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Ann Thorac Surg 2009;88:1828-1837
DOI: 10.1016/j.athoracsur.2009.08.008

Mitral Valve Repair for Degenerative Disease: A 20-Year Experience

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Background. **Recent advances in surgical technique allow repair of most mitral valves with degenerative disease. However, few long-term data exist to support the superiority of repair versus prosthetic valve replacement, and repair could be limited by late durability or other problems. This study was designed to compare survival characteristics of mitral valve repair versus prosthetic replacement for degenerative disorders during a 20-year period.**

Methods. **From 1986 to 2006, 2,580 patients underwent isolated mitral valve procedures (with or without coronary artery bypass grafting), with 989 classified as having degenerative origin. Of these, 705 received valve repair, and 284 had prosthetic valve replacement. Differences in baseline characteristics between groups were assessed, and unadjusted survival estimates were generated using Kaplan-Meier methods. Survival curves were examined after adjustment for differences in baseline profiles using a Cox model, and average adjusted survival differences were quantified by area under the curve methodology. Survival differences during 15 years of follow-up also were assessed with propensity matching.**

Results. **Baseline characteristics were similar, except for (variable: repair, replacement) age: 62 years, 68 years; concomitant coronary artery bypass grafting: 24%, 32%; ejection fraction: 0.51, 0.55; congestive heart failure: 68%, 43%; and preoperative arrhythmia: 11%, 7% (all $p < 0.05$). Long-term survival was significantly better in the repair group, both for unadjusted data ($p < 0.001$) and for risk-adjusted results ($p = 0.040$). Patient survival in the course of 15 years averaged 7.3% better with repair, and increased with time of follow-up: 0.7% better for 0 to 5 years, 4.9% better for 5 to 10 years, and 21.3% better for 10 to 15 years. Treatment interaction between repair or replacement and age was negative ($p = 0.66$). In the propensity analysis, survival advantages of repair versus replacement were similar in magnitude with a p value of 0.046.**

Conclusions. **As compared with prosthetic valve replacement, mitral repair is associated with better survival in patients with degenerative disease, especially after 10 to 15 years. This finding supports the current trend of increasing repair rates for degenerative disorders of the mitral valve.**

(Ann Thorac Surg 2009;88:1828–37)

Mitral valve repair is now the most frequently performed surgical procedure for mitral valve disease in North America [1]. Moreover, repair rates seem to be increasing with time, as newer techniques such as artificial chordal replacement and autologous pericardial leaflet augmentation are perfected [2–18]. Despite this trend, few long-term data exist to support the superiority of mitral repair versus prosthetic valve replacement [19], especially in patients with degenerative valve disease. In ischemic mitral regurgitation (IMR), survival appears better with repair, but the benefits are largely related to lower operative mortality in the acutely ill population [20–22]. In elective degenerative patients, operative mortality generally is low, and relative survival characteristics are less certain.

At present, multiple conflicting concepts exist: repair has fewer requirements for anticoagulation, but may not be as durable as replacement. Survival benefits of repair may be age-related, and elderly patients could benefit less from valve repair [23, 24]. Tissue valves may perform less well in the mitral position, but mechanical valves require anticoagulation with its associated morbidity. Prosthetic heart valves have a higher incidence of endocarditis as compared with repair [25]. Finally, repair and replacement may be applied to different subsets, and all of these factors make the subject difficult to understand from clinical experience alone. Thus, in degenerative disease, it is unclear whether repair or replacement provides the best long-term outcomes, and in which patients. Therefore, the purpose of this study was to examine the long-term survival characteristics of mitral

Accepted for publication Aug 6, 2009.

This paper is dedicated to Dr David C. Sabiston, Jr (1924–2009) whose vision established the Duke Cardiac Surgery Databank in 1982.

Presented at the Poster Session of the Forty-fifth Annual Meeting of The Society of Thoracic Surgeons, San Francisco, CA, Jan 26–28, 2009.

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Published by Elsevier Inc

0003-4975/09/$36.00
doi:10.1016/j.athoracsur.2009.08.008

repair versus replacement in patients having surgery for degenerative valve disease.

Material and Methods

This study was performed with approval from the Duke Institutional Review Board and under a waiver of informed consent, but approval was not afforded for new late patient contact, unless the patient was already being followed under existing protocols. Therefore, only all-cause mortality data were available consistently. In the Duke Databank for Cardiovascular Disease, 2,580 patients with isolated mitral valve disease who underwent cardiac surgery from January 1, 1986, through December 31, 2006, were reviewed. Patients having concomitant coronary artery bypass grafting (CABG) or electrophysiologic procedures were included, but those having other major cardiac procedures were excluded (eg, aortic valve procedures, tricuspid valve procedures, mitral valve operations for other causes, repair of postinfarct ventricular septal defect, ventricular aneurysm repair or restoration). Although patients with previous CABG were included, those with previous mitral valve procedures were excluded because they may not have been candidates for either repair or replacement. This selection process produced 989 consecutive patients having primary isolated mitral valve surgery for degenerative disease (with or without CABG) during the 20-year period. In all patients, the diagnosis of degenerative disease had been documented prospectively by the operating surgeon in the Duke automated operative note, and the data set consisted primarily of patients with myxomatous prolapse or annular dilatation. The preoperative presence and severity of mitral insufficiency was determined from ventriculograms performed at the time of preoperative left heart catheterization, or from transthoracic or transesophageal echocardiograms.

Preoperative baseline characteristics and intraoperative observations for all patients were recorded prospectively during the entire 20 years, with consistent variable collection throughout the period. Late outcome data were collected prospectively on patients with significant concomitant coronary disease per Duke Databank protocols. A National Death Index search was conducted through 2006 to acquire mortality results for patients without coronary disease. Patients were divided into two groups: group 1 (n = 705) consisted of patients having mitral repair, and group 2 (n = 284) were patients having prosthetic valve replacement. Group 2 patients in turn were subdivided into those receiving mechanical valve (n = 211) or tissue valve replacement (n = 73). Operative notes of all 989 patients having mitral valve procedures were audited to ensure proper categorization. Of the repairs, more than 85% had full ring annuloplasty (usually Edwards Physio, Carpentier classic, or Seguin rings) along with appropriate leaflet or chordal procedures, or artificial chordal replacement. Innumerable different combinations of leaflet and chordal procedures were used, probably depending on surgeon preference, anatomy encountered, and evolution of techniques with time. In the valve replacement group, 26% of patients received a bioprosthesis, and 74% a mechanical valve. Partial or total chordal sparing valve replacement was performed frequently, but this variable was not documented well and could not be assessed properly in the analysis. Survival outcomes and causes of mortality were obtained from mailed self-administered questionnaires or telephone follow-up (in patients with coronary disease), as well as review of hospital records. Mortality data were adjudicated by a multidisciplinary committee. Survival data were supplemented with information from the National Death Index and Social Security Death Index. Follow-up for survival was 92% complete, assessed July 2009. Only all-cause mortality data were available consistently for analysis.

Baseline characteristics and clinical event rates were described using medians with 25th and 75th percentiles for continuous variables and frequencies and proportions for categorical variables. Descriptive data were compared using the Wilcoxon rank-sum test for continuous and ordinal variables, and a Pearson χ^2 or Fisher's exact test for categorical variables, as appropriate. The analysis strategy was to adjust for the impact of baseline characteristics on survival using multivariable Cox proportional hazards regression modeling techniques [26, 27]. To develop the risk-adjustment model, a pool of all covariates that have been shown to be important in previous analyses was chosen. The candidate variable list for baseline adjustment included the following factors: age, sex, race, history of diabetes mellitus, hypertension, hyperlipidemia, history of peripheral vascular disease, history of cerebrovascular disease, history of renal failure, body mass index, smoking history, chronic lung disease, history of myocardial infarction, history of CABG, history of percutaneous coronary intervention, history of congestive heart failure, New York Heart Association class, ejection fraction, number of diseased

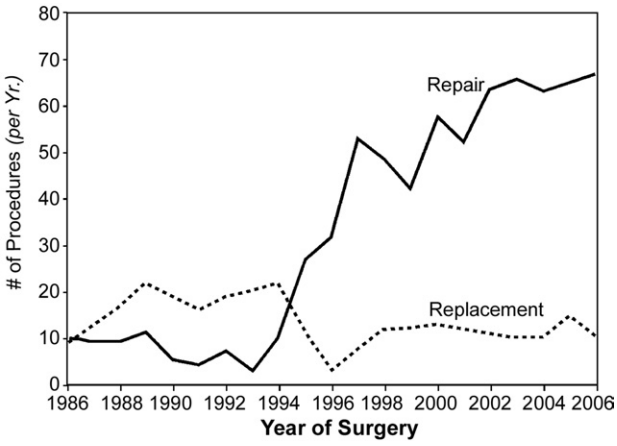

Fig 1. Frequency per year of mitral valve repair versus mitral replacement for degenerative disease at Duke University from 1986 through 2006.

Ann Thorac Surg
2009;88:1828–37

Table 1. Baseline Characteristics by Mitral Valve Repair Versus Replacement

Variable	Level	Total N (n = 989)	Overall[a]	Group 1 N (n = 705)	MV Repair[a]	Group 2 N (n = 284)	MV Replacement[a]	*p* Value[b]
Baseline characteristics								
Age	Mean	989	62.13	705	60.85	284	65.31	<0.0001
	SD		13.52		13.74		12.45	
	Median		64.00		62.00		68.00	
	25th		53.00		52.00		60.50	
	75th		73.00		72.00		74.00	
	Missing (%)		0.00		0.00		0.00	
Sex	Male	517	52.28	380	53.90	137	48.24	0.1068
	Female	472	47.72	325	46.10	147	51.76	
Race	Missing	54	. . .	44	. . .	10	. . .	0.6142
	Caucasian	783	83.74	545	82.45	238	86.86	
	Black	135	14.44	102	15.43	33	12.04	
	Native American	9	0.96	6	0.91	3	1.09	
	Hispanic	1	0.11	1	0.15	0	0.00	
	Asian	2	0.21	2	0.30	0	0.00	
	Other	3	0.32	3	0.45	0	0.00	
	Alaskan native	1	0.11	1	0.15	0	0.00	
	Multiracial	1	0.11	1	0.15	0	0.00	
BMI	Mean	892	26.40	647	26.50	245	26.15	0.3763
	SD		5.85		5.85		5.88	
	Median		25.45		25.47		25.34	
	25th		22.82		22.86		22.47	
	75th		29.07		29.30		28.97	
	Missing (%)		9.81		8.23		13.73	
CABG surgery performed	No	726	73.41	533	75.60	193	67.96	0.0138
	Yes	263	26.59	172	24.40	91	32.04	
History of smoking	No	646	65.32	471	66.81	175	61.62	0.1209
	Yes	343	34.68	234	33.19	109	38.38	
Family history of CAD	No	830	83.92	601	85.25	229	80.63	0.0739
	Yes	159	16.08	104	14.75	55	19.37	
Diabetes	No	883	89.28	630	89.36	253	89.08	0.8985
	Yes	106	10.72	75	10.64	31	10.92	
Hyperlipidemia	No	697	70.48	489	69.36	208	73.24	0.2265
	Yes	292	29.52	216	30.64	76	26.76	
Renal failure	No	955	96.56	681	96.60	274	96.48	1.0000
	Yes	34	3.44	24	3.40	10	3.52	
Hypertension	No	517	52.28	359	50.92	158	55.63	0.1795
	Yes	472	47.72	346	49.08	126	44.37	
Chronic lung disease	No	911	92.11	643	91.21	268	94.37	0.0952
	Yes	78	7.89	62	8.79	16	5.63	
Peripheral vascular disease	No	926	93.63	663	94.04	263	92.61	0.4025
	Yes	63	6.37	42	5.96	21	7.39	
Cerebrovascular disease	No	917	92.72	658	93.33	259	91.20	0.2421
	Yes	72	7.28	47	6.67	25	8.80	
Prior PCI	No	959	96.97	680	96.45	279	98.24	0.1562
	Yes	30	3.03	25	3.55	5	1.76	
Prior CABG	No	974	98.48	697	98.87	277	97.54	0.1493
	Yes	15	1.52	8	1.13	7	2.46	
Number of diseased vessels	0	688	69.57	514	72.91	174	61.27	0.0003
	1	103	10.41	57	8.09	46	16.20	
	2	77	7.79	49	6.95	28	9.86	
	3	121	12.23	85	12.06	36	12.68	

Continued

Table 1. Continued

Variable	Level	Total N (n = 989)	Overall[a]	Group 1 N (n = 705)	MV Repair[a]	Group 2 N (n = 284)	MV Replacement[a]	*p* Value[b]
Left main disease	Missing	312	...	240	...	72	...	0.3984
	No	651	96.16	445	95.70	206	97.17	
	Yes	26	3.84	20	4.30	6	2.83	
Ejection fraction	Mean	931	0.5157	660	0.5079	271	0.5347	0.0344
	SD		0.1516		0.1582		0.1323	
	Median		0.5200		0.5100		0.5500	
	25th		0.4500		0.4400		0.4500	
	75th		0.6200		0.6200		0.6300	
	Missing (%)		5.86		6.38		4.58	
Previous MI	No	869	87.87	619	87.80	250	88.03	0.9213
	Yes	120	12.13	86	12.20	34	11.97	
Congestive heart failure	No	387	39.13	224	31.77	163	57.39	<0.0001
	Yes	602	60.87	481	68.23	121	42.61	
NYHA class	I	284	28.72	201	28.51	83	29.23	0.2399
	II	187	18.91	136	19.29	51	17.96	
	III	331	33.47	245	34.75	86	30.28	
	IV	187	18.91	123	17.45	64	22.54	
Angina	No	875	88.47	617	87.52	258	90.85	0.1382
	Yes	114	11.53	88	12.48	26	9.15	
Cardiogenic shock	No	972	98.28	694	98.44	278	97.89	0.5904
	Yes	17	1.72	11	1.56	6	2.11	
Preoperative resuscitation	No	986	99.70	702	99.57	284	100.00	0.5615
	Yes	3	0.30	3	0.43	0	0.00	
Preoperative arrhythmia	No	889	89.89	625	88.65	264	92.96	0.0422
	Yes	100	10.11	80	11.35	20	7.04	
Operative characteristics								
Status of procedure	Missing	160		50		110		0.0495
	Elective	604	72.86	489	74.66	115	66.09	
	Urgent	208	25.09	155	23.66	53	30.46	
	Emergent	17	2.05	11	1.68	6	3.45	
Number of grafts placed		726		533		193		0.0489
	1	81	30.80	42	24.42	39	42.86	
	2	61	23.19	44	25.58	17	18.68	
	3	90	34.22	64	37.21	26	28.57	
	4	28	10.65	20	11.63	8	8.79	
	5	3	1.14	2	1.16	1	1.10	
Replacement valve type	Repair	705	71.28	705	100.00	0	0.00	<0.0001
	Tissue	73	7.38	0	0.00	73	25.70	
	Mechanical	211	21.33	0	0.00	211	74.30	

[a] For categorical variable, results are given as percentages. For continuous variable, results are given as mean, median, SD, quartile 1, and quartile 3. [b] Wilcoxon two-sample test for continuous variables; χ^2 test for categorical variables; and Fisher's exact test for categorical variables with cell counts less than or equal to 10.

BMI = body mass index; CABG = coronary artery bypass grafting; CAD = coronary artery disease; MI = myocardial infarction; MV = mitral valve; NYHA = New York Heart Association; PCI = percutaneous coronary intervention; SD = standard deviation.

vessels, concomitant CABG, preoperative arrhythmia, and year of surgery.

Continuous and ordinal variables were tested for linearity compared with the log hazard and were transformed as necessary to satisfy this modeling assumption. Cox regression analysis was used to identify the significant independent predictors of mortality in the multivariable setting. The adjusted survival estimates for each group were calculated by applying its estimated baseline hazard function, along with covariate Cox model parameter estimates, to all patients in the entire cohort and then averaged for all patients at each time point. The resulting curves represent an estimate of the survival that would have been realized had all patients been in each treatment group. Areas under each of the survival curves were calculated, using the trapezoidal rule, and presented for 15 years of follow-up, as well as for periods of 0 to 5 years, 5 to 10 years, and 10 to 15 years. Subsequent

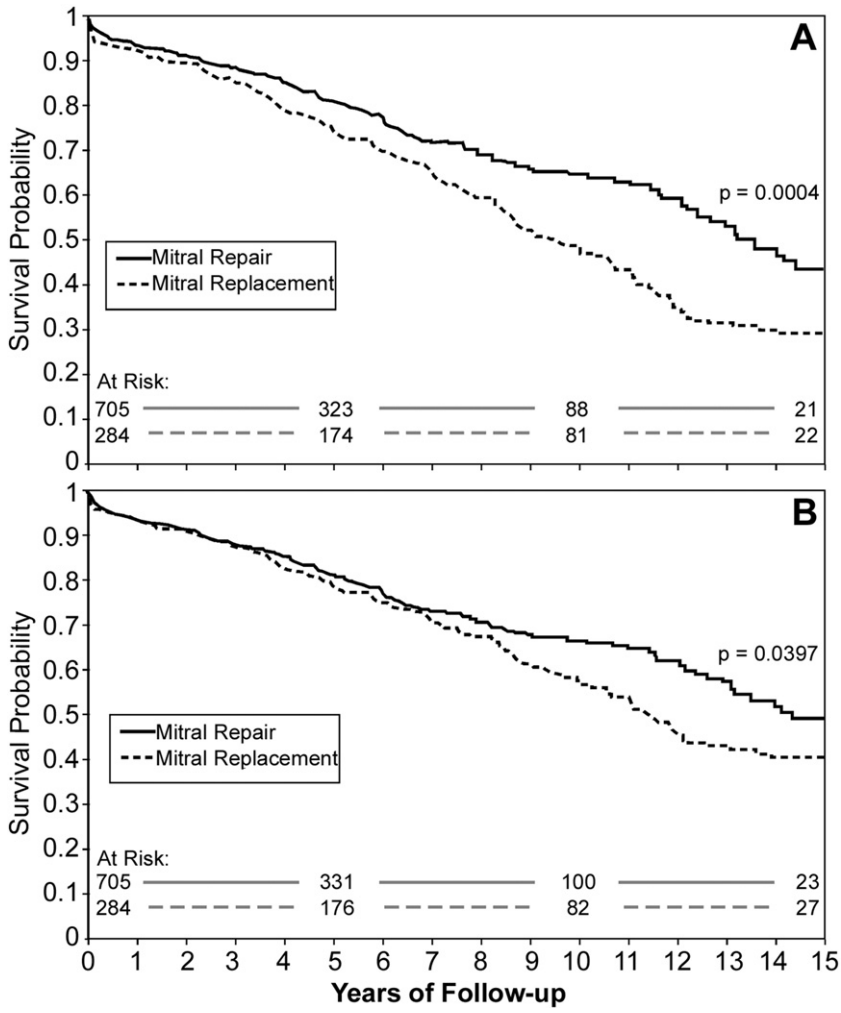

Fig 2. (A) Unadjusted Kaplan-Meier survival curves. (B) Survival curves for groups 1 and 2, after Cox model statistical adjustment for differences in baseline characteristics.

reoperations for valve procedure at Duke University Medical Center were documented for both groups. Causes of early and late mortality were examined for each group and classified as cardiac- versus non–cardiac-related mortality for descriptive purposes.

The comparison of mortality rates for mitral valve repair versus replacement was repeated using the method of subclassification on the estimated propensity score [28]. For this analysis, the propensity score was defined as the probability of receiving valve replacement (instead of repair) as determined by a logistic regression model that included the same set of covariates as in the Cox mortality model, as described above. Patients were divided into five equally sized subclasses based on their propensity for receiving mitral valve repair versus replacement. Standardized outcome rates then were calculated across the five propensity groups by applying direct adjustment with population total weights. A stratified log-rank test was used to test the hypothesis of no association between mitral valve repair versus replacement and operative mortality while stratifying on the propensity subclass. All statistical analyses were performed using SAS version 8.2 (SAS Institute, Cary, NC). A probability value less than 0.05 was considered statistically significant.

Results

The application of mitral valve repair versus replacement for degenerative disease increased steadily during the 20 years (Fig 1). Baseline characteristics for the total series of 989 patients subdivided into group 1 (repair) and group 2 (replacement) are shown in Table 1. Specifically worse risk factors for group 2 versus group 1 included greater age (68 years versus 62 years), more CABG surgery (32% versus 24%), and more nonelective surgery (34% versus 25%), whereas group 1 had worse congestive heart failure (68% versus 43%) and ejection fraction (0.51 versus 0.55; all $p < 0.05$). Severity of mitral regurgitation was less for repair patients (25% severe versus 50% severe for replacement). Median follow-up was 5.0 years (interquartile range, 2.4 to 8.6 years).

Unadjusted Kaplan-Meier survival was better for patients undergoing valve repair versus replacement (Fig

Table 2. Cox Survival Model

Risk Factor	Wald χ^2	HR	95% CI		p Value
Age (HR per 10 years)	78.0	1.76	1.55	1.99	<0.0001
History of renal failure	21.8	2.68	1.77	4.06	<0.0001
Ejection fraction (HR per 5% decrease)	18.6	1.09	1.05	1.14	<0.0001
Number of diseased vessels	13.5	1.20	1.09	1.32	0.0002
History of smoking	11.5	1.51	1.19	1.91	0.0007
History of peripheral vascular disease	8.3	1.74	1.20	2.54	0.0039
Caucasian race	8.0	0.68	0.52	0.89	0.0047
Hypertension	6.2	1.37	1.07	1.74	0.0124
Hyperlipidemia	6.0	0.71	0.54	0.93	0.0141
Male	4.9	0.78	0.62	0.97	0.0275
History of PCI	4.8	0.45	0.22	0.92	0.0281
Year of surgery (HR per 1 year increase)	4.8	0.97	0.95	0.99	0.0290
Mitral replacement	4.2	1.29	1.01	1.63	0.0397

CI = confidence interval; HR = hazard ratio; PCI = percutaneous coronary intervention.

2A). Survival curves risk-adjusted with the Cox model for differences in baseline characteristics are shown in Figure 2B, and details of the multivariable model are provided in Table 2. Part of the reduced survival for group 2 was related to worse baseline risk factors in the replacement group, and group 2 survival was more similar to that of group 1 after risk adjustment. However, valve replacement patients continued to demonstrate statistically and clinically inferior adjusted survival relative to repair ($p = 0.04$), with survival differences increasing with time. In the area under the curve analysis, replacement patients achieved 92.7% of repair survival during 15 years; 99.3% for years 0 to 5, 95.1% for years 5 to 10, and 78.7% for years 10 to 15 (Fig 2). In group 1, 24 of 705 patients (3.4%) subsequently underwent reoperation for valve procedure at Duke, whereas in group 2, 13 of 284 patients (4.6%) were reoperated on. The proportion of late deaths that were categorized as cardiac-related (Table 3) was approximately 5% lower for group 1, consistent in magnitude with the survival differences observed. It should be noted that reoperation and cause of death analyses are at best semiquantitative and not as objective as the all-cause death survival studies. However, they are presented as ancillary studies for support of the primary findings.

Using a Cox survival model, the treatment interaction between repair versus replacement and age was not significant ($p = 0.66$). In Figure 3, unadjusted survival curves for repair versus replacement, stratified for age older than and younger than 65 years, are shown to illustrate this point. Similarly, Figure 4A illustrates Kaplan-Meier and Figure 4B shows risk-adjusted survival for repair versus mechanical and tissue valve replacement. Although the sample sizes in these subgroups were too small for definitive conclusions, a suggestion exists that tissue valve performance was worse than that observed for either repair or mechanical valves.

The distribution of patients in the propensity logistic regression is shown in Table 4. Using propensity classes (quintiles) as the stratification variables, mitral replacement (versus repair) was still a significant predictor of mortality ($p = 0.046$; hazard ratio, 1.282; 95% confidence interval, 1.004 to 1.636), and detailed survival differences for each quintile of propensity at each follow-up time are shown in Figure 5. The full propensity model is shown in Table 5.

Comment

Many patients in this series may have received valve replacement because of surgeon choice, but it is also likely that most of the replacement patients had anatomy that was difficult to repair, such as Barlow's valves or bileaflet prolapse. Because these details of valve anatomy were not captured in the databank, one might argue that group comparisons in this paper are not strictly quantitative. However, significant changes in techniques of repair have occurred recently [29, 30], and now most degenerative valves can be repaired satisfactorily, even in the more complex anatomic categories [31]. At the present time, most patients in this study would be candidates for repair, and therefore, it seems logical to perform this comparison to guide future therapeutic choices. However, the groups may be different for other reasons that are not defined, and undefined selection biases or confounding variables may be present. Retro-

Table 3. Causes of Early (<90 days) and Late (>90 days) Mortality

Cause	Total (n = 989)	Group 1 MV Repair (n = 705)	Group 2 MV Replacement (n = 284)
Early mortality			
Procedure-related death	55% (26 of 47)	54% (16 of 30)	59% (10 of 17)
Cardiac death	26% (12 of 47)	23% (7 of 30)	29% (5 of 17)
Noncardiac death	19% (9 of 47)	23% (7 of 30)	12% (2 of 17)
Late mortality			
Procedure-related death	3% (8 of 273)	3% (5 of 145)	2% (3 of 128)
Cardiac death	47% (129 of 273)	45% (65 of 145)	50% (64 of 128)
Noncardiac death	50% (136 of 273)	52% (75 of 145)	48% (61 of 128)

MV = mitral valve.

Ann Thorac Surg
2009;88:1828–37

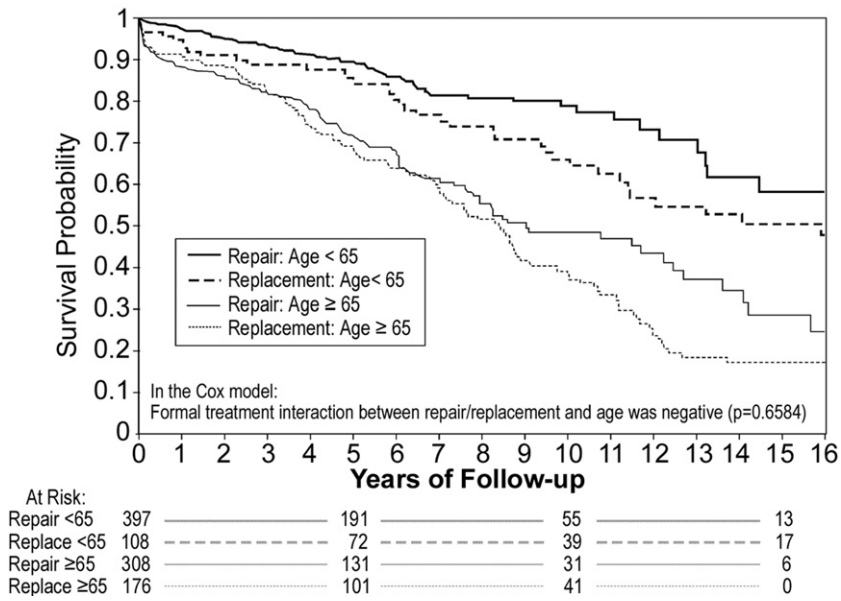

Fig 3. Unadjusted Kaplan-Meier survival curves for patients older than and younger than 65 years of age, stratified by valve repair versus replacement.

spective analyses also can be limited by lack of variable detail, such as specific valve anatomy or repair methodology, as in this study. Thus, like most observational studies, the results of this paper need to be interpreted within this context.

The present study found that mitral valve repair in patients with degenerative mitral valve disease is associated with better long-term risk-adjusted survival compared with prosthetic valve replacement. The pattern of the survival benefit in degenerative patients was unexpected, with minimal differences in operative mortality or survival during the first 5 years of follow-up. This is in contrast to repair for IMR [20], which affords major benefits in operative mortality in acutely ill IMR patients (40% to 49% of IMR patients were nonelective in the Duke series [20], as compared with 27% of degenerative patients). Perhaps an early-phase survival benefit of repair exists that is related to the magnitude of adverse risk factors, such as reduced ejection fraction and acute presentation, and therefore, is more evident in IMR. In both disorders, however, there appears to be a late-phase phenomenon in which mortality increases faster for replacement than repair. The survival advantage of repair in degenerative patients tends to increase across follow-up time and becomes more significant after 10 years. Thus, the absolute magnitude of survival difference appears smaller for degenerative disease than IMR, but in the late phase, the benefits of repair still appear to be statistically and clinically significant (Fig 2).

Because patients without coronary disease were not on a routine follow-up protocol and required a variety of follow-up sources, late morbidities could not be defined in full detail. However, there seemed to be a 5% to 10% higher incidence of cardiac death in the replacement patients, suggesting higher valve-related complications (which would have been categorized as cardiac-related). It is known that tissue valves have durability problems in the mitral position, and mechanical valves have more bleeding and thromboembolic complications [32, 33]. All valve prostheses have as much as a tenfold higher incidence of endocarditis in the long-term, as compared with repair [25]. The perceived problem with repair has been a concern about late failure. At least in these data, that concern did not materialize, and the incidence of valve reoperation after repair seemed similar or even lower than in the replacement group. Perhaps repairs have better durability than prosthetic valves because repairs primarily use the body's own tissues, which are not as prone to degenerate. Thus, repairs may perform better because they represent the best of both approaches: no need for anticoagulation and good durability. It is also possible that newer repair methods, like artificial chordal replacement, have even better durability [29, 30, 34, 35].

Because of sample size considerations, firm conclusions cannot be made about the relative merits of mechanical versus tissue valves, but the poor outcomes observed in the tissue valve cohort raises serious questions. This topic should be investigated further, especially given recent trends toward increasing use of bioprostheses for mitral replacement [1]. In practice, however, the relative propriety of various prosthetic valves is becoming less important, as the vast majority of mitral valves are now being repaired, and the results of this paper would support that trend. A differential benefit of repair with age has been discussed in several papers [23, 24], but was difficult to demonstrate in this series. The formal treatment interaction between repair or replacement and age was not significant, and direct comparison data suggested that the survival benefits of repair were similar in elderly patients (Fig

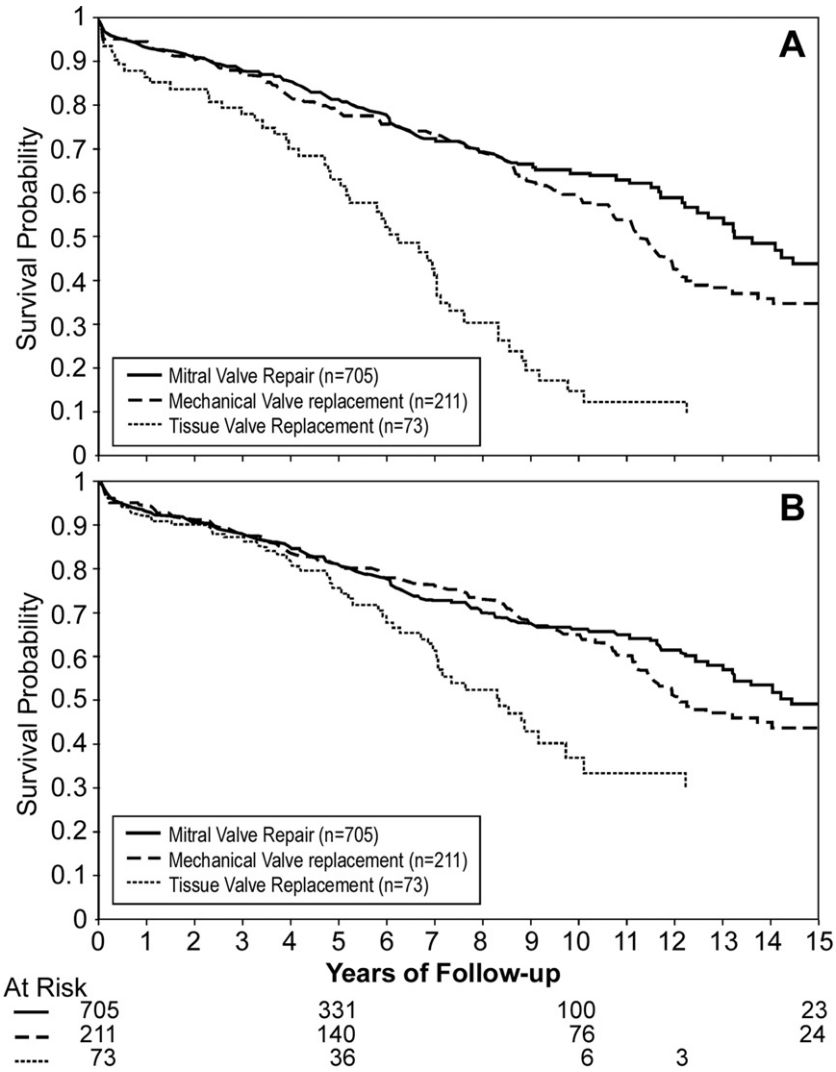

Fig 4. Unadjusted (A) and adjusted (B) survival curves for repair versus mechanical valve replacement versus tissue valve replacement. No statistical comparison was performed because of concerns about the small sample size of tissue valves, but the trend is apparent.

3). Certainly, age itself is an important predictor of late death, and elderly patients have markedly reduced survival. However, the relative benefits of repair seemed similar and justify liberal application of valve repair strategies in the elderly.

Newer techniques of mitral repair may be especially applicable to degenerative valve disease, and have markedly expanded the percentage of patients repaired [29–31]. Specifically, artificial chordal replacement, without leaflet resection, allows durable repair in most valves with myxomatous prolapse, and recent data suggest that late mitral regurgitation recurrence and reoperation may be especially low after artificial chordal procedures [30]. In general, surgeons at Duke have used full ring annuloplasty in most patients, not only to reduce annular circumference toward normal but also to shorten the anteroposterior dimension and increase surface area of leaflet coaptation [36, 37]. In this manner, holding leaflet geometry in a fixed relationship may compensate for minor deficiencies in leaflet coaptation or late problems that might occur. The introduction of minimally invasive approaches for repair of degenerative disease has facilitated patient satisfaction, and currently, most simple prolapse cases are being repaired with either port-access or robotic technology [38, 39]. Each of these technical innovations has helped to transform procedures for management of degenerative mitral disease into some of the safest and most effective in surgery. Currently, a trend exists toward earlier operations in these patients [40], and certainly, the excellent repair rates and low mortalities observed nationally [1], together with the improved late

Table 4. Distribution of Patients in Propensity Model[a]

	Quintile				
Group	1	2	3	4	5
Repair (n)	179	166	162	136	62
Replacement (n)	18	32	36	62	136
Total (n)	197	198	198	198	198

[a] Quintile 5 had the greatest propensity for replacement and quintile 1 had the greatest propensity for repair.

Ann Thorac Surg
2009;88:1828–37

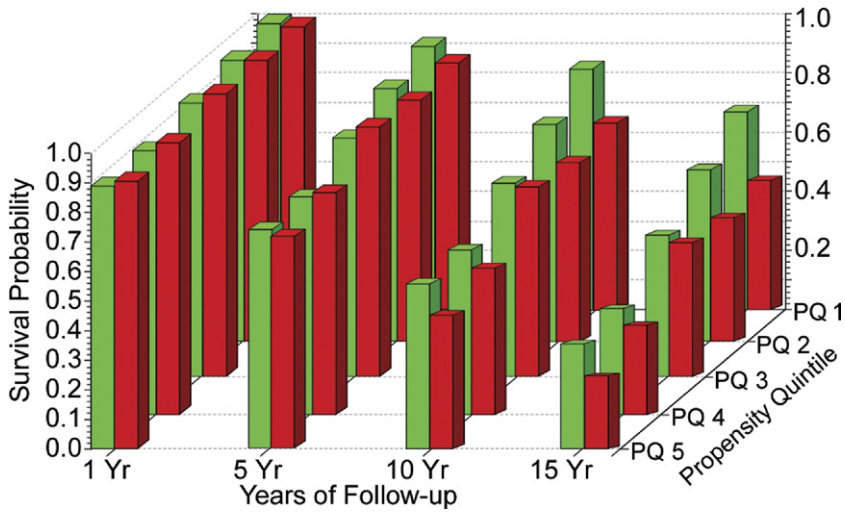

Fig 5. Observed survival of propensity-matched quintiles during 15 years of follow-up. Quintile 5 has the greatest propensity for replacement (red), and 1 has the greatest propensity for repair (green). Late survival was better with repair in all quintiles (p = 0.046).

survival in the present study, support this trend into the future.

In conclusion, mitral repair for patients with degenerative mitral valve disease appears to be associated with better long-term survival as compared with prosthetic valve replacement. The relative benefits of repair seem to be evident across the spectrum of baseline risk and in the elderly. An early suggestion exists of inferior results with tissue valve replacement, but this topic will require further investigation. Within the limits of observation analysis, these data support the current trend of increasing repair rates in patients with degenerative mitral valve disease.

Table 5. Logistic Regression Model for Propensity to Perform Mitral Valve Repair Versus Replacement[a]

Risk Factor	Wald χ^2	OR	95% CI		*p* Value
Year of surgery (HR per 1 year increase)	123.9	0.85	0.82	0.87	<0.0001
Age (HR per 10 years)	11.8	1.33	1.13	1.56	0.0006
History of smoking	6.0	1.52	1.09	2.12	0.0142
Male	3.5	0.74	0.54	1.01	0.0613
Ejection fraction (HR per 5% decrease)	2.9	0.95	0.90	1.01	0.0863
History of PCI	2.6	0.43	0.15	1.21	0.1082
Caucasian race	1.0	1.23	0.82	1.84	0.3217
History of renal failure	0.3	1.27	0.55	2.92	0.5739
History of peripheral vascular disease	0.1	1.12	0.59	2.12	0.7289
Number of diseased vessels	0.01	1.01	0.86	1.18	0.9396
Hyperlipidemia	0.03	1.03	0.71	1.51	0.8626
Hypertension	0.002	0.99	0.70	1.40	0.9626

[a] All variables examined in the Cox model were tested. Year of surgery was by far the most important variable determining repair versus replacement, with repair procedures increasing dramatically in recent years (Fig 1).

CI = confidence interval; HR = hazard ratio; OR = odds ratio; PCI = percutaneous coronary intervention.

This work was funded in part by grants from Edwards Lifesciences, St. Jude Medical, and Sorin Group.

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Ann Thorac Surg 2009;88:1828-1837
DOI: 10.1016/j.athoracsur.2009.08.008