinefilm (Siemens Angioscope) at 50 frames/sec, with simultaneous recording of the electrocardiogram, the numerical code for the frames recorded, and left ventricular pressure (or the pulmonary artery wedge pressure) on an oscillograph (Electronics for Medicine VR12) at a paper speed of 250 mm/sec in all patients. A metal sphere of known diameter was filmed for calibration of left ventricular volumes.

For the digital-subtraction studies, the cinefilm was digitized with a high-resolution Eikon 78/99 diode array camera (2048 × 2048 × 12) and processed on a de Anza IP8500 image system in combination with a VAX 750 using standard software. Subtraction masks were obtained by both the mask mode and time interval difference methods. Contour detection was performed with a “mouse”-directed, contour point selection system, with connection of the points by a cubic spline function.

Left ventricular volumes were calculated by the area-length method, with modification for monoplane angiograms. End-diastole was defined by either the pressure trough immediately after the A wave (if present) or the point 20 msec preceding the peak of the R wave of the electrocardiogram for data from tip-micromanometers, and by the peak of the R wave of the electrocardiogram for data from fluid-filled catheters (n = 1). End-systole was defined by the minimal angiographic left ventricular volume. In those studies for which data from left ventricular catheterization were used, the cardiac index was calculated by the Fick method, while in the right heart studies the angiographic cardiac index was used. Left ventricular muscle mass was calculated after the method of Rakkey and associates. In the late study mean arterial pressure (MAP, in mm Hg) was estimated by:

\[
MAP = (SAP + 2·DAP)/3
\]

where SAP is the systolic cuff pressure (mm Hg), and DAP is the diastolic cuff pressure (mm Hg).

As an index of the pattern of left ventricular hypertrophy, the ratio of left ventricular wall thickness to left ventricular short-axis radius was calculated, with levels above the control group (see below) considered to represent a pattern of "concentric hypertrophy," while those equal to or less than the control group were considered to represent "eccentric hypertrophy." As an index of left ventricular shape, left ventricular "eccentricity" (e) was calculated with the following formula:

\[
e = (r_l^2 - r_s^2)/r_l
\]

where \(r_l\) is the long-axis radius, and \(r_s\) is the short-axis radius.

Eleven patients with no or minimal cardiac disease on routine diagnostic cardiac catheterization served as control subjects. These included two patients with noncritical coronary artery disease (< 50% stenosis epicardial coronary arteries), and one with mitral valve prolapse syndrome without regurgitation. In these patients left ventricular catheterization was performed by the retrograde approach. Left ventricular pressures were measured with tip-micromanometer catheters and standard contrast ventriculography was performed.

Statistical comparison of data from the three study points (preoperative, intermediate, and late postoperative) was made by one-way analysis of variance for repeated measures and if significant (defined as p < .05) differences were found, multi-sample comparison was performed with the Newman-Keuls test. For comparison of the data from the subgroups with aortic stenosis and aortic insufficiency at similar time junctures, one-way analysis of variance was performed on data from all three study groups (baseline study for the controls was used at each comparison point), and if significant differences were found the Newman-Keuls test was again applied. All values are expressed as the mean ± SD.

Results

Patient characteristics are presented in table 1. The hemodynamic data from the three studies are presented in tables 2A and 2B. Left ventricular end-diastolic pressure was significantly lower at the intermediate (12 ± 5 mm Hg, p < .01) and late postoperative (9 ± 3 mm Hg, p < .01) studies than at the preoperative (19 ± 9 mm Hg) study. The cardiac index was higher at the late postoperative study (4.0 ± 0.6 liters/min/m², p < .01) than at either the preoperative (3.3 ± 0.8 liters/min/m²) or intermediate (3.5 ± 0.8 liters/min/m²) studies.
TABLE 1

Patient characteristics

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17 54 M AI 6 61 CE 29 0 16 74
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19 56 M AI 0 49 BS 31 0 15 107
20 48 M AI 0 58 BS 27 0 36 157
21 64 M AI 0 77 BS 31 0 14 105
Mean 49 4 59 30 6 20 85
SD 8 12 10 1 6 ^ ^

22 40 M Minimal CAD
23 40 M Normal
24 63 M Normal
25 46 M Normal
26 57 M Normal
27 58 M Normal
28 52 M Normal
29 49 F MVP
30 54 M Normal
31 44 M Normal
32 53 M Minimal CAD
Mean 51
SD 7

AI = aortic insufficiency; AS = aortic stenosis; AS/AI = combined aortic stenosis and insufficiency; CAD = coronary artery disease; MVP = mitral valve prolapse syndrome; BS = Bjork-Shiley valve prosthesis; LK = Lillehei-Kaster prosthesis; CE = Carpentier-Edwards bioprosthesis; Study 2 = the intermediate postoperative study; Study 3 = the late postoperative study.

^p < .01, compared with patients with preoperative aortic stenosis; see text for details.

Postoperatively, both intermediate (105 ± 35 ml/m², p < .01) and late (96 ± 41 ml/m², p < .01), the left ventricular end-diastolic volume index was significantly smaller than preoperatively (164 ± 73 ml/m²). The left ventricular ejection fraction was not significantly different at the intermediate postoperative time point (64 ± 10%) than preoperatively (60 ± 13%); however, at the late postoperative study it was significantly higher (66 ± 15%, p < .05) than preoperatively.

Left ventricular end-diastolic wall thickness was significantly thinner intermediate (0.97 ± 0.12 cm, p < .01) and late (0.91 ± 0.13 cm, p < .01) after surgery than preoperatively (1.09 ± 0.15 cm). Left ventricular muscle mass index was significantly lower at the intermediate (120 ± 29 g/m², p < .01) than at
### TABLE 2A
Hemodynamic data: heart rate, left ventricular systolic and end-diastolic pressure, right atrial pressure, cardiac index, and left ventricular end-diastolic volume index

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To estimate the extent to which the pulmonary capillary wedge pressure underestimated left ventricular end-diastolic pressure in these patients, a regression analysis between left atrial pressure and left ventricular end-diastolic pressure was performed for all patients at the preoperative and intermediate postoperative studies, combined with those 12 patients with transseptal studies at the late study (n = 54). The correlation coefficient was .8195, with the equation y = 0.539x + 1.151; calculation of left ventricular end-diastolic pressure from the measured pulmonary capillary wedge pressure led to a revised mean left heart filling pressure at the last study of 13 ± 4 mm Hg (vs 10 ± 3 mm Hg), with no change in the statistical results.

1 = preoperative study; 2 = intermediate postoperative study; 3 = late postoperative study; HR = heart rate (beats/min); LV-SYS = left ventricular systolic pressure or (*) systolic cuff pressure (mm Hg); LV-DIA = left ventricular end-diastolic pressure or (*) pulmonary capillary wedge pressure (mm Hg); RA = right atrial pressure (mm Hg); CI = cardiac index (l/min/m²); EDVI = left ventricular end-diastolic volume index (ml/m²).

*p < .01 vs control; ^p < .05 vs preoperative study; çp < .05 vs preoperative study; ^p < .05 vs control; çp < .01 vs preoperative and intermediate study; ç^p < .01 vs aortic stenosis; ççp < .01 vs aortic stenosis and control; ^çp < .05 vs aortic stenosis and control.
## TABLE 2B

Hemodynamic data: left ventricular end-systolic volume index, ejection fraction, wall thickness, muscle mass index, wall thickness-to-radius ratio, and eccentricity

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A = control; B = time 1; C = time 2; D = time 3; E = late postoperative; F = intermediate postoperative; G = preoperative

1 = preoperative study; 2 = intermediate postoperative study; 3 = late postoperative study; ESVI = left ventricular end-systolic volume index (ml/m²); EF = left ventricular ejection fraction (%); WT = left ventricular end-diastolic wall thickness (cm); MMI = left ventricular muscle mass index (g/m²); h/r = the ratio of left ventricular end-diastolic wall thickness to short-axis radius; E = eccentricity (as detailed in the text).

* indicates statistical significance with the values being:
- *p < .05 vs preoperative study
- **p < .01 vs preoperative study
- "p < .01 vs control
- "p < .01 vs control and preoperative study
- "p < .05 vs control
- "p < .05 vs intermediate study
- "p < .01 vs preoperative and intermediate studies
- "p < .01 vs aortic stenosis and control
- "p < .05 vs aortic stenosis
- "p < .01 vs aortic stenosis
- "p < .05 vs preoperative and intermediate studies

the preoperative study (174 ± 38 g/m²), and significantly lower at the late (105 ± 32 g/m², p < .05) than intermediate study.

When patients who had received an aortic valve replacement for aortic stenosis were compared with those with an aortic valve replacement for aortic insufficiency, those with prior aortic stenosis had a significantly higher postoperative transvalvular gradient. This was likely due to the significantly smaller valve size that could be inserted in the aortic position in those...
with aortic stenosis vs those with aortic insufficiency. Nevertheless, resting peak systolic left ventricular pressure was not significantly different in those with aortic stenosis and those with aortic insufficiency or control subjects at either the intermediate or late postoperative studies.

Postoperatively in both groups there was a significant and sustained fall in left ventricular end-diastolic pressure to levels no longer significantly different from the control value (figure 1).

Preoperatively, the left ventricular end-diastolic volume index was significantly larger in the patients with aortic insufficiency than in those with aortic stenosis or the control subjects (figure 2). This difference persisted to the intermediate postoperative study, but not to the time of the late postoperative study.

Left ventricular end-diastolic wall thickness, conversely, was significantly greater preoperatively in the patients with aortic stenosis than in those with aortic insufficiency, and greater in those with aortic insufficiency than in the control subjects. In those with prior aortic stenosis and those with prior aortic insufficiency left ventricular wall thickness remained significantly greater than in control subjects at the intermediate and late postoperative studies.

At the preoperative study left ventricular muscle mass index was greatest in the patients with aortic insufficiency, and greater in those with aortic stenosis compared with control subjects (figure 3). At the intermediate postoperative study left ventricular muscle mass index remained significantly higher in those with preoperative aortic insufficiency or stenosis than in the control subjects. However, by the time of the late postoperative study there was only a trend ($p < .06$) to a higher left ventricular muscle mass index in those with aortic insufficiency vs control subjects, and no significant difference for those with preoperative aortic stenosis.

Preoperatively the ratio of left ventricular wall thickness to radius was significantly higher in those with aortic stenosis than in those with aortic insufficiency or the control subjects (figure 4). At both the intermediate and late postoperative studies those with aortic stenosis continued to have a greater wall thickness-to-radius ratio than the control subjects. While those with aortic stenosis had a significant fall from the

![FIGURE 1. Left heart filling pressure and cardiac index in patients with aortic stenosis preoperatively (closed circles) and with aortic insufficiency preoperatively (open circles), referenced against the control values (shaded area), at the preoperative, intermediate postoperative, and late postoperative studies. After surgery a significant improvement was seen, and there was no late deterioration evident. See text for details.](image-url)
preoperative to the postoperative levels, those with aortic insufficiency had a significant rise in the left ventricular wall thickness-to-radius ratio postoperatively.

With this ventricular remodeling there were small but significant differences in left ventricular geometry as assessed by the eccentricity index. In patients with preoperative aortic stenosis, at the intermediate postoperative study there was no significant change from the preoperative index. However, by the late postoperative study this had decreased significantly, reflecting a change to a more spheroidal left ventricular shape. In contrast, in patients with aortic insufficiency there was a significant increase in the eccentricity index (i.e., a return to a less spheroidal shape with a reduction in volume overload) at the intermediate postoperative study and a late fall, although to levels still greater than preoperative values.

**Discussion**

Aortic valve disease, whether stenotic or insufficient, is associated with substantial hemodynamic stresses on the left ventricle in the form of pressure and volume overload. To adapt to the particular stress sustained, the left ventricle hypertrophies in a fashion to maintain systolic wall stresses at or near normal levels, and dilates in response to the diastolic stresses sustained. With aortic valve replacement there is a reduction in the degree of hemodynamic stress faced by the left ventricle as the stenotic or regurgitant valve is replaced with a prosthetic valve with a relatively stenotic orifice.

The compensatory response to the volume and pressure overload sustained by the left ventricle develops over a prolonged period involving many years, even decades, as the body grows and the disease progresses. The rate and extent of postoperative regression of the patterns of hypertrophy and dilation seen with these disorders has not been defined.

We found that the regression of left ventricular hypertrophy after aortic valve replacement was a prolonged process in our patients, with substantial change in the intermediate period postoperatively, but with further change late after reduction in ventricular load. The patterns of regression were different for patients with preoperative aortic stenosis and for those with...
preoperative aortic insufficiency, and depended on the primary adaptation. In the former, with regression of myocardial hypertrophy there was a significant reduction in the pattern of concentric hypertrophy, but that pattern persisted even to the late study 9 years postoperatively. In the latter the regression of hypertrophy was characterized by a reduction in wall thickness that was less than the reduction in chamber size, such that it assumed a more concentric pattern. Thus, with aortic regurgitation in the regression of cardiac hypertrophy and dilation, it is reduction in the latter that predominates (a seeming “conservation of mass” greater than “conservation of volume”).

Left ventricular geometry as reflected in the eccentricity index showed no changes with regression of the hypertrophy of aortic stenosis at the intermediate study; however, with continued resolution of myocardial hypertrophy, late there was a more spheroidal shape assumed. Whether this constitutes an evolution to normalcy or is evidence of subtle left ventricular dysfunction is unclear. In patients with aortic insufficiency, with the residual “excess” of hypertrophy over volume through the seeming differential rates of regression of the two, at the intermediate postoperative study left ventricular geometry was more ellipsoidal than preoperatively, as previously observed by Tous-saint et al.  This likely reflects the substantial improvement in end-diastolic wall stress that followed corrective surgery and likely occurred because longitudinal stresses tend to be relatively higher than circumferential and radial stresses, allowing greater resolution of the dimensional changes in those directions. This persisted through to the late study, although it was less pronounced with further regression of myocardial mass. Nevertheless, these changes were small in magnitude and of uncertain clinical significance.

It should be emphasized that these studies are not those of a complete alleviation of the excess hemodynamic load sustained by the left ventricle. All prosthetic aortic valves are relatively stenotic12, 13 compared with the normal aortic valve, and as such constitute an obligatory chronic afterload excess on the myocardium. This is particularly so in those patients with prior aortic stenosis in whom, because of the narrower aortic root, only smaller valve sizes could be inserted, and in whom a larger resting transprosthetic-valvular gradient

FIGURE 3. Left ventricular muscle mass index in patients with aortic stenosis preoperatively (closed circles) and in those with aortic insufficiency (open circles) preoperatively, at the intermediate postoperative study, and late postoperatively. While the greatest fall in muscle mass index occurred by 1 to 2 years after aortic valve surgery, significant further reductions continued to occur, and at the late study it was no longer significantly higher than control subjects. See text for details.
was present. This may in part explain the greater persistence of the pattern of concentric hypertrophy seen even to the time of the late study, and also the trend in those with preoperative aortic insufficiency to a more concentric pattern of hypertrophy. However, this alone would not explain the differences observed between the intermediate and late postoperative studies.

These changes in myocardial mass and configuration were associated with substantial improvements in resting hemodynamics at the intermediate study, and with further improvement from the intermediate to the late study. While a substantial reduction in myocardial load likely explains much of the improvement seen at the intermediate postoperative study, this would not explain the improvement between the intermediate and late postoperative studies. This may, as observed in animal preparations of the regression of myocardial hypertrophy, reflect effects of the adrenergic nervous system and other factors influencing the response of the myocardium to load. Alternatively, this may reflect some myocardial dysfunction in the hypertrophied state that improves with regression of the hypertrophy. One explanation for such an improvement in hemodynamics late after regression of myocardial hypertrophy would be the late resorption of myocardial collagen. Myocardial collagen has been shown to be substantially increased in patients with aortic valve disease both preoperatively and in the intermediate postoperative period. Furthermore, in animal preparations, while regression of deposited collagen may be seen with regression of myocardial hypertrophy, its rate is slower than that of the contractile elements.

It has been suggested that there are fundamental differences in the mechanisms of hypertrophy seen in disorders associated with "concentric" as compared with "eccentric" hypertrophy. The results of this study do not support this because, with the establishment of similar loads, the hypertrophied myocardium (whether initially of a concentric or of an eccentric pattern) assumed similar configurations of wall thickness, chamber radius, and shape. It must be recognized that the patterns of hypertrophy are derived from the configuration of the left ventricle at end-diastole (and thus determined by the stresses at that point), while the stimuli for hypertrophy appear to be systolic stresses. With the postoperative state in both groups being one...
characterized by mild valvular (prosthetic) stenosis, we cannot exclude the possibility that there was an evolution to the patterns seen in response to that new stimulus, but the demonstration of Gaasch et al. 8 of similar findings as early as 1 week after surgery would argue against this.

Others have obtained similar results with regard to the regression of myocardial hypertrophy in the intermediate postoperative period, with an approximate 30% reduction in myocardial mass after successful aortic valve replacement. Pantely et al. 9 found similar regressions of left ventricular hypertrophy, and observed that there was less recovery of function in patients with aortic insufficiency than in patients with aortic stenosis. Krayenbuehl et al. 9 found that postoperative myocardial function was inversely related to the extent of preoperative myocardial hypertrophy. Kennedy et al. 4 while observing a significant reduction in left ventricular muscle mass, found that 1 1/2 years postoperatively there still remained significant hypertrophy compared with control. Gaasch et al. 8 found in an echocardiographic study that improvement of the volumetric or dimensional abnormalities could be seen as early as 7 to 10 days after successful surgery, and that late systolic function may be better than preoperative function as myocardial regression proceeds. Observing that early (6 to 12 months) after aortic valve replacement for aortic insufficiency there were two groups of patients, those with substantial reductions in left ventricular chamber diameter and muscle cross-sectional area and those without, and that it was in the latter that the only two postoperative deaths occurred, they concluded that this constituted a "poor outcome" group. However, while persistent left ventricular dilation and hypertrophy may indeed herald substantial heart failure in certain patients, our results suggest that further normalization of these variables may occur with time and that those who demonstrate this may continue to do well postoperatively. Schuler et al. 28 in an echocardiographic study, also observed that reconfiguration of the left ventricle from a pattern of eccentric to one of concentric hypertrophy occurred early after surgery for aortic insufficiency. In other echocardiographic studies, regression of myocardial hypertrophy and dilation with treatment of systemic hypertension has been demonstrated over periods of 18 months 39 and 5 years. 30 In these studies a concentric pattern of hypertrophy was observed with a decrease in left ventricular muscle mass and in the wall thickness-to-chamber dimension ratio after therapy.

There are several limitations to this study. First, as with any long-term, follow-up study with retrospective selection, patients who fared poorly in the early period could not be included for assessment. Additionally, self-selection by the patients, who had to consent to repeat catheterization, also may have led to nonrandomness in the patient population. Second, because of the constraints in performing the study on an ambulatory basis, at the late study two methods of obtaining left ventricular volume and mass were used (direct contrast ventriculography and digital-subtraction ventriculography). While not optimal, we have shown that results with these two methods correlate extremely well; furthermore, their use was similarly divided between our patients with aortic stenosis and insufficiency, and so any nonrandom variations potentially present should not have affected the results of this study. Third, the pulmonary capillary wedge pressure measured in several of the patients at the late study may somewhat underestimate left ventricular end-diastolic pressure in patients such as these with myocardial hypertrophy (this underestimation is on the order of 3 mm Hg at a left ventricular end-diastolic pressure of 13 mm Hg; see footnote to table 2A). Finally, two patients had Lillehei-Kaster mechanical prostheses, which perform poorly with a significant postoperative resting gradient (> 40 mm Hg). Greater intermediate and late improvement might have been seen had these patients received other prostheses with less residual prosthetic stenosis.

In summary, the remodeling of left ventricular volume, mass, and shape is a process that may proceed up to almost a decade after correction of the valvular abnormality and excess load that incited it. Even late after aortic valve surgery in our patients there remained evidence of the prior hypertrophic process. In patients with a pattern of concentric hypertrophy (i.e., preoperative aortic stenosis), left ventricular end-diastolic wall thickness and the ratio of left ventricular wall thickness to chamber radius decreased over time, although it remained abnormally elevated even at the late study. In patients with a pattern of eccentric hypertrophy (i.e., preoperative aortic insufficiency), left ventricular wall thickness also progressively decreased over time, and remained elevated above normal even at the late study. However, at the intermediate study the ratio of left ventricular wall thickness to chamber dimension had increased to a more concentric pattern, suggesting that the resolution of chamber volumetric adaptations occurred faster than those of mass. The effect on these observations of a persistent, if small, relative stenosis from the prosthetic valve orifice must be considered. These changes were accompanied by progressive improvements in resting hemodynamics and thus suggest that the improvement in load present-
ed by the prosthetic valve and the resolution of myocardial hypertrophy may lead to improved myocardial performance. Finally, our results showed no evidence of late deterioration of myocardial function as a late effect of the primary hemodynamic insult or as a result of the regression of myocardial hypertrophy.

We thank Dr. Bernard J. Ransil for his help with the statistical analysis.

References

Time course of regression of left ventricular hypertrophy after aortic valve replacement.
E S Monrad, O M Hess, T Murakami, H Nonogi, W J Corin and H P Krayenbuehl

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