Superiority of mitral valve repair in surgery for degenerative mitral regurgitation

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Objectives We aimed to assess the influence of type of operation on outcome in degenerative mitral regurgitation.

Methods We compared outcomes in 278 consecutive patients who underwent mitral valve repair (167 patients), replacement with subvalvular preservation (22 patients) and without subvalvular preservation (89 patients) for degenerative mitral regurgitation.

Results There was a trend towards lower mortality with repair and replacement with subvalvular preservation compared to replacement without subvalvular preservation. Thirty-day mortality was 1-2% vs 0-0% vs 4-7% (ns) respectively. Six-year survival was, respectively, 67-8 ± 7-4% (P=0.088) vs 80-8 ± 11-0% (P=0.25) vs 63-3 ± 5-9% for all-cause death, 78-5 ± 6-8% (P=0.063) vs 95-5 ± 4-4% (P=0.092) vs 67-6 ± 5-9% for all complication-related death and 80-5 ± 6-9% (P=0.076) vs 100-0 ± 0-0% (P=0.045) vs 72-8 ± 5-8% for complication-related death due to myocardial failure. Multivariate analysis confirmed independent beneficial effects from repair compared to replacement without subvalvular preservation on complication-related death (hazard ratio 0-42, P=0.010) and death from myocardial failure (hazard ratio 0-40, P=0.014), and from repair compared to mechanical replacement on thromboembolism (hazard ratio 0-45, P=0.029) and anticoagulation-related haemorrhage (hazard ratio 0-19, P=0.026).

Conclusions Mitral valve repair is superior to replacement. The greatest survival advantage is in reduced mortality from myocardial failure. Repair should be the operation of choice for degenerative mitral regurgitation. (Eur Heart J 1997; 18: 655-663)

Key words: Mitral regurgitation, mitral valve repair, degenerative valve disease.

Introduction

Outcome from total valve excision and replacement for mitral regurgitation remains relatively poor, with survival rates of only 60-80% at 5 years and 40-50% at 10 years.1-7 Mitral bioprostheses frequently fail after 7-8 years.7 Mechanical prostheses have greater risks of systemic thromboembolism, particularly in the mitral position, and of anticoagulation-related haemorrhage.8 Mitral valve repair9,10 has reduced mortality and morbidity compared to replacement14-6. Results are better with degenerative11,12 than rheumatic valve disease13-16 and many now consider repair to be the operation of choice for degenerative mitral regurgitation. However, not all valves are reparable. There is concern that patients who undergo unsuccessful repair may fare worse than if they underwent valve replacement in the first instance. Clearly, we need to know the risks of aggressive surgical policies favouring repair, but there is little published data on this. Postoperative myocardial failure is still a major problem because of suboptimal timing of surgery, but the optimum timing is difficult to determine. We therefore performed a study of 278 patients who underwent surgery for degenerative mitral regurgitation to assess the impact of type of operation and failed mitral valve repair on outcome and to identify prognostic indicators for clinical risk stratification.

Methods

Patients and surgery

The study group comprised 278 consecutive patients who underwent mitral valve repair (167 patients) or replacement with subvalvular preservation (MVR/SVP, 22 patients) or without subvalvular preservation (MVR/NoSVP, 89 patients) for degenerative mitral regurgitation between 1987 and 1994. Mean age was 65-6 ± 9-2 years. Mean follow-up was 38-8 ± 26-7 months. Seven
(4.2%) patients in the repair group and one (1.2%) in the MVR/NoSVP group had incomplete follow-up. Three patients in the repair group required re-operation for recurrent mitral regurgitation within 1 year of surgery and are also included in the MVR/NoSVP group. Two patients in the MVR/NoSVP group had undergone repair 20 years previously, and 10 proceeded to MVR/NoSVP at the same operation after unsuccessful repair. These 15 patients formed the ‘failed repair’ subgroup. The remainder of the MVR/NoSVP group formed the ‘no repair’ subgroup.

Repair was preferred to replacement. The final decision whether to attempt repair was made at operation after mitral valve inspection. The proportion of valves repaired increased from 25% in 1987 to ≥80% after 1990 as surgical experience increased. Techniques used were prosthetic ring annuloplasty (95.8% of repairs), sliding annuloplasty, posterior leaflet quadrangular resection, anterior leaflet wedge resection, annular or leaflet decalcification, chordal shortening and chordal transposition. Rigid Carpentier–Edwards rings were used in 89% of ring annuloplasties and Sculptor rings in 11%. Carpentier–Edwards bioprostheses were used in 25% of replacements and Björk–Shiley and St. Jude Medical mechanical prostheses in 75%. All patients had coronary angiograms. Concomitant coronary artery bypass grafting was performed for all stenoses of ≥60% diameter in large arteries and aortic valve replacement for all haemodynamically significant aortic valve disease. Levels of medical, surgical and anaesthetic protection techniques (cold cardioplegia only, no coronary perfusion techniques) and patient selection criteria for surgery were constant during the study period.

**Postoperative care.**

All patients were anticoagulated with warfarin post-operatively. Anticoagulation in patients in sinus rhythm was stopped 3 months after repair or bioprosthetic MVR unless other indications were present. The remaining patients were anticoagulated long-term. The target international normalized ratio was 3.0–4.0 in patients with and 2.0–3.0 in patients without mechanical valve prostheses. All patients were routinely maintained on frusemide 40 mg and amiloride 5 mg daily. Higher doses of diuretics and angiotensin converting enzyme inhibitors were started only when overt heart failure developed.

**Left ventricular and mitral valve function.**

Left ventricular ejection fraction was estimated by echocardiography and angiography pre-operatively and by echocardiography postoperatively, using visual estimation and the Hewlett-Packard Sonos 1500 internal analysis package to calculate the echocardiograms. Mitral regurgitation was graded from 0 (none) to 4 (severe) by pulsed wave Doppler. All patients had grade 3 or 4 mitral regurgitation pre-operatively. Mitral valve areas following valve repair were measured by continuous wave Doppler using the pressure half-time method.

**Statistical methods.**

The end-point in survival analysis was the first episode of each complication. The guidelines for reporting results of valve surgery exclude myocardial failure from the definition of valve-related complications. However, subvalvular preservation in mitral valve surgery preserves left ventricular function. Myocardial failure may result directly from surgery as well as from the original valve disease. It is the largest single cause of death and morbidity. Hence, we included it as a complication and defined it as either (a) overt left ventricular failure due primarily to myocardial impairment and unrelated to postoperative myocardial infarction, or (b) unexplained sudden death or ventricular arrhythmia in patients with clinical left ventricular failure or left ventricular ejection fraction ≤40% and no other causes. Systemic thromboembolism included transient ischaemic attacks, all strokes except when cerebral haemorrhage was proven, and peripheral emboli. Anticoagulation-related haemorrhage included only episodes requiring hospital treatment or investigation. The Statistical Package for Social Sciences version 6.0 program was used for statistical analysis. Means are expressed as mean ± standard deviation. Survival was calculated by life table analysis and expressed as survival ± standard error. Age and ischaemic time were compared by the t-test, patient numbers by the χ² test or Fisher’s exact test, and survival by the Wilcoxon (Gehan) statistic for univariate analysis and by Cox regression for multivariate analysis. Outcomes in the repair, MVR/SVP and MVR/NoSVP groups were compared pairwise. Only the P values for comparison with the MVR/NoSVP group have been quoted. None of the comparisons between the repair and MVR/SVP groups approached statistical significance and their P values have not been quoted. Factors assessed by multivariate analysis were: age, sex, year and type of operation, mitral leaflet pathology, left ventricular function, pre-operative New York Heart Association (NYHA) class, aortic valve disease, ischemic heart disease and heart rhythm.

**Results.**

**Baseline characteristics.**

The repair group differed from the MVR/SVP and MVR/NoSVP groups, with more posterior and less anterior mitral valve leaflet pathology, longer ischaemic operation times, less ischaemic heart disease and more patients in NYHA class III or IV pre-operatively. There
were no significant differences between the MVR/SVP and MVR/NoSVP groups. The 'failed repair' MVR/NoSVP subgroup had longer ischaemic operation times than the 'no repair' subgroup. Baseline patient characteristics are shown in Tables 1 and 2.

### Mortality

Mortality was lower with repair and MVR/SVP than with MVR/NoSVP. Although most differences did not reach statistical significance on univariate analysis, they were significant on multivariate analysis. Thirty-day mortality was 1.2% (ns) vs 0.0% (ns) vs 4.5%, respectively. Six-year survival was 67.8 ± 7.4% (P = 0.082) vs 80.8 ± 11.0% (P = 0.25) vs 63.3 ± 5.9% for all-cause death, 78.5 ± 6.8% (P = 0.063) vs 95.5 ± 4.4% (P = 0.092) vs 67.6 ± 5.9% for complication-related death, 80.5 ± 6.9% (P = 0.076) vs 100.0 ± 0.0% (P = 0.045) vs 72.8 ± 5.8% for death due to myocardial failure and 97.5 ± 1.2% (P = 0.22) vs 95.5 ± 4.4% (P = 0.75) vs 89.9 ± 3.7% for death due to other complications respectively (Fig. 1). Table 3 details the causes of death.

Multivariate analysis showed independent beneficial
Myocardial failure

Six-year cumulative freedom from myocardial failure in the repair, MVR/SVP and MVR/NoSVP subgroups was 60.7 ± 8.6% (ns) vs 68.0 ± 14.7% (ns) vs 58.3 ± 6.5%, respectively (Fig. 2). On multivariate analysis, pre-operative NYHA class and left ventricular ejection fraction ≤40% were significant predictors of post-operative myocardial failure, but type of operation was not (Table 4). Functional results were good with 91.3% (ns) vs 83.3% (ns) vs 91.8% of survivors in the repair, MVR/SVP and MVR/NoSVP groups respectively in NYHA class I or II at follow-up.

Other complications

Six-year complication-free survival in the repair, MVR/SVP and MVR/NoSVP groups was 98.0 ± 1.1% (ns) vs 100.0 ± 0.0% (ns) vs 98.7 ± 1.3% for endocarditis, 85.4 ± 3.5% (ns) vs 100.0 ± 0.0% (ns) vs 77.3 ± 5.4% for systemic thromboembolism, 98.2 ± 1.1% (ns) vs 88.1 ± 8.2% (ns) vs 88.2 ± 4.4% for anticoagulation-related haemorrhage and 96.2 ± 1.5% (ns) vs 100.0 ± 0.0% (ns) vs 95.5 ± 2.6% for mitral valve failure, respectively (Fig. 2). Complications are summarized in Table 3. Multivariate analysis showed independent beneficial effects from repair compared to mechanical replacement (hazard ratio 0.45, P=0.029, for thromboembolism and hazard ratio 0.19, P=0.026, for anticoagulation-related haemorrhage, Table 4).

### Table 3 Deaths and complications

<table>
<thead>
<tr>
<th>Complication-related deaths</th>
<th>Repair (10-2%)</th>
<th>MVR/SVP (4-5%)</th>
<th>MVR/NoSVP (25-6%)</th>
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<tbody>
<tr>
<td>Deaths within 30 days postoperatively</td>
<td>17</td>
<td>1</td>
<td>22</td>
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<tr>
<td>Myocardial failure</td>
<td>1</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>Major bowel infarction</td>
<td>1</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Thrombosis of mechanical prosthesis</td>
<td>1</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Late deaths</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Left ventricular failure</td>
<td>11</td>
<td>17</td>
<td></td>
</tr>
<tr>
<td>Leukaemia and left ventricular failure</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Endocarditis</td>
<td>2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stroke</td>
<td>1</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Anticoagulation-related brain haemorrhage</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right heart failure</td>
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</table>

<table>
<thead>
<tr>
<th>Complication-unrelated deaths</th>
<th>Repair (4-2%)</th>
<th>MVR/SVP (13-6%)</th>
<th>MVR/NoSVP (4-5%)</th>
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<tbody>
<tr>
<td>Malignant neoplasia</td>
<td>6</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Ischaemic heart disease</td>
<td>1</td>
<td>3</td>
<td>2</td>
</tr>
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</table>

<table>
<thead>
<tr>
<th>Complications</th>
<th>Repair</th>
<th>MVR/SVP</th>
<th>MVR/NoSVP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Myocardial failure</td>
<td>36 (21-6%)</td>
<td>4 (18-2%)</td>
<td>30 (33-7%)</td>
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<tr>
<td>Endocarditis</td>
<td>3 (1-8%)</td>
<td>1 (1-1%)</td>
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</tr>
<tr>
<td>Systemic thromboembolism</td>
<td>18 (10-8%)</td>
<td>2 (9-1%)</td>
<td>18 (20-2%)</td>
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<tr>
<td>Anticoagulation-related haemorrhage</td>
<td>3 (1-8%)</td>
<td>2 (9-1%)</td>
<td>7 (8-1%)</td>
</tr>
<tr>
<td>Mitral valve failure</td>
<td>6 (3-6%)</td>
<td></td>
<td>4 (4-5%)</td>
</tr>
</tbody>
</table>

### Table 4 Significant predictors of outcome from multivariate analysis

<table>
<thead>
<tr>
<th>Risk factors:</th>
<th>Death: complication-related</th>
<th>Death from myocardial failure</th>
<th>Myocardial failure</th>
<th>Thromboembolism</th>
<th>Anticoagulation-related haemorrhage</th>
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<tbody>
<tr>
<td></td>
<td>HR</td>
<td>P</td>
<td>HR</td>
<td>P</td>
<td>HR</td>
</tr>
<tr>
<td>Repair*</td>
<td>0-4</td>
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<td>0-4</td>
<td>0-014</td>
<td></td>
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<tr>
<td>Repair†</td>
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<td></td>
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<td>0-5</td>
</tr>
<tr>
<td>Age &gt;70 years</td>
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<td>2-5</td>
<td>0-012</td>
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<tr>
<td>LVEF ≤40%</td>
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<td>0-030</td>
<td>2-8</td>
<td>0-016</td>
<td>4-8</td>
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<td>NYHA III or IV</td>
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<td>0-004</td>
<td>5-0</td>
<td>0-010</td>
<td>6-6</td>
</tr>
</tbody>
</table>

HR = hazard ratio, LVEF = left ventricular ejection fraction, NYHA = New York Heart Association class, *as compared to MVR/NoSVP, †as compared to mechanical MVR/NoSVP.

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Outcome in MVR subgroups

There were no significant differences in mortality or complications of myocardial failure in the 'failed repair' and 'no repair' subgroups. Thirty day mortality was 6.7% (one patient) vs 4.2% (three patients, ns) respectively. Cumulative freedom at 6 years from all-cause death was 73.2 ± 14.0% vs 61.5 ± 6.6% (ns), from complication-related death 73.2 ± 14.0% vs 68.8 ± 6.5% (ns), and from death due to myocardial failure was 73.2 ± 14.0% vs 74.0 ± 6.4% (ns), respectively.

Discussion

The superiority of mitral valve repair to replacement

Degenerative valve disease is the commonest cause of severe mitral regurgitation in developed nations and accounted for >60% of operations for mitral regurgitation during the study period. Myocardial failure is the predominant postoperative problem. It caused >75% of complication-related deaths; 6-year mortality from myocardial failure was 20% and 6-year freedom from myocardial failure was only 60%. Preservation of left ventricular function is clearly of paramount importance and has been shown to be better in repair than MVR/NoSVP in both experimental24-26 and clinical studies22, 27-29. This effect translates well into better clinical outcome. Mitral valve repair was superior to MVR/NoSVP with respect to complication-related death (hazard ratio 0.42), particularly death due to myocardial failure (hazard ratio 0.40) rather than other complications (ns). Repair was also superior to mechanical replacement with respect to systemic thromboembolism and anticoagulation-related haemorrhage (hazard ratios 0.44 and 0.19 respectively). These results concur with other comparative clinical studies4-6,29 and provide further evidence that repair is the operation of choice for degenerative mitral regurgitation.
Complications following mitral valve repair

Outcome from mitral valve repair with respect to mitral valve failure, endocarditis, and anticoagulation-related haemorrhage was excellent with 6-year complication rates of ≤2% each. The relatively high rate of systemic thromboembolism (14.7% at 6 years) could be explained by the presence of multiple risk factors unrelated to repair. Apart from an abnormal mitral valve, >50% of our patients had chronic atrial fibrillation, >90% had left atrial enlargement, >35% had clinical heart failure and/or significant left ventricular impairment on.....
The rate of unsuccessful repair has been underestimated. Replacement at the same operation or at re-operation for repair failed in 15 (7-3%) patients who required valve replacement at the same operation were not reported. In our study, classification is based on clinical history, not exercise echocardiography, >50% were >65 years old at operation and 20% had pre-existing coronary, cerebrovascular or peripheral vascular disease. Risks of thromboembolism in warfarinised non-rheumatic atrial fibrillation range from 2% per year overall to 7%-17% per year in patients with multiple other clinical or echocardiographic risk factors[31]. Our results are consistent with these estimated risks.

The need for early surgery
Repair only reduced the severity of, and hence the mortality from, myocardial failure. It did not affect the overall incidence of postoperative myocardial failure which was high in all groups, probably because the main contributing factor is pre-existing irreversible left ventricular impairment. NYHA class III or IV symptoms are useful markers of left ventricular impairment in severe mitral regurgitation, particularly as the regurgitation masks the decline in left ventricular contractility and ejection fraction. The risk of postoperative myocardial failure is markedly increased by pre-operative NYHA class III or IV symptoms (hazard ratio 6-6) and a left ventricular ejection fraction of ≤40% (hazard ratio 4-8). Early surgery before these features appear is essential to optimise outcome. Unfortunately, >65% of our patients were in NYHA class III or IV and 20% had left ventricular ejection fractions of ≤40% pre-operatively. Until the problem of suboptimal timing of surgery is overcome, myocardial failure is likely to remain a major postoperative problem.

The influence of age on outcome
Age >70 years was not an independent predictor of postoperative myocardial failure, but myocardial failure was significantly more likely to be fatal in this age group (hazard ratio 2-5). The reason for this is unclear. NYHA classification is based on clinical history, not exercise tests. It is a subjective, broad and imprecise classification and may be systematically underestimated in the elderly because of lower expectations, more sedentary lifestyles and a slower pace of life, resulting in delayed presentation or delayed referral for surgery. The elderly may have had more severe left ventricular dysfunction than younger patients in the same NYHA class. General frailty may also reduce the tolerance of the elderly to myocardial failure and contribute to the higher mortality.

Surgery in patients with irreparable mitral valves
The rate of unsuccessful repair has been underestimated in most studies of mitral valve repair as patients who underwent failed repair and proceeded to replacement at the same operation were not reported. In our study, repair failed in 15 (7-3%) patients who required valve replacement at the same operation or at re-operation for residual or recurrent mitral regurgitation. An aggressive policy favouring repair could theoretically worsen outcome in such patients. Ischaemic time is longer, re-operation may be needed and complexity of surgery increased. However, we found no significant difference in mortality or myocardial failure between patients who required valve replacement following unsuccessful repairs and those who underwent MVR in the first instance.

The proportion of patients with irreparable valves has not been formally studied, although it has been estimated at <10% for non-rheumatic mitral valve disease[32]. Our rate of repair rose with increasing experience from 25% to >80% within 5 years. The indications have extended from uncomplicated posterior leaflet prolapse and/or annular dilatation to include complicated lesions of either or both leaflets and annular calcification. The introduction of artificial chordae may further increase the rate of repair. Nevertheless, it is likely that a significant proportion of valves, perhaps 10%, will remain irreparable because of severe disorganisation of the valve or further degeneration following repair leading to recurrent mitral regurgitation.

It has been suggested that if repair is not feasible, then the increased rates of thromboembolism and haemorrhage associated with mechanical prostheses may obviate the benefits of early surgery and constitute sufficient reason to delay surgery[33,34], particularly in patients aged ≤70 years in whom bioprostheses are unsuitable because of low durability[35]. However, this may not be true. The chief cause of mortality and morbidity after surgery for mitral regurgitation is myocardial failure. The highest priority should therefore be preservation of left ventricular function, and early surgery with subvalvular preservation is required to achieve this. Complicated repairs can now be performed successfully and it is often difficult to predict pre-operatively whether a diseased mitral valve is repairable or not. Even if repair is not possible, left ventricular function may be maintained as effectively in mitral valve replacement as in repair if the subvalvular apparatus is preserved[29,34-35]. When subvalvular preservation is impossible, implantation of artificial chordae[36] may be an effective alternative. Thromboembolic risks of early surgery have probably been overestimated. Most studies involved many elderly patients, some of whom would have had coincidental cerebrovascular disease. Patients undergoing surgery early are less likely to be in atrial fibrillation postoperatively, and should have better left ventricular function and smaller left atria. Their thromboembolic risks will therefore be lower[38] than those of the unselected patient populations in published studies.

The influence of concomitant ischaemic heart disease
The lack of influence of ischaemic heart disease on outcome in our study may be explained by a number of factors. Concomitant bypass surgery was performed in all patients with significant coronary artery disease.
Bypass graft failure mainly occurs late, outside the time frame of our study. This may explain the low incidence of postoperative myocardial infarction or ischaemia. One major reason for poor outcome with ischaemic heart disease with mitral regurgitation is its association with poor pre-operative left ventricular function. Since continued ischaemia was prevented by coronary artery bypass grafting and left ventricular function accounted for in the multivariate analysis, ischaemic heart disease itself had no independent effect on outcome.

Limitations and confounding factors

Our study was limited by lack of randomization and its retrospective nature. Despite multivariate analysis, which is an established method of accounting for baseline group differences, observational studies have been criticised for the lack of comparability between patient groups. It has been suggested that irreparable mitral valve disease may differ from reparable disease and have independent deleterious effects leading to worse outcome with valve replacement. However, outcomes were similar in the MVR/NoSVP subgroup who underwent surgery in 1987–1988, 75% of whom would have had repair if the level of surgical experience in valve repair had been higher, and the subgroup who underwent surgery in 1993–1994, almost all of whom had irreparable valves. The main reason for valve replacement rather than repair was major anterior leaflet pathology as anterior leaflet repair is more difficult. The affected leaflet was not a significant prognostic indicator on multivariate analysis. Furthermore, MVR/SVP was also performed in patients with irreparable valves and showed trends towards better outcome than with MVR/NoSVP, although trends were not significant because of small patients numbers. Valve irreparability is unlikely to be a significant independent risk factor and the differences attributed to type of operation are likely to be genuine.

Our MVR/SVP group was too small to demonstrate significant benefits of subvalvular preservation in mitral valve replacement although the trends towards better results are consistent with other studies. The apparent superiority of MVR/SVP to repair in our study also had no statistical significance. Larger studies are required to resolve these issues. Similarly, there were too few patients with bioprosthesis to demonstrate significant effects on outcome.

Conclusion

Outcome from mitral valve repair for degenerative mitral regurgitation is superior to replacement without subvalvular preservation. Repair should therefore be the operation of choice whenever feasible. However, although repair reduces the severity and hence the mortality from myocardial failure, the overall rate of myocardial failure remains high, as a consequence of pre-existing left ventricular impairment. Pre-operative NYHA class III or IV heart failure and left ventricular ejection fraction of ≤40% are major independent predictors of postoperative myocardial failure. Early surgery prior to the development of these features is also needed to optimise outcome.

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