Durability of mitral valve repair in Barlow disease versus fibroelastic deficiency

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Objective: Durability assessment of mitral valve repair for degenerative valve incompetence is limited to reoperation as a primary indicator and valve-related risk factors for late death as a secondary indicator. We assessed serial echocardiographic follow-up of valve function as an indicator of the durability of mitral valve repair.

Methods and Results: In 348 patients having undergone mitral valve repair for degenerative valve incompetence, clinical outcome was excellent: 10 years after repair, survival was 80.1% and freedom from reoperation 94.4%. However, freedom from mitral regurgitation ($>2/4$), 98.7% at 1 month, decreased to 82.2% at 5 years and 64.9% at 10 years. The linearized recurrence rate of mitral regurgitation ($>2/4$) was 3.2% per year. Recurrence rate was higher in patients with Barlow disease (6.0%) and lower in those with fibroelastic deficiency (2.6%) ($P = .01$). Performing chordal shortening, the nonuse of sliding plasty and the nonuse of an annuloplasty ring were determined to be factors predicting recurrence of mitral regurgitation. In reconstructions avoiding these risk factors, recurrence rate decreased to 2.4%. There was no difference between Barlow disease and fibroelastic deficiency: 2.9% versus 2.2% ($P > .05$). Recurrent regurgitation is characterized by leaflet prolapse, thickening, and calcification.

Conclusion: When optimal surgical techniques are used, the residual recurrence rate of mitral valve regurgitation remains between 2% and 3% per year and is related to progressive degeneration of the chordae and the leaflets. Long-term results of mitral valve repair in Barlow disease are essentially the same as in fibroelastic deficiency.

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In a recent report, we¹ demonstrated that the linearized recurrence rate of recurrent mitral regurgitation greater than 2/4 after surgical repair was 3.7% per year in patients with degenerative valve disease. Inadequate surgical techniques could only partially explain this recurrence of regurgitation. In patients receiving optimal repair techniques, the recurrence rate of regurgitation dropped to 2.5% per year. We suggested that the progression of the degenerative disease of the valve was responsible for this small but constant recurrence rate. Recurrence rates of regurgitation were not previously reported because most studies so far were focusing on survival and reoperation rates, which were found to be excellent.^{2–9} In our study, survival was also high (80.1% at 10 years), as was freedom from reoperation (94% at 10 years). Diseased valves can be successfully repaired by a variety of surgical techniques, and our results confirmed this high immediate operative success: 98.3% of the patients was free of significant ($>2/4$) regurgitation 1 month postoperatively as defined by echocardiography.¹ However, as recent studies have demonstrated, myxomatous valve leaflets are structurally, biochemically, physically, and

Abbreviations and Acronyms

NYHA = New York Heart Association
 SAM = systolic anterior motion

mechanically abnormal¹⁰ and a certain progression of the disease can be expected after repair. Therefore, we were not surprised to find that 7 years postoperatively only 71% of the patients were still free of greater than 2/4 regurgitation.¹ This finding was confirmed by David and associates⁹ in a more recent study. They found that 12 years postoperatively, only 73% of the patients were free from moderate or severe mitral insufficiency of more than 3/4, a result is even worse than that of our study.

In patients with degenerative mitral valve disease, Carpentier and colleagues¹¹ distinguished two different forms: Barlow disease and fibroelastic degeneration. These two diseases may not have the same outcome, because Barlow disease is a more generalized degeneration of the valve.

The goal of the present study was to analyze the recurrence of mitral regurgitation in terms of incidence and degree in a series of patients undergoing mitral valve repair for degenerative disease, with special reference to Barlow disease. All patients were operated on in the same institution, by the same surgeon, with standard repair techniques.

Materials and Methods**Patient Population**

During the period from July 1989 through December 2005, 1596 mitral valve operations were performed by the same surgeon (W.F.), 945 replacements and 651 repairs. Four hundred fifty-two patients had degenerative mitral valve disease: 104 underwent replacement and 348 (77%) repair. The repair rate increased from 43% in 1989 to 95% in 2005. The indications to prefer mitral valve replacement above repair were as follows: Barlow disease (62%), nonsinus rhythm (41%), anterior mitral leaflet involvement (30%), severe calcification of the mitral annulus (20%), concomitant aortic valve replacement (12%), and systolic anterior motion (SAM) (3%).

Mitral Valve Characteristics

On the basis of the preoperative echocardiogram and a detailed drawing of the operative findings, valve pathologic status was identified and coded at the end of the operation. We differentiated between Barlow disease and fibroelastic deficiency on the basis of gross appearance of the valve, according to Carpentier,¹¹ Fornes,¹² and their associates. The following criteria were used to classify the patients into these two groups:

- Barlow disease: myxoid appearance of the whole valve with excess tissue and a dilated annulus.
- Fibroelastic deficiency: restriction of thickening to the prolapsed area(s), the remaining valve tissue being more transparent, not thickened, without excess tissue, and the annulus being dilated or not.

TABLE 1. Demographic data and mitral valve characteristics

	Total	Barlow	FED
No. of patients (%)	348	83 (24%)	265 (76%)
Age, y (range)	63 (29–86)	61 (29–79)	64 (31–86)
Male sex	247	55 (62%)	192 (61%)
Cardiac rhythm			
Sinus	268 (77%)	61 (73%)	207 (78%)
Atrial fibrillation	75 (21.5%)	22 (27%)	53 (20%)
Other	5 (1.5%)	0 (0%)	5 (2%)
Leaflet involvement			
Anterior (%)	18 (5%)	2 (2%)	16 (6%)
Posterior (%)	220 (63%)	14 (17%)	206 (78%)
Bileaflet (%)	110 (32%)	67 (81%)	43 (16%)
Leaflet condition			
Prolapse	321 (92%)	79 (94%)	242 (91%)
Calcifications	6 (1.7%)	0 (0%)	6 (2%)
Normal aspect	8 (2.3%)	0 (0%)	8 (3%)
Cleft formations	14 (4%)	5 (6%)	9 (3%)
Chordae			
Rupture	241 (69%)	34 (41%)	207 (78%)
Elongations	57 (16%)	31 (37%)	26 (10%)
Thickening	5 (2%)	3 (4%)	2 (1%)
Normal aspect	45 (13%)	15 (18%)	30 (11%)
Annulus			
Dilated, not calcified	320 (92%)	73 (88%)	247 (93%)
Dilated, calcified	24 (7%)	10 (12%)	14 (5%)
Not dilated	4 (1%)	0 (0%)	4 (2%)

FED, Fibroelastic deficiency.

Eighty-three patients were classified as having Barlow disease (24%) and 265 (76%) as having fibroelastic deficiency. Demographic data and mitral valve characteristics are listed in Table 1.

Surgical Techniques

Overall in our institution, the average repair rate was 77%, and this rate increased continuously from 43% in 1989 to 95% in 2005. The repair rate for Barlow disease was 33% in 1989, 33% in 1995, 77% in 2000, and 85% in 2005. For fibroelastic deficiency it was 50% in 1989, 88% in 1995, 88% in 2000, and 100% in 2005. For repair, classic techniques described by Carpentier and others were used. Prolapse of the posterior leaflet was preferentially corrected by quadrangular resection when the prolapsing segment was billowing and in most cases combined with chordal rupture or elongation, to remove this diseased part of the valve as much as possible. When the remaining part of the posterior leaflet showed excess tissue (as in Barlow disease), the resection was combined with a sliding leaflet procedure including two triangular resections of the remnants of the nonprolapsing posterior leaflet. When there was no excess tissue in the remaining portion of the posterior leaflet (as in fibroelastic deficiency), a sliding procedure was performed, but without removal of tissue of the remaining posterior leaflet. The size of the annulus was adjusted by mattress sutures passed through the annulus, which is a procedure focused rather on an adequate reduction of the dilated annulus and is altered from Carpentier's described techniques. This was done to obtain an evenly distributed reduction of the

size of the posterior annulus without deformation of the normal parts of the posterior leaflet. When the annulus showed no or mild dilatation, the prolapse was preferentially corrected by artificial chordae. Prolapse of the anterior leaflet was never corrected by leaflet segment resection but by chordal transfer or artificial chorda implantation. Prolapse induced by chordal elongation was treated by chordal shortening. This was done by either chordal burring or papillary muscle repositioning. The latter procedure was mostly used for anterior leaflet prolapse or commissural prolapse of both leaflets. Mitral ring annuloplasty was performed in most cases to complete the repair, except in extreme situations either when the annulus was not or only slightly dilated or when the anterior leaflet was so large that SAM could be expected after ring annuloplasty.

In every patient, the specific surgical repair techniques used were identified and coded at the end of the operation: intervention at the leaflet (none, quadrangular resection, triangular resection, plication, cleft closure), intervention at the annulus (none, sliding plasty, plication, decalcification), at the chordae (none, shortening, transposition, artificial chordae), at the papillary muscles (none, shortening), and the placement of an annuloplasty ring (yes or no). The frequency with which these different techniques were used is listed in Table 2.

Follow-up

Clinical and echocardiographic follow-up were performed shortly before hospital discharge, at 1 month, and then every 6 months by the referring cardiologist. Survival, reoperation, cerebrovascular

accidents, bleeding complications, anticoagulation therapy, New York Heart Association (NYHA) function class, and cardiac rhythm were registered. On the echocardiogram, mitral regurgitation was classified from grade 1 to 4.

Statistical Analysis

The Cox proportional hazards methods were used to analyze the data on recurrence of mitral regurgitation in time. For survival and follow-up of events, Kaplan–Meier techniques were used with log–rank testing. For recurrence of mitral regurgitation, a classic Kaplan–Meier technique was used with the first echocardiographic follow-up date demonstrating the recurrence of regurgitation as date of the event. Since the mitral regurgitation did in fact recur between the last echocardiogram without regurgitation (or the date of the operation if the echocardiogram before hospital discharge showed mitral regurgitation) and the first echocardiogram with regurgitation, an interval-censored survival curve using the Turnbull algorithm

TABLE 2. Surgical data in mitral valve repair

	Total	Barlow	FED
MV repair			
Segmental resection leaflet	274	59 (71%)	215 (81%)
Other leaflet intervention (patch, plicature, cleft suture)	13	4 (5%)	9 (3%)
Chordal shortening	22	12 (15%)	10 (4%)
Chordal transfer	27	5 (6%)	22 (8%)
Chordal replacement	48	38 (46%)	10 (15%)
with PTFE sutures			
Decalcification and reconstruction of mitral annulus	8	3 (4%)	5 (2%)
Mitral annulus reduction			
By sliding leaflet technique	243	48 (58%)	195 (74%)
By annulus plication	17	6 (7%)	11 (4%)
Mitral ring annuloplasty			
None	11	4 (5%)	7 (3%)
Rigid Carpentier ring	326	79 (95%)	247 (93%)
Flexible ring	11	0 (0%)	11 (4%)
Ring size			
Smaller than 34		32 (39%)	223 (84%)
34 or larger		51 (61%)	42 (16%)
Concomitant cardiac surgery			
CABG	74	10 (12%)	64 (24%)
Aortic valve	9	0 (0%)	9 (3%)
Tricuspid valve	30	6 (7%)	24 (8%)
Redo surgery	2	0 (0%)	2 (1%)

FED, Fibroelastic deficiency; MV, mitral valve; PTFE, polytetrafluoroethylene; CABG, coronary artery bypass grafting.

Figure 1. A, Survival of the whole patient group. B, Freedom from reoperation for the whole patient group and for the two subgroups (fibroelastic deficiency and Barlow disease; $P = .002$).

was constructed additionally. For plotting the nonparametric maximum likelihood estimate on the basis of interval-censored data, the mass was always placed at the at the rightmost limit of the interval.

Results

Survival and Reoperation Rate

Hospital mortality was 1.6%. Survival at 5 years was $93.1\% \pm 1.5\%$ and $80.1\% \pm 3.7\%$ at 10 years (Figure 1, A). Survival was identical between patients having Barlow disease and patients having fibroelastic deficiency ($P > .05$). Survival did not differ significantly between patients with or without associated coronary artery bypass graft procedures ($P > .05$). Freedom from reoperation at 5 years was $95.4\% \pm 1.4\%$ and $94.4\% \pm 1.6\%$ at 10 years for all patients (Figure 1, B). Freedom from reoperation for fibroelastic deficiency was better (96.6% at 10 years) than for Barlow disease (86.1% at 10 years) ($P = .002$).

Clinical Outcome and Morbidity

No patient was lost to clinical follow-up. At least one echocardiographic examination was obtained in every patient. In 19 patients, the only echocardiogram was the discharge echocardiogram. The remaining patients received several follow-up echocardiograms with a mean of 4.6 echocardiograms per patient, ranging from 2 to 16. In total, the clinical follow-up represents 1836 patient-years, with a mean follow-up of 5.9 ± 3.4 years (range 0.5–15.3 years).

During the most recent postoperative follow-up period, NYHA class was recorded. Corresponding to the mean follow-up time of the entire population, 91.3% of the patients improved by at least one NYHA functional class. Seventy-five percent of the patients were in class I, 19% in class II, 5% in class III, and 1% in class IV. Postoperatively, 71% of the patients had sinus rhythm, 22% had atrial fibrillation, and 7% had a pacemaker. Anticoagulation therapy using coumarin was given during the follow-up period in 35% of the patients. Freedom from thromboembolic events and/or major anticoagulant-related bleeding was $93.5\% \pm 1.5\%$ at 5 years and $86.7\% \pm 2.7\%$ at 10 years for the whole group.

Immediate Surgical Result of Mitral Valve Repair

Operative success of the mitral valve repair was assessed by the echocardiographic examination of mitral valve function within the first 4 weeks postoperatively. At 1 month postoperatively, 98.7% of all patients had no or trivial mitral regurgitation (0/4 or 1/4). In patients having fibroelastic degeneration, this was 99.8% and in Barlow disease 97.1%. Suture dehiscence of the ring annuloplasty was encountered in 2 patients. Endocarditis of the repaired valve did not occur in this series.

Recurrence of Mitral Regurgitation

Postoperative echocardiography was performed serially at 6-month intervals. According to the classic Kaplan–Meier ap-

Figure 2. Freedom from recurrent mitral regurgitation (MR) of greater than 2/4 for the whole patient group and for the two subgroups (fibroelastic deficiency and Barlow disease; $P = .01$).

proach, freedom from failing repair (regurgitation $> 2/4$) was $98.7\% \pm 1.2\%$ at 1 month, $82.2\% \pm 3.7\%$ at 5 years, and $64.9\% \pm 5.6\%$ at 10 years (Figure 2). When the interval-censored Turnbull approach is used to calculate the freedom from recurrence of mitral incompetence, similar results are obtained. Freedom from failing repair is better in fibroelastic deficiency and worse in Barlow disease ($P = .01$). Remarkable is the constant rate of recurrence of mitral regurgitation after the first 6 months postoperatively. When the classic Kaplan–Meier curves are restricted to the period from 6 months until 7 years postoperatively, a linear regression can be made, allowing calculation of recurrence rates per year. Recurrence of mitral regurgitation of greater than 2/4 occurs at a constant rate of 3.2% per year for the whole patient group (Table 3). The linearized curves are shown for the patients with fibroelastic disease and Barlow disease separately in Figure 3, A and B.

TABLE 3. Linearized recurrence rates (in percent per year) for recurrent mitral regurgitation greater than 2/4

	Linearized recurrence rates for mitral regurgitation $> 2/4$		
	All patients	FED	Barlow
Overall	3.2	2.6	6.0
Without surgical risk	2.4	2.2	2.9
With surgical risk	4.7	3.6	14.9

FED, Fibroelastic deficiency. When only patients without surgical risk factors are included, the rates for fibroelastic disease and Barlow disease are similar.

Figure 3. Linearized recurrence curves for patients with fibroelastic deficiency (A) and patients with Barlow disease (B). Separate curves are shown for patients having surgical risk factors (nonuse of annuloplasty ring, nonuse of sliding plasty, or performance of chordal shortening) and for patients not having these factors. The concomitant slopes of these curves are shown in Table 3. MR, Mitral regurgitation.

Predictive Factors of Recurrent Mitral Valve Regurgitation After Repair

During the follow-up period, a subpopulation of 55 patients was identified as showing recurrence of significant mitral valve regurgitation ($>2/4$) and could be compared with the remaining 293 patients who did not. Cox proportional hazard analysis (Table 4) revealed that the subpopulation showing recurrence of regurgitation was mainly characterized by a higher incidence of Barlow disease ($P = .01$). Bileaflet mitral valve degeneration as such showed a trend toward higher recurrence of regurgitation ($P = .06$). The absence of ruptured chordae ($P = .01$) as well as the presence of elongated chordae ($P = .003$) predicts also recurrence of re-

TABLE 4. Factors predictive for mitral regurgitation greater than 2/4

	Cox analysis; mitral regurgitation $> 2/4$		
	All patients	FED	Barlow
Barlow	0.01	—	—
Bileaflet disease	0.06	0.6	0.7
Leaflet prolapse	0.04	0.05	0.9
Annulus dilatation	0.69	0.7	0.9
Annulus calcification	0.74	0.8	0.9
Chordae abnormal	0.75	0.9	0.02
Elongation (presence of)	0.003	0.8	0.02
Rupture (absence of)	0.01	0.1	0.4
Nonuse of quadrangular resection	0.06	0.02	0.6
Nonuse of sliding plasty	0.002	0.01	0.2
Nonuse of PTFE	0.6	0.2	0.02
Performing chordal shortening	0.01	0.9	0.04
No ring	0.001	0.04	0.004

FED, Fibroelastic deficiency; PTFE, polytetrafluoroethylene.

gurgitation. Specific surgical repair techniques had a predictive value for recurrence of regurgitation: the nonuse of a sliding annuloplasty ($P = .002$), the use of chordal shortening ($P = .01$), and the nonuse of an annuloplasty ring in general ($P = .001$) predict inferior outcome.

We separated the study populations into subgroups. The first bears the surgical risk factors as determined above (ie, absence of a stiff annuloplasty ring and/or presence of chordal shortening and/or no use of sliding annuloplasty) and the second does not. We could define two significantly different recurrence rates of mitral regurgitation (Table 3): patients having the surgical risk factors had a recurrence rate of regurgitation greater than 2/4 of 4.7% per year. However, patients not having these surgical risk factors still had a recurrence rate of 2.4% per year.

When we divided the patient population into patients having Barlow disease and patients having fibroelastic deficiency, the risk factors became more focused (Table 4). In fibroelastic deficiency, the nonuse of a quadrangular resection predicts recurrence of regurgitation ($P = .02$) as well as the nonuse of an annuloplasty ring ($P = .04$) and the nonuse of a sliding annuloplasty ($P = .01$). In Barlow disease, the presence of abnormal or elongated chordae ($P = .02$) is the main predictor of regurgitation, as well as the use of chordal shortening ($P = .04$), the nonuse of polytetrafluoroethylene chordae ($P = .02$), and the nonuse of an annuloplasty ring ($P = .004$). When the two populations bear the corresponding risk factors, recurrence rates are 3.6% per year for fibroelastic deficiency and 14.9% per year for Barlow disease ($P < .05$) (Table 3, Figure 3, A and B). When the correction for the corresponding surgical risk factors is made for these two patient populations (Table 3), the following linearized recurrence rates are found: 2.9% per year for Barlow

disease and 2.2% per year for fibroelastic deficiency. The difference between the two rates is not statistically significant ($P > .05$).

Echographic Characteristics of Recurrent Mitral Valve Regurgitation

The echocardiograms from the patients showing recurrence of greater than 2/4 mitral regurgitation were analyzed in an attempt to define possible mechanisms of valve incompetence. We found that 42% of these patients had a leaflet prolapse that was mostly an anterior leaflet prolapse (in 81% of cases). In the group with fibroelastic deficiency, 41% of the patients had a leaflet prolapse (80% anterior), and about the same results were seen in the group with Barlow disease: 43% showed leaflet prolapse (83% anterior). Chordal rupture or elongation was found in 24% of the patients. Leaflet thickening was diagnosed in 50% of the patients (50% with fibroelastic deficiency and 43% with Barlow disease), and leaflet calcifications were detected in 25% of the patients (29% of patients with fibroelastic deficiency and 7% of those with Barlow disease). These calcifications were pre-existent in only 4% of the patients. Left ventricular function was described as reduced in 55% of the patients, and 59% had a non-sinus rhythm.

Discussion

From the early days of mitral valve repair, Carpentier and associates¹¹ made a clear distinction between Barlow disease and fibroelastic deficiency. In later work, Fornes and colleagues¹² investigated the histologic differences but were unable to obtain a clear distinction between the two entities on this basis. They concluded “obviously the surgeons’ diagnosis based on clinical examination, echocardiographic findings and operative macroscopical findings of the mitral valve apparatus should remain the gold standard.” We followed their macroscopic criteria to separate Barlow disease from fibroelastic deficiency.

Our results are in concordance with the available clinical reports showing good clinical outcome and low reoperation rates after repair.²⁻⁹ We also found a low reoperation rate at 10 years (94.4% freedom) and an excellent clinical outcome. On the other hand, the present article confirms our previous findings that recurrence of mitral valve insufficiency based on echocardiographic studies is more frequent than the reoperation rate indicates.¹ Obviously, reoperation rate is not an optimal predictor of recurrent mitral valve regurgitation and is not the best parameter to estimate durability of valve repair. However, long-term results (over 20 years) after repair for mitral valve prolapse were reported in 2001,¹³ and they suggest that the durability of mitral valve repair will be limited but that the therapeutic consequences remain delayed. Reoperation rate after repair constantly increases: at 5 years it is 7%, at 10 years 11%, at 15 years 16%, and at 19.5 years 20%. Survival at 19.5 years is 30%.

As mentioned earlier, our immediate postoperative echocardiographic results were excellent: 98.7% of the patients had no or trivial (0/4 or 1/4) mitral valve regurgitation. In fibroelastic deficiency this was 99.8% and in Barlow disease 97.1%. However, despite such highly successful repair, recurrence of mitral valve incompetence occurs at a constant rate during the following years. Causes of recurrence of regurgitation after mitral valve repair may be classified as procedure-related or valve-related.

Concerning procedure-related or surgical factors, our study revealed nothing new: the nonuse of an annuloplasty ring, nonuse of a sliding plasty, and the use of chordal shortening instead of transposition or artificial chordae were factors of recurrence of mitral valve incompetence, as already shown by others.⁶ In a larger study, Gillinov and coworkers⁵ demonstrated that the instantaneous risk of reoperation consists of two hazard phases: a peaking early hazard phase in the first year after the operation followed by a slow-rising late hazard phase of reoperation. They showed that patients with an isolated anterior leaflet prolapse had an increased early risk of reoperation. Also, chordal shortening increased the risk of early repair failure and the use of ring annuloplasty and leaflet resection decreased the risk of reoperation in the late hazard phase. Nevertheless, most of these surgical factors are indirectly related to the process of valve degeneration, mainly in the chordae, which are severely structurally altered by the degenerative process. Therefore, besides overall progression of the valve disease, specific repair techniques may influence recurrence of regurgitation. For example, quadrangular resection of the billowing and degenerated portion of a posterior leaflet may fully exclude the diseased part of the valve and can be expected to be more durable than shortening of elongated and severely affected chordae, which are shortened but left in place. Obviously, the degeneration of the chordae progresses after repair, which is shown by the postoperative echocardiographic studies. More than 40% of the patients showing significant recurrence of regurgitation have a new leaflet prolapse, mainly from the anterior leaflet. This may originate either from progressive degeneration of the valve or from retraction of the repaired posterior leaflet segment, resulting in relative prolapse. The high incidence of chordal rupture or elongation suggests the first mechanism. Furthermore, half of these valves have leaflet thickening and one third have new calcifications. These findings suggest that the chordae elongate and the leaflet degenerates further. This explains also why implantation of artificial chordae is associated with superior results.¹⁴ Maybe the use of artificial chordae should be promoted and even used as a standard technique in every repair as a prevention of recurrence of regurgitation. Regarding the risk for SAM, the use of large annuloplasty rings (sizes 36 to 40) has recently been advocated instead of avoiding the use of an annuloplasty ring.¹⁵

Myxomatous degeneration of the valve affects the leaflets as well as the subvalvular apparatus, and the extent of

structural and mechanical changes differs between them.¹⁰ Although histologic alterations in myxomatous valves have been well described in the past,¹⁶⁻²⁰ only recently have the mechanical properties of myxomatous mitral valves been extensively studied,¹⁰ and it is shown that myxoid leaflets are more extensible and less stiff than normal valves. It is also demonstrated¹⁶ that cells of the chordae of myxomatous valves produce more glycoaminoglycans, giving them their characteristic thickening and gelatinous nature, which may account for their mechanical weakness.²¹ All these pathophysiologic findings may help to explain why a progressive incidence of mitral valve incompetence is found after initial adequate repair. When patients bearing the surgical risks (ie, use of chordal shortening, nonuse of an annuloplasty ring, and nonuse of sliding plasty) are excluded from the analysis, recurrence rate drops from 3.2% per year to 2.4% per year. This residual rate of 2.4% per year can be attributed to the phenomenon of valve degeneration. However, two distinct forms of valve degeneration were described: fibroelastic deficiency and Barlow disease.^{11,12} Because Barlow disease is a more generalized form of valve degeneration, the progression of the degenerative process in these valves can be expected to be more pronounced and reflected in the recurrence rates of mitral regurgitation. At first glance this is correct: the recurrence rate in Barlow disease is 6.0% per year and in fibroelastic deficiency 2.6% per year. However, the impact of the surgical risk factors is so high that after correction for these techniques, the residual recurrence rate decreases to almost that of fibroelastic deficiency (2.9 vs 2.2% per year).

It can be concluded that the long-term result of mitral valve repair in Barlow disease is essentially the same as in fibroelastic deficiency provided optimal surgical techniques are used. A constant recurrence rate of regurgitation (2%–3% per year) remains present owing to progression of the degenerative process in the native valve.

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Discussion

Dr Aubrey C. Galloway (New York, NY). This was an excellent paper. You should be congratulated on achieving echographic and clinical follow-up over time so that we can draw some significant conclusions. To review a couple of points: 348 patients, about 24%, had Barlow disease by gross pathologic determination. The linearized rate of recurrence of mitral regurgitation was higher clearly in the Barlow population, but you were able to identify certain other risk factors. Most prominently, you showed, similar to what others have shown, that chordal shortening does not work well in Barlow patients and that the absence of an annuloplasty device or use of a flexible annuloplasty device was associated with a higher failure rate. In contrast, you showed improved late results with chordal replacement and rigid devices. Therefore, if you used those "optimal techniques," the late recurrence rate dropped to an acceptable but still significant 2.9% per year over time.

For fibroelastic deficiency, absence of the use of a sliding plasty technique and absence of the use of a rigid ring were predictors of late recurrence, which you did not put in the presentation but did put in your manuscript. You also observed that a billowing anterior leaflet was predictive of late failure in fibroelastic deficiency, which brings me to my first point. It seems that the gross characterization of valve disease into Barlow versus fibroelastic deficiency is probably not that useful or specific a way to fine tune our predictions as to who is at risk for late failure. There is probably a spectrum of disease that we have not figured out that is not apparent by gross visual inspection at the time of surgery. Maybe we need better phenotypic characterizations or even genotypic ways to characterize these valves.

This brings me to the fundamental question: if we use optimal surgical techniques but still have a built-in failure rate of somewhere between 2.2% and 2.9%, can a valve phenotype be identified that would have a high enough risk of failure that chordal replacement should be done prophylactically since you showed that this technique improved outcomes?

Dr Flameng. Yes, we clearly do not have any idea what the underlying mechanism of this disease is, and I realize that it might well be that the spectrum from Barlow to fibroelastic deficiency is one disease. The point of the paper is that once we recognize the nondurable techniques, we still find that there is a progression of the disease. There is almost no difference anymore between the global sick valve and the localized sick valve. If you call it Barlow or fibroelastic deficiency, it does not matter.

Dr Galloway. That was my point. However, the question is, even in the “optimal technique” group you have almost a 3% per year failure rate in Barlow type valves. That is 30% over 10 years in which you are going to have reoperation or 3 or 4+ mitral regurgitation. Would you advocate that we intervene prophylactically and do chordal replacement on those patients now or not, or do you just accept that late recurrence rate?

Dr Flameng. At the moment, I think we have to accept it, because there is no technique that can improve it above this or get rid of this 2% or 3%.

Dr Galloway. That was the conclusion of the panel in the valve repair postgraduate course. The recommendation was to leave a billowing but nonprolapsing anterior leaflet alone and repair only the prolapsing posterior leaflet disease and put in an annuloplasty. It is just an interesting point for consideration based on your data.

Second, you reported that flexible rings had a higher failure rate. We have always bought into that idea, agreeing with Dr Carpentier early on in the concept of more rigid geometric remodeling of the annulus to make the repaired valve more convex to take tension off, lower the strain, and therefore lessen the late failure rate. However, there are a lot of people who do not think that way and there are a lot of people who believe that flexible devices are appropriate in patients with degenerative disease. Do you have any data from your echocardiography as to how valves with flexible devices failed or why you think your failure rate was higher in that group?

Dr Flameng. We do not have any evidence that a flexible ring is really inferior to a rigid ring. Not that many patients of our population have a flexible ring, and when they do, there are different kinds of flexible rings. Therefore, it is very difficult to say.

Dr Galloway. One last brief question. You showed in patients with fibroelastic deficiency that lack of use of sliding plasty was associated with a higher failure rate. I think most people would use the

sliding plasty technique mostly in patients who have a big height to their posterior leaflet, more the Barlow type patients, trying to lower the height of the posterior leaflet repair after repair. What techniques did you use in patients with fibroelastic deficiency when you did not do sliding plasty, and which of these techniques had the highest failure rate so we can stay away from them?

Dr Flameng. The point is that the sliding leaflet technique as described by Professor Carpentier includes reduction of the posterior leaflet by two additional triangular resections, and then you make the posterior leaflet short. In fibroelastic deficiency, there is no need for that. What we do then is just cut the posterior leaflet loose and diminish the size of the ring by the mattress sutures put from the left atrium to the ventricle. Then we replace the posterior leaflet. It is more an annuloplasty than a leaflet plasty.

Dr Galloway. I understand what you did, but what about the ones that failed? You said that patients who did not get a sliding plasty had a higher failure rate. What techniques did you use in those patients?

Dr Flameng. Many of these patients had no ring annuloplasty because the ring dilatation is not so pronounced in fibroelastic deficiency; you have patients with normal rings or with only mild dilatation of the ring. They either received no ring or no sliding annuloplasty, which induced a higher failure rate.

Dr Galloway. A higher failure rate.

Dr Alain F. Carpentier (Paris, France). Dr Flameng, I enjoyed your presentation very much. I remember very well when you published your paper in *Circulation*. I was struck by your incidence of recurrence, which was much higher than the one we had. Now today you give us at least a part of the answer. Obviously, increasing experience and careful analysis of the risk factors involved may help to identify this explanation.

You introduce an interesting distinction between an unfavorable and a favorable operation. However, there are several factors involved, patient-related or surgeon-related or technique-related, and it is not easy to identify or to differentiate these different factors. If we take the patient-related factor first, I notice that you had a rather small incidence of Barlow compared with fibroelastic deficiency. In our experience, it is about 50:50. Does it mean that many patients with Barlow disease in your experience are operated on by valve replacement or does it mean that it was difficult for you to identify the two groups?

Dr Flameng. It is well taken, because the overall rate of repair in our population of patients having degenerative mitral disease was nearly 80%, to be exact, 77%. But it changed over the years. In the late 1980s the rate of repair was much lower than it is now. Now it is almost 100%. At the beginning, it was mainly the Barlow valves that were replaced instead of repaired, and that is why the total number of Barlow valves in the series is lower, as in your series, for example.

Dr Carpentier. Another question I have relates to the management of the P2 prolapse in fibroelastic deficiency. In most instances, we did not find it necessary to use a sliding plasty, so how do you explain that? This may also relate to the difficulty in differentiating the two groups.

Dr Flameng. It is really difficult to differentiate between the groups, because there is always a gray zone in between. When you have the clear fibroelastic deficiency as you described it, then the number would be lower. Some patients we classified as having

fibroelastic deficiency had clear excess tissue in the posterior leaflet and consequently we performed more resections and sliding plasties.

Dr Carpentier. I congratulate you for having made the effort to make the distinction between Barlow and fibroelastic deficiency. This is important not so much to predict the outcome but to predict the feasibility and complexity of the repair, which is very important information for the patient.

Dr Flameng. Thank you for your comment.

Dr David H. Adams (*New York, NY*). Professor, I would like to congratulate you for your seminal work in teaching us the difference between freedom from reoperation and freedom from recurrent regurgitation, and teaching us to think more about recurrence of mitral regurgitation. I think you and Tirone David deserve a lot of credit for looking at your data that way.

I do not think this paper is going to give us the answer that we all seek, which is the true difference between fibroelastic deficiency

and Barlow disease. I would submit to you, and I would like your response, that there is a gray zone, and you have classified every patient, which makes your data, in my mind, hard to interpret. Why not take large ring sizes, for example, where there is less argument. Take your size 36, 38, and 40 ring groups, and we will be more comfortable calling that Barlow disease. Take your size 28, 30, and 32 ring groups with single segment prolapse, and we will be more comfortable calling that fibroelastic deficiency. Do a comparison of those two groups, and then I think we can get the answer. What do you think? I think your gray zone is complicating the interpretation of this information.

Dr Flameng. I do not have a clear answer for that. The only thing I can say is that we followed Professor Carpentier's identification of the disease. And Professor Carpentier is working in Paris. Paris is the capital of France, which is the biggest country in Europe. Belgium's capital is Brussels, and Belgium is the smallest country in Europe. When it rains in Paris, it drops in Brussels. [Laughter].