Left Ventricular Function in Chronic Mitral Regurgitation: Preoperative and Postoperative Comparison

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Objectives. The present study was designed to evaluate the effects of surgical procedure on left ventricular systolic and diastolic function in patients with mitral regurgitation.

Background. Left ventricular systolic function has been shown to decline after operation in patients with chronic mitral regurgitation.

Methods. Using simultaneous cineangiography and left ventricular micromanometry, we evaluated left ventricular systolic and diastolic function in 14 patients with chronic mitral regurgitation both preoperatively and at an average of 22 months after operation. Eight patients underwent mitral valve reconstruction, and six had a valve replacement with interruption of the chordae tendineae. We compared these patients with 10 control subjects.

Results. Preoperatively, patients with mitral regurgitation demonstrated normal global and regional left ventricular systolic function. Peak rate of diastolic filling was increased (p < 0.01), and passive chamber stiffness was decreased, compared with that in control subjects (p < 0.01), and there was normal myocardial stiffness. Postoperatively, systolic and diastolic function returned to normal in patients undergoing mitral valve reconstruction. In contrast, global systolic function was depressed in patients after valve replacement (p < 0.05), with regional dysfunction in the area of papillary muscle attachment (p < 0.01). Diastolic function was depressed in this group, with a prolonged time constant of pressure decay (p < 0.01) and a depressed rate of early diastolic filling and strain rate (p < 0.05). Passive elastic stiffness was within the normal range in all postoperative patients.

Conclusions. The type of operation performed to correct chronic mitral regurgitation has an important effect on postoperative left ventricular function. Systolic and diastolic function are preserved after mitral valve reconstruction. Mitral valve replacement with chordal interruption is associated with global and regional systolic dysfunction and early diastolic filling and relaxation abnormalities.

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In chronic mitral regurgitation, the left ventricle responds to the increased filling volume with alterations in ventricular size and shape. The ventricle undergoes eccentric hypertrophy, with a greater increase in volume than mass, whereas the chamber becomes more spheric (1–3). In association with the geometric changes, passive chamber stiffness decreases (3). Passive myocardial stiffness remains normal in patients with preserved ejection performance but is increased in those with systolic dysfunction (3). Preoperative left ventricular systolic function is typically preserved in a large proportion of patients (4–6).

Surgical correction of the volume overload state has a major impact on left ventricular mechanics. Postoperatively, there is regression of left ventricular hypertrophy, with a decrease in volume and muscle mass (5,7–10). Valve reconstruction appears to preserve left ventricular systolic function, whereas valve replacement with severing the chordae tendineae is associated with depressed systolic performance (5,6,8,10,11). The mechanism of the postoperative decrease in ejection fraction after valve replacement has not been definitively established (7,8,10,12). Few clinical studies have examined the effect of operation and changes in ventricular shape on postoperative diastolic function (11,13).

Accordingly, the present study was designed to evaluate preoperative and postoperative left ventricular function in patients with chronic mitral regurgitation. We compared the effect of mitral valve reconstruction and replacement on postoperative systolic and diastolic function, relating hypertrophy and geometry with ventricular performance.

Methods

Patients. The present study is based on the retrospective analysis of left ventricular function in patients who underwent cardiac catheterization, both preoperatively and after surgical correction of chronic mitral regurgitation at the Cardiology Department of the University of Zurich between January 1979 and December 1989. The patients included in this study were those who specifically agreed to undergo a second catheterization after operation. Patients with any of the following criteria were excluded: mitral stenosis (mean gradient >5 mm Hg),
more than trace aortic regurgitation, aortic stenosis (mean gradient >5 mm Hg), significant (>70% stenosis) coronary artery disease, QRS duration >0.10 s or ventricular asynergy. Fourteen patients (12 men, 2 women) with isolated mitral regurgitation were evaluated both preoperatively and postoperatively. Twelve were in normal sinus rhythm, and two were in atrial fibrillation. Functional class evaluation by New York Heart Association assignment was made from ambulatory clinic records; all patients were preoperatively symptomatic (functional class II or higher). Ten patients who had been evaluated for symptoms of chest pain and found to have no significant coronary artery or valvular heart disease served as control subjects. Preoperatively, the medication of patients included digoxin (n = 12), a diuretic agent (n = 12) and an afterload-reducing agent (n = 4). Postoperative medications of patients included digoxin (n = 7), a diuretic agent (n = 9) and beta-adrenergic blocking agents (n = 3); three patients were taking no medication after operation.

**Catheterization.** Patients received chloradiazepoxide (10 mg orally) 1 h before the procedure. Cardiovascular medications were withheld 12 to 24 h before the procedure. Right and left heart catheterization were performed in all patients. Biplane contrast left ventriculography was performed in the right anterior oblique (30°) and left anterior oblique (60°) projections using 35-mm film at a rate of 50 frames/s. Preoperatively, left ventricular pressure was measured simultaneously with ventriculography using a 7F micromanometer catheter (Millar Instruments) introduced transseptally into the left ventricle through an 11.5F Brockenbrough catheter. Postoperatively, an 8F Millar pigtail catheter was inserted retrogradely into the left ventricle across the aortic valve. All pressures were recorded at a paper speed of 250 mm/s (model VR-16, Electronics for Medicine) with the electrocardiogram, first derivative of left ventricular pressure (dP/dt) and a cineangiographic time marker. Cardiac output was determined by the Fick method and indexed to body surface area; regurgitant fraction was calculated by the angiographic Fick method. Coronary angiography was performed in all 14 patients preoperatively and in 10 patients postoperatively.

**Data analysis.** Data were collected from beats with simultaneous cineangiography and micromanometry, using the first beat providing adequate opacification of the left ventricle. Extrasystolic and postextrasystolic beats were excluded. In the two patients with atrial fibrillation, two representative beats were evaluated, and the results were averaged. Heart rate was derived from the right heart study done before left ventricular angiography.

Left ventricular volume was calculated on a frame-by-frame basis using the area-length method (14). The left ventricular long axis was the longest axis from either the right or left anterior oblique projection. The short axis was calculated as a geometric mean of the derived short axes in the right and left anterior oblique views. The dimensional and volume data were filtered using a moving average technique (4). The first time derivative of circumference (dC/dt [cm/s]) and strain (de/dt [s⁻¹]) were calculated as previously described (3).

Left ventricular pressure tracings were digitized for an entire cardiac cycle using an electronic digitizer (Numonics Corp.) interfaced to a PDP 11/34 computer (Digital Equipment Corp.). Left ventricular pressure was measured every 5.0 ms. Pressure and volume data were matched at 20-ms intervals using the digital time indicated on each angiographic frame with the corresponding time marks on the pressure recordings (3).

End-diastole was defined as the cineangiographic frame occurring immediately before the systolic upstroke of the simultaneously inscribed dP/dt. Aortic valve opening was defined as the first frame after end-diastole in which the intraventricular pressure exceeded aortic pressure. Aortic valve closure was defined as the last frame in systole in which intraventricular pressure was greater than incisural pressure. Simultaneous aortic and ventricular pressures were available in all patients preoperatively. Postoperatively, aortic pressure was determined from the pressure recording obtained immediately before left ventriculography. The ejection period was defined as the time from aortic valve opening to aortic valve closure for controls and postoperative patients and from end-diastole to minimal volume in preoperative patients (4). End-systole was defined in all patients and control subjects as the frame of aortic valve closure.

End-diastolic wall thickness was determined by the method of Rackley et al. (15). Left ventricular wall thickness for each subsequent frame throughout the cardiac cycle was calculated using the method of Hugenholz et al. (16). Midwall circumferential stress was calculated for each frame using Mirsky's thick wall model (17):

\[
\text{Midwall stress} (\text{kdynes/cm}^2) = \frac{P}{h} (1 - \frac{b}{2a} - \frac{h^2}{2a^2}) 1.322,
\]

where \(P\) = ventricular pressure (mm Hg); \(h\) = left ventricular wall thickness (cm); \(a\) = midwall semimajor axis (cm); and \(b\) = midwall semiminor axis (cm). Left ventricular eccentricity was calculated as follows (18):

\[
\text{Eccentricity} = \sqrt{\frac{a^2 - b^2}{a}}.
\]

**Determination of systolic and diastolic function.** Ejection fraction (%) was calculated as [(End-diastolic volume − Minimal volume)/End-diastolic volume] × 100. Peak systolic stress was defined as the maximal value of circumferential stress in each beat. Mean systolic stress was determined as the arithmetic mean of the calculated stress of each frame during the ejection period: from aortic valve opening to aortic valve closure in control subjects and postoperative mitral regurgitation patients and from end-diastole to minimum ventricular volume in preoperative patients (4). End-systolic stress was defined as midwall circumferential stress at aortic valve closure.

Regional dimensions were automatically determined by computer from the left ventricular tracings in the right anterior oblique view (19). Using the end-diastolic and end-systolic frames, regional function was represented as fractional short-
enning in each of six segments (inferior-base, inferior-middle, inferior-apex, superior-base, superior-middle, superior-apex) from end-diastole to aortic valve closure in postoperative patients and to minimal volume in preoperative patients.

Left ventricular isovolumic pressure decay was evaluated in controls and postoperative patients by calculating the time constant of relaxation (τ). This constant was calculated using left ventricular pressure (P [mm Hg]) (20):

\[
\frac{dP}{dt} = \frac{-1}{\tau} (P - P_B),
\]

from maximal \(-dP/dt\) to a pressure 5 mm Hg above left ventricular end-diastolic pressure (P_B = baseline pressure).

Passive diastolic function was assessed from minimal ventricular pressure until end-diastole. An elastic model with shifting asymptote was used to evaluate left ventricular chamber properties, mathematically represented as follows (3):

\[
dP/dV = b(P - c*),
\]

where P = left ventricular pressure (mm Hg); b = constant of left ventricular chamber stiffness (ml⁻¹); V = left ventricular volume (ml); c* = asymptote of the exponential pressure-volume relation (mm Hg); and dP/dV = instantaneous chamber stiffness (mm Hg/ml). Values of c* were iterated until the closest linear curve fit was obtained (3).

Diastolic myocardial stiffness was determined assuming an elastic stress-strain relation with a shifting asymptote as previously reported (3). Briefly, a reference midwall circumference (L₁ [cm]) was determined from the stress-length relation at a common wall stress of 1 kdyne/cm². This circumference was used to calculate wall strain (ε) using the Lagrangian definition. Diastolic myocardial stiffness was then calculated from the elastic stress-strain relation with shifting asymptote,

\[
\frac{dS}{de} = \beta(S - c),
\]

where S = left ventricular circumferential stress (kdyne/cm²), \( \beta \) = constant of normalized myocardial stiffness; e = Lagrangian strain; c = asymptote of the elastic stress-strain relation (kdyne/cm²); and dS/de = instantaneous myocardial stiffness (kdyne/cm²). The values of c were iterated by the computer to establish the best linear curve fit (3).

To compare the relative end-diastolic sarcomere length (L), end-diastolic strain was calculated in each patient as EDC/L₁, where EDC = end-diastolic midwall circumference (cm). The maximal rate of midwall circumferential lengthening during early diastole (dC/dt_max [cm/s]) was calculated for each patient. The maximal strain rate (dC/dt_max [s⁻¹]) was the greatest value of (dL/dt/L₁) during diastole.

Statistics. Comparison of the control subjects and preoperative and postoperative data for the respective surgical groups was performed by a two-factor analysis of variance with repeated measures (SAS statistical package). Correlation between variables were determined using a linear least-squares method. Differences with a statistical probability <0.05 using a two-tailed approach were considered significant.

Results

Clinical data. All patients were symptomatic preoperatively, and nearly all were taking digoxin and a diuretic agent before catheterization. The cause of mitral regurgitation included myxomatous degeneration in 10 patients, ruptured chordae in 3 and endocarditis in 1. Twelve patients were in sinus rhythm both preoperatively and postoperatively, and two were in atrial fibrillation. Patients were more symptomatic before (mean functional class 2.4) than after operation (mean functional class 1.3; p < 0.01). Patients were significantly older than control subjects (mean ±SD age 42 ± 13 vs. 56 ± 5 years, p < 0.01) and underwent repeat cardiac catheterization at a mean of 22 months (range 6 to 62) after operation.

Eight patients underwent mitral valve reconstruction, six of whom had insertion of a Carpentier ring. Of the six patients who underwent mitral valve replacement, five received a St. Jude valve, and one patient received a Björk–Shiley valve. The chordae tendineae were severed in all patients undergoing valve replacement. A perioperative myocardial infarction was not documented in any patient. Mean bypass time was 45 ± 10 min in patients undergoing reconstruction and 47 ± 18 min in valve replacement (p = NS). Mean body surface area was similar among control subjects (1.71 ± 0.13 m²) and patients with mitral regurgitation preoperatively (1.81 ± 0.15 m²) and postoperatively (1.82 ± 0.15 m²).

Hemodynamic and cineangiographic data. Heart rate was similar among control subjects and preoperative and postoperative patients and did not change postoperatively (Table 1). Left ventricular end-diastolic pressure was increased in patients with mitral regurgitation preoperatively compared with control subjects and decreased to normal postoperatively. Left ventricular peak systolic, end-systolic and mean right atrial pressures were similar among patients and control subjects. Left atrial wave pressure was increased preoperatively and declined postoperatively. Left ventricular end-diastolic volume index and short-axis dimension were larger in patients than in control subjects and larger preoperatively than postoperatively. Ejection fraction was normal preoperatively and decreased after valve replacement. Ejection time was significantly prolonged in preoperative patients and returned to normal postoperatively. Regurgitant fraction was significantly reduced by surgical correction. End-diastolic wall thickness was normal in patients with mitral regurgitation, both preoperatively and postoperatively. Left ventricular muscle mass index was greater in patients than control subjects and greater preoperatively than postoperatively. Cardiac index was depressed preoperatively and increased postoperatively. It was lower than at baseline in patients after valve replacement.

Systolic function and stress data. End-diastolic and peak systolic stress were elevated in patients preoperatively and significantly declined to normal after operation (Table 2). End-systolic and mean systolic stress were similar in patients and in control subjects both preoperatively and postoperatively.

The afterload–shortening relation for control subjects and
Table 1. Hemodynamic and Cineangiographic Data

<table>
<thead>
<tr>
<th></th>
<th>Control Subjects (n = 10)</th>
<th>Pts With MV Reconstruction (n = 8)</th>
<th>Pts With MV Replacement (n = 6)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before</td>
<td>After</td>
<td>Before</td>
</tr>
<tr>
<td>HR</td>
<td>74 ± 13</td>
<td>64 ± 11</td>
<td>66 ± 12</td>
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<tr>
<td>EDP</td>
<td>11 ± 4</td>
<td>22 ± 4*</td>
<td>12 ± 5*</td>
</tr>
<tr>
<td>PSP</td>
<td>128 ± 13</td>
<td>125 ± 11</td>
<td>143 ± 22</td>
</tr>
<tr>
<td>ESP</td>
<td>105 ± 13</td>
<td>105 ± 11</td>
<td>104 ± 13</td>
</tr>
<tr>
<td>PCW</td>
<td>6 ± 2</td>
<td>17 ± 2*</td>
<td>10 ± 4</td>
</tr>
<tr>
<td>VWP</td>
<td>9 ± 2</td>
<td>37 ± 19*</td>
<td>12 ± 5*</td>
</tr>
<tr>
<td>ΔP</td>
<td>—</td>
<td>1.6 ± 1.9</td>
<td>—</td>
</tr>
<tr>
<td>RAP</td>
<td>2 ± 1</td>
<td>3 ± 1</td>
<td>4 ± 1</td>
</tr>
<tr>
<td>d</td>
<td>5.4 ± 0.6</td>
<td>8.2 ± 0.5*</td>
<td>6.6 ± 0.6*§</td>
</tr>
<tr>
<td>EDVI</td>
<td>82 ± 18</td>
<td>182 ± 30*</td>
<td>100 ± 16§</td>
</tr>
<tr>
<td>EF</td>
<td>65 ± 3</td>
<td>64 ± 5</td>
<td>61 ± 16</td>
</tr>
<tr>
<td>ET</td>
<td>260 ± 30</td>
<td>360 ± 30*</td>
<td>300 ± 40§</td>
</tr>
<tr>
<td>RF</td>
<td>—</td>
<td>60 ± 11*</td>
<td>5 ± 10§</td>
</tr>
<tr>
<td>h</td>
<td>0.81 ± 0.08</td>
<td>0.82 ± 0.04</td>
<td>0.84 ± 0.09</td>
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<td>LMMI</td>
<td>86 ± 15</td>
<td>128 ± 19*</td>
<td>95 ± 16§</td>
</tr>
<tr>
<td>CI</td>
<td>4.3 ± 0.7</td>
<td>3.0 ± 0.7*</td>
<td>3.4 ± 0.7</td>
</tr>
</tbody>
</table>

*p < 0.01, t < 0.05 versus control subjects. t < 0.05, t < 0.01 versus before operation. Data presented are mean value ± SD. CI = cardiac index (liters/min per m²); d = left ventricular end-diastolic short-axis dimension (cm); EDP (ESP) = end-diastolic (end-systolic) pressure (mm Hg); EDVI = end-diastolic volume index (ml/m²); EF = ejection fraction (%); ET = ejection time (ms); h = end-diastolic wall thickness (cm); HR = heart rate (beats/min); LMMI = left ventricular muscle mass index (g/m²); MV = mitral valve; PCW = mean pulmonary capillary wedge pressure (mm Hg); PSP = peak systolic pressure (mm Hg); Pts = patients; RAP = mean right atrial pressure (mm Hg); RF = mitral regurgitant fraction (%); VWP = left atrial v wave pressure (mm Hg); ΔP = diastolic pressure gradient.

Table 2. Stress Data

<table>
<thead>
<tr>
<th></th>
<th>Control Subjects (n = 10)</th>
<th>Pts With MV Reconstruction (n = 8)</th>
<th>Pts With MV Replacement (n = 6)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before</td>
<td>After</td>
<td>Before</td>
</tr>
<tr>
<td>EDS</td>
<td>36 ± 11</td>
<td>92 ± 15*</td>
<td>50 ± 16</td>
</tr>
<tr>
<td>PSS</td>
<td>336 ± 45</td>
<td>415 ± 35*</td>
<td>365 ± 52</td>
</tr>
<tr>
<td>ESS</td>
<td>163 ± 32</td>
<td>190 ± 32</td>
<td>188 ± 35</td>
</tr>
<tr>
<td>MSS</td>
<td>257 ± 30</td>
<td>307 ± 21</td>
<td>306 ± 49</td>
</tr>
</tbody>
</table>

*p < 0.01, t < 0.05 versus control subjects. t < 0.01, t < 0.05 versus before operation. Data presented are mean value ± SD. EDS = end-diastolic stress (kdynes/cm²); ESS = end-systolic stress (kdynes/cm²); ESS = mean systolic stress (kdynes/cm²); PSS = peak systolic stress (kdynes/cm²); other abbreviations as in Table 1.
Figure 1. Afterload–shortening relation for the 10 control subjects and the 95% confidence band of the control group. Patients are classified as those undergoing mitral valve reconstruction and replacement, both preoperatively and postoperatively. Afterload is represented as mean systolic stress from aortic valve opening to aortic valve closure in control subjects and postoperative patients and from end-diastole to minimal ventricular volume in preoperative patients. + = control subjects; • (○) = preoperative (postoperative) patients with mitral valve reconstruction; Δ (■) preoperative (postoperative) patients with mitral valve replacement.

creased to normal postoperatively. The time constant of relaxation was prolonged in those undergoing valve replacement compared with that in control subjects and patients with valve reconstruction.

The constant of myocardial stiffness was normal before and after operation in patients with mitral regurgitation both preoperatively and postoperatively (Table 3 and Fig. 5). The normalized length \( L_1 \) was significantly increased preoperatively; it decreased postoperatively but remained greater than that in control subjects. End-diastolic strain was similar among control subjects and patients with mitral regurgitation and did not change after operation. Among patients, peak diastolic strain rate was increased preoperatively and was depressed postoperatively after valve replacement.

Discussion

The results of this retrospective study indicate that in patients with chronic mitral regurgitation, normal preoperative left ventricular systolic function declines in those undergoing valve replacement but is preserved by valve reconstruction (Fig. 1). Among patients with mitral valve replacement, regional systolic function was significantly impaired in the segment of papillary muscle insertion (Fig. 2). As evaluated by mean systolic stress, left ventricular afterload was similar to normal in preoperative patients. The postoperative decline in systolic function among patients with mitral valve replacement was associated with prolonged relaxation and a decrease in the rate of early diastolic filling. For patients in both surgical groups, we found that preoperative left ventricular passive chamber stiffness was reduced before surgery and returned to normal postoperatively, whereas myocardial stiffness remained normal.

Systolic function. Evaluation of preoperative systolic function in chronic mitral regurgitation has provided conflicting results. Eckberg et al. (21) found a decrease in contractility among patients compared with control subjects, but Cohn et al. (22) found normal function. A study from this laboratory (4) suggested that patients with a normal ejection fraction may have normal systolic performance, whereas those with a depressed ejection fraction showed depressed left ventricular function. Ejection fraction cannot be used alone to evaluate systolic function in patients with chronic mitral regurgitation. The afterload–shortening relation has been used clinically to examine contractility (4,18,21,22). There is an inverse relation between ejection and afterload, although this clinical relation...
Table 3. Diastolic Function Data

<table>
<thead>
<tr>
<th></th>
<th>Control Subjects</th>
<th>Pts With MV Reconstruction</th>
<th>Pts With MV Replacement</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before</td>
<td>After</td>
<td>Before</td>
</tr>
<tr>
<td>b</td>
<td>0.031 ± 0.008</td>
<td>0.016 ± 0.004*</td>
<td>0.025 ± 0.013</td>
</tr>
<tr>
<td>dCdmin</td>
<td>23.5 ± 3.9</td>
<td>39.3 ± 8.9†</td>
<td>23.6 ± 3.7†</td>
</tr>
<tr>
<td>LVV/LMM</td>
<td>0.96 ± 0.11</td>
<td>1.45 ± 0.8</td>
<td>1.67 ± 0.13</td>
</tr>
<tr>
<td>ECC</td>
<td>1.8 ± 0.2</td>
<td>1.3 ± 0.2*</td>
<td>1.6 ± 0.3</td>
</tr>
<tr>
<td>r</td>
<td>50 ± 15</td>
<td>—</td>
<td>56 ± 13</td>
</tr>
<tr>
<td>β</td>
<td>13.7 ± 6.4</td>
<td>16.1 ± 4.1</td>
<td>17.2 ± 9.1</td>
</tr>
<tr>
<td>L1</td>
<td>15.4 ± 2.1</td>
<td>23.0 ± 1.9†</td>
<td>18.0 ± 2.6‡</td>
</tr>
<tr>
<td>StrainEnd</td>
<td>1.29 ± 0.17</td>
<td>1.26 ± 0.10</td>
<td>1.30 ± 0.19</td>
</tr>
<tr>
<td>de/dtmax</td>
<td>1.58 ± 0.23</td>
<td>1.86 ± 0.39</td>
<td>1.31 ± 0.13</td>
</tr>
</tbody>
</table>

*p < 0.05, †p < 0.01 versus control subjects. ‡p < 0.05 versus before operation. §p < 0.01 versus after reconstruction. Data presented are mean value ± SD. b = constant of chamber stiffness (mt ~); dCdmin = maximal rate of circumferential lengthening (cm/s); de/dtmax = maximal diastolic strain rate (s−1); ECC = left ventricular chamber eccentricity at end-diastole; L1 = myocardial length at a left ventricular stress of 1 kdyne/cm2 (cm); LVV/LMM = ratio of left ventricular end-diastolic volume to mass (ml/g); β = constant of normalized myocardial stiffness; r = time constant of left ventricular isovolumic pressure decay (ms).

is preload dependent (23). In the present study, normal preoperative contractility was found in patients with a normal ejection fraction. After surgical correction of the mitral valve, systolic stress (peak, mean and end-diastolic stress) was normalized. Despite normalization of the afterload conditions, left ventricular ejection fraction decreased in the replacement group (24). In two of the six patients with valve replacement, afterload increased after operation, whereas in the other four patients afterload decreased. Ejection fraction decreased in the majority of patients (five of six) irrespective of changes in afterload. Thus, other mechanisms than changes in afterload have to be assumed to be responsible for the deterioration of left ventricular systolic function (see later).

Preservation of the chordae tendineae and papillary muscles during mitral valve surgery is necessary to maintain postoperative regional and global left ventricular function. Bonchek et al. (7) demonstrated preserved ejection fraction after mitral valve repair. David et al. (5) found a significant decline in postoperative ejection fraction after conventional valve replacement but preservation of function with retained chordae and papillary muscles. After surgical interruption, there was depression of regional and global elastance with dyssynergy of contraction (25). Dyskinesia was also demonstrated at the site of the papillary muscle insertion (12,25). This group found that severing either the anterior or posterior chordae is detrimental to global systolic function (26). Furthermore, in an animal preparation, Sarris et al. (27) demonstrated...
There was an increase in the end-diastolic volume, but the ratio of volume/volume may also be mediated by a rearrangement of fibers and may have reduced elastic recoil and contributed to depression of early diastolic filling.

In patients with mitral regurgitation, preoperative diastolic function reflected the effects of chronic volume overload. In the setting of an elevated preload (end-diastolic stress), there was an increase in the end-diastolic volume, ratio of volume/mass and rates of circumferential lengthening and strain. In addition, left ventricular eccentricity declined and chamber stiffness decreased, although myocardial stiffness was normal.

Patients undergoing mitral valve replacement demonstrated normal postoperative diastolic function. Chamber stiffness was normalized in both groups (Fig. 4) after correction of chronic volume overload caused by the decrease in chamber size and the normalization of left ventricular geometry. However, myocardial stiffness remained unchanged preoperatively and postoperatively in both groups (Fig. 5).

In contrast, postoperative abnormalities of diastolic function were apparent in patients after valve replacement. These patients demonstrated prolonged relaxation and a reduced rate of early diastolic filling. In dogs, the chordae tendineae were severed, and systolic asynergy and regional systolic abnormalities were found in the region of papillary muscle insertion (25-27). The increase in systolic heterogeneity is likely to have increased left ventricular nonuniformity during pressure decay, thereby causing an impairment of relaxation (31). Systolic heterogeneity has been shown to prolong relaxation and reduce early diastolic filling (32). Zile et al. (33) found that enhanced preoperative diastolic function returned to normal after mitral valve replacement. In contrast to the present results, systolic function remained normal after mitral valve replacement in this canine preparation (34). Although this group used a "chronic" animal preparation, mitral regurgitation was only present in these animals for 3 months (34); clinically, chronic mitral regurgitation is usually present for decades. The marked difference in duration of mitral regurgitation may explain the contrasting results between the experiment and the present study.

Because of the absence of an isovolumic period preoperatively, it was not possible to calculate the time constant of pressure relaxation in patients before surgery. Preoperatively, patients with chronic aortic insufficiency demonstrated left ventricular hypertrophy, which is associated with prolonged relaxation (20). In the present study, normal postoperative relaxation in the reconstruction group may have been the result of the decrease in left ventricular mass after surgery. However, without knowledge of the preoperative time constant, we cannot describe the preoperative to postoperative evolution of relaxation in these patients.

Left atrial driving pressure and left ventricular relaxation, elastic recoil and passive compliance determine the early diastolic filling rate (35). Left atrial driving pressure and left ventricular passive compliance were similar in the two groups. Prolonged relaxation may have been a major factor causing the decrease in early diastolic filling rate (35-37). Although recoil has not been systematically assessed in mitral regurgitation, the decreased ejection fraction in patients with valve replacement may have reduced elastic recoil and contributed to depression of early diastolic filling.

The observation of normal preoperative myocardial stiffness is in consonance with previous evaluations of diastolic function (3,38,39). Normal stiffness was present after operation in patients undergoing both mitral valve reconstruction and replacement despite a decrease in systolic function in the latter group. The dissociation of postoperative ejection performance and myocardial stiffness is in apparent contradiction to observations that these preoperative factors are inversely related (3,39). In a study of patients with aortic insufficiency, there was no change in diastolic myocardial function after valve replace-

References


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