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**ORIGINAL ARTICLE**

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# Re-repair of the failed mitral valve: insights into aetiology and surgical management†

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## Abstract

**OBJECTIVES:** Mitral valve (MV) repair is the gold standard for treatment of degenerative mitral regurgitation. A variety of surgical techniques allow surgeons to achieve a high rate of MV repair even with MV diseases of other aetiologies. However, a certain number of repairs fail over time. The aim of this study was to review our single-centre experience of MV re-repair and analyse the mode of repair failure, re-repair safety and efficiency in relation to the initial aetiology.

**METHODS:** Between 1997 and 2015, 91 patients underwent redo MV re-repair. The first MV repair was performed in our institution in 59% of cases. Follow-up information was available for 93% of our patients. The median follow-up was 56 months.

**RESULTS:** The initial aetiology was degenerative disease in 40 (44%) patients, rheumatic disease in 25 (27.5%), endocarditis in 10 (11%), ischaemic in 6 (7%), severe mitral annulus calcification in 5 (5.5%), congenital disease in 4 (4%) and unknown in 1 (1%). The mean age was 58 ± 15 years. The median delay between the 1st and 2nd repair was 49 months with 6 early re-repairs. Re-repair was urgent or emergent in 19% of cases; indications for surgery were mitral regurgitation in 48%, stenosis in 19%, endocarditis in 19%, mitral disease in 11%, ring thrombosis in 2% and systolic anterior motion in 1%. The main mechanisms of failure included technical error (30%), progression of disease (35%), new disease (29%) and unknown (6%.) Re-repair was performed through a median sternotomy in 96% of cases, and 34% of patients had concomitant procedures. Eight (9%) postoperative deaths (4 of mitral annulus calcification, 2 of endocarditis, 1 of degenerative disease, 1 of ischaemia) and 5 (6%) early failures occurred (3 of rheumatic disease, 1 of degenerative disease, 1 of a congenital condition), requiring MV replacement in 4 and new repair in 1. Overall survival at 5 and 10 years was 76% and 57%, 83% and 49% in patients with degenerative diseases and 95% and 95% in patients with rheumatic disease. Overall freedom from reoperation at 5 and 10 years was 82% and 61%, 94% and 87% with degenerative disease and 60% and 45% with rheumatic disease.

**CONCLUSIONS:** MV re-repair is feasible and has good mid-term results in patients with degenerative MV disease. Rheumatic MV disease is associated with a certain risk of failure over time; nevertheless, these patients show excellent survival after re-repair.

**Keywords:** Mitral valve re-repair • Degenerative aetiology • Rheumatic • Failure

## INTRODUCTION

Mitral valve repair (MVr) is the gold standard for treatment of degenerative mitral regurgitation (MR) [1, 2]. It is associated with improved long-term survival, better preservation of left ventricular function and freedom from thromboembolic or bleeding events compared with mitral valve replacement (MVR) [2–4]. A large armamentarium of surgical techniques (Carpentier techniques [5] and Gore-Tex neochordae [6, 7]) allows surgeons to achieve a high rate of repair in degenerative diseases.

In non-degenerative MV diseases (rheumatic, endocarditis), the main issue is the quantity and quality of the remaining tissue. To overcome inadequate conditions, a tissue replacement strategy, such as a pericardial patch, is necessary to achieve a competent valve [8, 9]. Nevertheless, classical repair techniques together with a patch allow MVr in up to 80% of cases of active endocarditis and rheumatic disease with acceptable mid-term results [9, 10]. Despite the fact that repair is less durable than MVr in diseases of degenerative aetiology, repair has some advantages over replacement.

Moreover, each aetiology has a specific rate of repair failure. The reasons for failure after MVr can be classified as technical failure, progression of the native disease or new disease [11]. Re-repair in cases of degenerative disease has been feasible, with very good results [12, 13]. However, the process remains challenging. Even if the surgeon is an expert, the procedure can be

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performed in only about 50% of redo cases [14, 15]. No data are available for results of re-repair of MV disease of other aetiologies, such as rheumatic disease.

The aim of this study was to review our single-centre experience of the re-repair of diseased MVs of all aetiologies. We analysed the mode of repair failure, re-repair safety and efficiency in terms of the initial aetiology.

## MATERIALS AND METHODS

### Patient selection

Between 1997 and 2015, 2515 mitral procedures were performed in our department; 2057 (82%) of them were valve repairs and 458 (18%) were valve replacements. For this study, we selected only patients who had a redo MV procedure after a 1st MVr for MV disease of all aetiologies. We found 186 (7%) patients: 95 (51%) underwent replacement and 91 (49%) had re-repair. This last group represents our study cohort ($n = 91$). In this cohort, the initial MVr was performed in our institution in 54 (59%) patients and in another hospital in 37 (41%) patients.

The study was approved by the ethics review board of the hospital.

### Indication for re-repair and surgical management

Demographic and echocardiographic characteristics of patients with indications for surgery are summarized in Table 1. About 60% of the patients had mild symptoms [New York Heart Association (NYHA) I or II]; the majority of patients (62%) presented with severe mitral disease. MV re-repair was proposed rather than the standard replacement options in selected patients depending on the feasibility of re-repair following evaluation with preoperative transoesophageal echocardiography (TOE). The criteria considered for feasibility of re-repair were the absence of diffuse valve calcification or retraction and the presence of a sufficiently large, mobile anterior mitral leaflet. Patients were nonetheless warned that the final decision to repair would be made only after the surgical assessment. Therefore, a plan B was always agreed on with the patient in case re-repair was deemed not feasible.

The standard procedure was resternotomy, but a few patients had a sternotomy after a first (mini)thoracotomy and a few others had a right minithoracotomy. Cardiopulmonary bypass was usually initiated using central cannulation. Cardiac arrest was obtained using anterograde warm Calafiore cardioplegia. The MV was approached through a left atriotomy and exposed using a Cosgrove retractor.

After exposure of the MV, if re-repair was chosen as the first option by the surgeon and the patient, then the MV was carefully analysed to assess and confirm the mechanism of regurgitation, as suggested by the pre- and intraoperative TOE scans. Once it was identified, the mechanism of regurgitation was recorded as technical failure, progression of the initial MV disease or new MV disease, as proposed by other authors [11]. The definitions and proportions of these 3 modes of failures are presented in Table 2.

A previously implanted annuloplasty ring was almost always systematically removed to avoid damage to the leaflets yet resecting the fibrous pannus around it. This procedure also facilitates MV analysis and repair when the dysfunction is related to a valvular or subvalvular lesion. In some instances, such as ring infection or dehiscence and MV stenosis due to pannus, the ablation of the ring is enough to restore valve function. The techniques used to repair these lesions include classical Carpentier techniques, artificial chordae and leaflet repair with patches (autologous or xenopericardium, tricuspid valve autograft or partial mitral homograft valve).

All patients had intraoperative pre- and post-repair TOE performed by an anaesthesiologist experienced in echocardiographic imaging. All patients who received prosthetic ring annuloplasty were given oral anticoagulants for 2 months postoperatively. A TOE scan was performed before discharge. Patients were followed up at 6 months, 1 year and then annually. TOE was performed at each follow-up visit.

**Table 1:** Preoperative patient characteristics (premitral valve re-repair)

| Parameters | $n = 91$ |
|---|---|
| Demographics | |
| Age (years), mean ± SD | 58 ± 15 |
| Male gender, *n* (%) | 51 (56) |
| Diabetes, *n* (%) | 8 (9) |
| Coronary artery disease, *n* (%) | 12 (13) |
| Chronic kidney failure, *n* (%) | 14 (15) |
| Dialysis, *n* (%) | 3 (3) |
| EuroSCORE II, *n* (%) | 11 (4) |
| NYHA I, *n* (%) | 16 (18) |
| NYHA II, *n* (%) | 39 (43) |
| NYHA III, *n* (%) | 29 (32) |
| NYHA IV, *n* (%) | 7 (8) |
| Indications for surgery, *n* (%) | |
| Mitral regurgitation | 44 (49) |
| Mitral stenosis | 17 (19) |
| Active endocarditis | 17 (19) |
| Mitral disease | 10 (11) |
| Ring thrombosis | 2 (2) |
| Systolic anterior motion | 1 (1) |
| Echocardiographic data | |
| Mitral regurgitation, *n* (%) | |
| Grade II | 19 (21) |
| Grade III | 13 (14) |
| Grade IV | 33 (36) |
| Mitral stenosis, *n* (%) | 27 (30) |
| LVEF (%), *n* (%) | |
| >50 | 75 (82) |
| 30–50 | 14 (15) |
| <30 | 2 (2) |
| LVEDD (mm), mean ± SD | 54 ± 8 |
| LVESD, mean ± SD | 36.5 ± 9 |
| Systolic PAP >40 mmHg, *n* (%) | 16 (18) |

LVEF: left ventricular ejection fraction; LVEDD: left ventricular end-diastolic diameter; SD: standard deviation; LVESD: left ventricular end-systolic diameter; NYHA: New York Heart Association; PAP: pulmonary artery pressure; SD: standard deviation.

### Data collection and follow-up

Operative data were extracted from a prospectively collected database containing all of the cardiac operations performed in our institution. Preoperative patient characteristics and postoperative outcomes were collected retrospectively from the patients' charts. Complete follow-up data on survival status, cause of death, valve-related events including thromboembolic or haemorrhagic events,

**Table 2:** Modes of failure of mitral valve repair in terms of disease aetiology

| Modes of failure | Overall (*n* = 91) | Degenerative (*n* = 40) | Rheumatic (*n* = 40) | Endocarditis (*n* = 10) | Ischaemic MAC congenital | | |
|---|---|---|---|---|---|---|---|
| | | | | | (*n* = 6) | (*n* = 5) | (*n* = 4) |
| Technical failure | 28 (31) | 17 (42.5) | 0 | 2 (20) | 4 (67) | 4 (80) | 1 (25) |
| Progression of disease | 32 (35) | 8 (20) | 22 (88) | 1 (10) | 0 | 0 | 1 (25) |
| New disease | 26 (29) | 15 (37.5) | 3 (12) | 4 (40) | 2 (33) | 1 (20) | 1 (25) |
| Unknown | 5 (5) | 0 | 0 | 3 (30) | 0 | 0 | 1 (25) |
| Details of failure of the first mitral valve repair | | | | | | | |
| Technical failure (*n* = 28) | | | | | | | |
| Patch dehiscence | | | | 3 (11) | | | |
| Ring dehiscence | | | | 4 (14) | | | |
| Artificial chordae rupture | | | | 4 (14) | | | |
| Recurrent prolapse | | | | 12 (43) | | | |
| Leaflet retraction post repair | | | | 2 (7) | | | |
| Leaflet perforation post repair | | | | 4 (14) | | | |
| Systolic anterior motion | | | | 1 (4) | | | |
| Progression of the disease (*n* = 32) | | | | | | | |
| Rheumatic disease process | | | | 22 (69) | | | |
| Prolapse of a new segment | | | | 7 (22) | | | |
| Recurrent endocarditis | | | | 1 (3) | | | |
| Others | | | | 2 (6) | | | |
| New disease (*n* = 26) | | | | | | | |
| Endocarditis | | | | 16 (62) | | | |
| Severe pannus on the ring | | | | 6 (23) | | | |
| Ring thrombosis | | | | 2 (8) | | | |
| New leaflet calcification | | | | 2 (8) | | | |

Values are presented as *n* (%).
MAC: mitral annulus calcification.

endocarditis, MV reoperation, clinical condition and echocardiographic follow-up scans were obtained by consulting outpatient clinic reports or by contacting referent physicians. Patients who did not have recent check-ups were interviewed by phone.

Follow-up information was available for 93% of the patients (6 patients were lost to follow-up). The median follow-up time was 56 months (interquartile range 26–94).

## Statistical analysis

The follow-up time was calculated either to death or to the last verified contact with the living patient. The follow-up time for valve-related events was calculated until the last valid assessment of these complications. Morbidity and mortality data were reported according to the 2008 Society of Thoracic Surgeons (STS)/American Association for Thoracic Surgery (AATS)/European Association for Cardio-Thoracic Surgery (EACTS) guidelines [16]. Hospital death was defined as any death occurring during the hospital stay or during the first 30 days after the operation; any other death was considered a late death. The primary outcome of the study was survival (time to all-cause mortality) including both in-hospital and long-term deaths. Secondary outcomes included valve-related complication rates including reoperation on the MV, major bleeding, thromboembolic events and endocarditis. Outcomes were reported for the entire cohort and for different subgroups depending on the initial aetiology of the MV disease.

Continuous variables were reported as the mean ± standard deviation for variables with a normal distribution or as median and interquartile range for non-parametric distributions. Time-to-event analysis was performed with the product-limit method (Kaplan–Meier). Curves were compared using the log-rank test. Statistical analyses were performed using GraphPad Prism 7.0 (San Diego, CA, USA).

# RESULTS

## Aetiologies of mitral valve disease and first repair data

At the initial MVr, the aetiologies of the MV diseases were mainly degenerative in 40 (44%) patients and rheumatic in 25 (27.5%); the remaining aetiologies were endocarditis in 10 (11%), ischaemic in 6 (7%), degenerative with severe mitral annulus calcification (MAC) in 5 (5.5%), congenital in 4 (4%) and unknown in 1 (1%). The majority of the patients [84 (92%)] were operated on by sternotomy; the remaining, by (mini) thoracotomy (5 right and 2 left thoracotomy). Associated procedures were performed in 33 (36%) patients including 20 aortic valve repairs or replacements, 10 coronary artery bypass grafts and 3 tricuspid valve repairs. Repair techniques used at the initial MVr were triangular or quadrangular resection in 18 (20%), sliding plasty in 6 (7%), artificial chordae in 13 (14%), commissurotomy in 15 (17%), leaflet repair with a patch in 9 (10%), extensive decalcification of MV annulus in 5 (6%), Alfieri stitch in 3 (3%) and prosthetic ring annuloplasty in 55 (60%).

## Modes of failure and re-repair surgery

At redo surgery, the mean age was 58 ± 15 years; 51 (56%) were men. The mean delay between the 2 repairs was 90 ± 84 months

(range 0–440, median: 49 months, interquartile range 10–130). In 6 (7%) patients, MV re-repair was necessary during the same hospitalization or within 30 days after the initial MVr. Excluding those 6 early re-repair procedures, the mean delay for re-repair was 96 ± 85 months. MV re-repair was elective in 74 (81%) patients, urgent in 14 (16%) and emergent in 3 (3%).

The modes of failure are detailed in Table 2 for the entire cohort and for each aetiologic subgroup. Technical failure was the mode of failure in 28 patients (31%), progression of the disease in 32 (35%) and new disease in 26 (29%). In the degenerative subgroup ($n = 40$), technical failure was the more prevalent mechanism and was observed in 17 (42.5%) patients, whereas new disease and disease progression were observed in 15 (37.5%) patients and 8 (20%) patients, respectively. In the rheumatic subgroup ($n = 25$), no technical failures were noted, but 22 (88%) patients presented with disease progression.

In the subgroup of patients with degenerative disease and operated on initially in our institution ($n = 26$), 9 (35%) patients had technical failure. Three patients were treated immediately after their first repair failure: 1 for SAM and 2 for residual MR (1 after a port-access procedure failure). In addition to these 3 technical failures, 2 patients had at least moderate MR after the 1st repair (1 after a port-access procedure and 1 after a transaortic Alfieri procedure associated with a valve-sparing root replacement procedure). These 2 patients had successful MV re-repairs 6 and 8 months postoperatively. In this subgroup, the mean delay between the 1st MVr, which was done in our institution, and the redo repair for technical failure, was 5 months.

Table 3 summarizes operative data related to the re-repair procedure. Ninety-six percentof re-repairs were done by sternotomy, with associated procedures in 34% of cases.

**Table 3:** Intraoperative data for mitral valve re-repair procedures

| | |
|---|---|
| Approach, *n* (%) | |
| Resternotomy | 82 (90) |
| Sternotomy after (limited) thoracotomy | 5 (6) |
| Right minithoracotomy after sternotomy | 3 (3) |
| Redo right thoracotomy | 1 (1) |
| Re-repair techniques, *n* (%) | |
| Ring removal | 47 (52) |
| Triangle or quadrangular resection | 15 (17) |
| Sliding plasty | 6 (7) |
| Chordae transfer | 7 (8) |
| Artificial chordae | 34 (37) |
| Papillary muscle splitting | 10 (11) |
| Commissurotomy | 9 (10) |
| Commissure closure | 13 (14) |
| Leaflet patch repair | 26 (29) |
| Annulus repair with patch | 4 (4) |
| Prosthetic ring annuloplasty | 55 (60) |
| Pericardial band annuloplasty | 3 (3) |
| Associated procedures, *n* (%) | Total = 31 (34) |
| CABG | 6 (6) |
| Aortic procedures | 13 (14) |
| Tricuspid alone or with others | 15 (17) |
| Others | 7 (7) |
| Operative time | |
| Mean CPB time (min) | 124 |
| Mean cross-clamp time (min) | 85 |
| Second run, *n* (%) | 6 (7) |

CABG: coronary artery bypass graft; CPB: cardiopulmonary bypass.

## Hospital outcomes

Eight (9%) patients died in the hospital; half of them were in the MAC subgroup. Causes of deaths were congestive heart failure in 4 (4%), severe brain injury after cardiac arrest in 1 (1%), septic shock in 1 (1%), stroke with aortic dissection in 1 (1%) and unknown in 1 (1%). Other significant complications after MV re-repair were reoperation for tamponade or bleeding in 8 (9%), sternal dehiscence in 2 (2%) and stroke in 2 (2%).

Early re-repair failure requiring a 3rd MV reoperation occurred in 5 (6%) patients, mostly those with disease of rheumatic aetiology ($n = 3$) (Table 4). One patient died after early re-repair failure. The 3rd MV operation for those 5 patients comprised replacement in 4 and repair in 1 case.

## Follow-up

During the follow-up period, 15 (17%) patients died after a median follow-up of 56 months. The deaths were mainly cardiac related ($n = 12$, 80%), with no valve-related deaths. Table 4 summarizes the follow-up results. Survival at 5 and 10 years was 76% and 57% for the entire cohort (Fig. 1); 83% and 49% for those whose disease was degenerative, 95% and 95% for those whose disease was rheumatic in origin. Survival of the rheumatic subgroup was significantly better than that of the degenerative subgroup (log-rank $P = 0.04$), but rheumatic patients were also on average significantly younger (53 vs 63 years, $P = 0.001$). The 5-year survival rate was 42% among those with endocarditis; 20%, with ischaemia; 20%, with MAC and 100%, with congenital disease (Fig. 2). No comparison was done with the other subgroups because of the small number of patients.

Freedom from reoperation at 5 and 10 years was 82% and 61%, respectively, for the entire cohort: 94% and 87% for those whose disease had a degenerative aetiology; 60% and 45% for those whose disease had a rheumatic aetiology (Fig. 3). Late re-repair failure necessitating a 3rd MV operation occurred in 12 (13%) patients: 8 of them had disease of rheumatic aetiology; 2, degenerative aetiology; 1, congenital aetiology and 1, ischaemic aetiology. In the subgroup whose disease had a rheumatic aetiology, 4 out of 8 patients who had late re-repair failure, the mechanism responsible for the failure was disease progression. In 2 patients, the reason for failure was rupture of the artificial chordae (associated with leaflet calcification in 1 case, also suggesting disease progression). Progression of residual postoperative MR and patch perforation were each responsible for failure in 1 case. Regarding the durability of re-repair in the degenerative subgroup, 2 patients had late reoperation during follow-up: 1 had a second re-repair because of ring dehiscence and 1 underwent MV replacement for disease progression and heart failure. Freedom from reoperation was statistically different between rheumatic and degenerative subgroups (log-rank test, $P = 0.003$).

Functional class and echocardiographic data obtained at the latest available follow-up visit for patients who were not reoperated on are presented in Table 4. In the degenerative and rheumatic subgroups, the clear majority of patients were asymptomatic at a mean follow-up of 5 years. In the degenerative subgroup, 25 (62.5%) had an echocardiographic follow-up: 3 of them had moderate MR, 1 had severe MR (with mitral stenosis) and 1 had moderate mitral stenosis. In the rheumatic subgroup, 12 (48%) had echocardiographic follow-up; 4 of the 12 had moderate stenosis.

**Table 4:** Results

| | All cohorts ($n = 91$) | Degenerative ($n = 40$) | Rheumatic ($n = 25$) | Endocarditis ($n = 10$) | Ischaemic ($n = 6$) | MAC ($n = 5$) | Congenital ($n = 4$) | Unknown aetiology ($n = 1$) |
|---|---|---|---|---|---|---|---|---|
| Operative deaths | 8 (9) | 1 (2.5) | 0 | 2 (20) | 1 (17) | 4 (80) | 0 | 0 |
| Early reoperation | 5 (5.5) | 1 (2.5) | 3 (12) | 0 | 0 | 0 | 1 (25) | 0 |
| Late deaths | 15 (16) | 8 (20) | 1 (4) | 3 (30) | 3 (50) | 0 | 0 | 0 |
| Cardiac related | 12 (13) | 6 (15) | 1 (4) | 2 (20) | 3 (50) | 0 | 0 | 0 |
| Non-cardiac related | 3 (3) | 2 (5) | 0 | 1 (10) | 0 | 0 | 0 | 0 |
| Late reoperation | 12 (13) | 2 (5) | 8 (32) | 0 | 1 (17) | 0 | 1 (25) | 0 |
| Clinical follow-up | 47 (52) | 28 (70) | 13 (52) | 3 (30) | 1 (17) | 1 (20) | 1 (25) | 0 |
| NYHA I | 37 (78) | 24 (86) | 8 (61) | 3 (100) | 0 | 1 (100) | 1 (100) | |
| NYHA II | 9 (19) | 4 (14) | 4 (31) | 0 | 1 (100) | 0 | 0 | |
| NYHA III | 1 (2) | 0 | 1 (8) | 0 | 0 | 0 | 0 | |
| Echocardiographic follow-up | 46 (50) | 25 (62.5) | 12 (48) | 4 (40) | 2 (33.3) | 1 (20) | 1 (25) | 1 |
| MR II | 3 (6.5) | 3 (12) | 0 | 0 | 0 | 0 | 0 | 0 |
| Moderate stenosis[a] | 6 (13) | 1 (4) | 4 (33) | 0 | 0 | 0 | 0 | 1 |
| Mitral disease[b] | 1 (2) | 1 (4) | 0 | 0 | 0 | 0 | | 0 |

Values are presented as *n* (%).

[a]Mitral stenosis is defined as valve area <1.5 $cm^2$; it was always associated with a mean gradient >5 mmHg.

[b]Patient with mitral disease was the only one to have significant MR associated with stenosis.

MAC: mitral annulus calcification; MR: mitral regurgitation; NYHA: New York Heart Association.

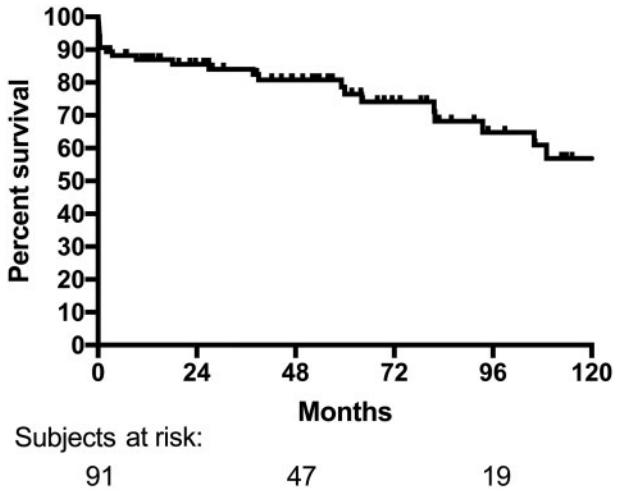


**Figure 1:** Overall survival after mitral valve re-repair at 5 and 10 years was 76% and 57%, respectively, for the entire cohort.

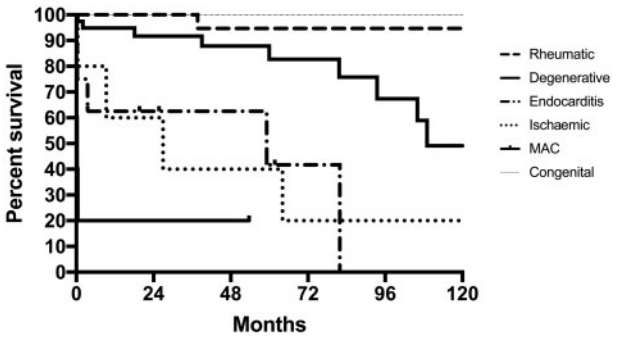


**Figure 2:** Survival for each aetiology after mitral valve re-repair: the 5-year survival rate was 83% for degenerative disease; 95% for rheumatic disease; 42% for endocarditis; 20% for ischaemia; 20% for mitral annulus calcification and 100% for congenital conditions. The 8- and 10-year survival rates were 67% and 49% for degenerative disease and 95% for rheumatic disease. MAC: mitral annulus calcification.

Two strokes occurred during follow-up (2%) but no haemorrhagic or endocarditis events were recorded.

## DISCUSSION

### Mortality

In-hospital deaths in our study were 9%, similar to the results of Kwedar *et al.* [17], who reported a 12% mortality rate in their series after reoperation on the MV. In our series, the mean EuroSCORE II was 11.4%. Our study cohort was indeed heterogeneous: This high EuroSCORE II reflects previous cardiac surgery, a high prevalence of associated procedures, active endocarditis, urgent procedures and a critical preoperative state in 3 patients. Nevertheless, the mean EuroSCORE II calculated for patients with disease of degenerative aetiology ($n = 40$) and for the rheumatic population ($n = 25$) was 5.3% and 4.5, respectively. Operative mortality in these 2 subgroups was only 2.5% and 0%, respectively. On the other hand, the mean EuroSCORE II of the 'in-hospital death population' was 18.6%. Furthermore, in the MAC subgroup with extensive decalcification at the 1st MVr, re-repair for endocarditis or MR with patch dehiscence was associated with an extremely high mortality rate (80%).

### Re-repair in rheumatic mitral valve disease

In this subgroup of patients, the main reason for redo repair was the progression of the rheumatic process rather than technical failure. Thus, even in the case of a perfect result from the 1st repair, rheumatic valves have a certain risk of late reoperation due to evolution of the underlying disease. The rate of progression of

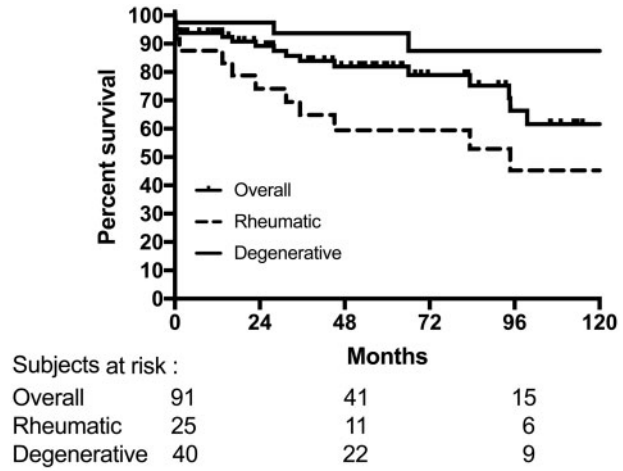


**Figure 3:** Freedom from reoperation: Freedom from reoperation at 5 and nearly 10 years was 82% and 61%, respectively, for the entire cohort; 94% and 87% for degenerative aetiology and 60% and 45% for rheumatic aetiology.

the disease is not always predictable; many of our patients had 2 or even 3 repair procedures. The mean delay between the 2 first interventions (first MVr and MV re-repair) in the rheumatic population of our cohort ($n = 25$) was 207 months, but the mean delay between the 2nd and 3rd operation (8 patients with late failure of the re-repair) was 43 months, suggesting the probable negative impact of the poor quality of the tissue on the repair.

Even though no deaths were associated with the reinterventions in our experience, we believe that re-repair in patients whose disease is of rheumatic aetiology yields suboptimal results in terms of durability and should be carefully evaluated for each patient and reserved only for those whose valves are of good tissue quality. Nonetheless, given the excellent survival after re-repair in this subgroup, despite the risk of failure, re-repair should not be abandoned. To our knowledge, there are no current studies in the literature on re-repair for rheumatic MVs whose findings compare with our findings.

## Re-repair in degenerative mitral valve disease

The mean delay between the 2 operations for this subgroup ($n = 40$) was 48 months. Many patients did not have their 1st operation in our institution. Sometimes the exact surgical technique used at the 1st repair and the postoperative echocardiographic scans were not available, so the only way to determine the repair techniques used at the first procedure was to review the pre-re-repair TOE scans and the perioperative surgical evaluation.

Technical failure was the main mechanism behind early failure. Progression of the disease and new disease occurred later, although sometimes it was difficult to distinguish between the 2 causes. Dumont *et al.* [13] suggested that technical failure of the procedure may occur early (median = 3 months) and that re-repair is a good option for these patients. The predominant reasons for technical failure of MVr were recurrent prolapse of a treated segment, ring dehiscence, leaflet retraction or perforation. In 4 cases, we observed rupture of a calcified Gore-Tex neochordae at its midpoint in what we call the 'dry spaghetti syndrome'. Although we noted only a small number of cases, these ruptures pinpointed a potential limitation of the neochordae. Consequently, we usually consider adjunctive techniques such as chordal transfer in order to create a secure, durable repair. Anyanwu et al. [12] reported a technical failure of the 1st repair in 34% with degenerative disease. Their findings were similar to our findings (42.5%), especially when the 1st repair was done in our institution (35%). Nishida *et al.* [18] performed 86 redo MV procedures for degenerative repair failure and found procedure-related failure in 23% of cases. In their cohort, only 27% of patients had a re-repair procedure. A total of 61% of this re-repair population had procedure-related failures, which means that, in a case of technical failure, the valve is more susceptible to being re-repaired. Increasing experience together with application of appropriate surgical techniques and immediate perfect results should result in a significant reduction in technical failures.

Progression of disease is a prolapse of a segment of the MV not treated (and not present) at the 1st intervention. Can we prevent the progression by supporting segments adjacent to the prolapsed area? Does the use of 3-dimensional (3D) echocardiography result in a more accurate perioperative assessment of the valve, thereby permitting the surgeon to address all abnormal areas? Al Fakhouri *et al.* [19] showed the superiority of 3D TEE over 2-dimensional TOE for accurate MV assessment in MR, especially for the determination of the length of the functional posterior mitral leaflet in case of interventional mitral repair. Witschey *et al.* [20] described their clinical experience with 1 patient with a normal MV, 2 patients with ischaemic MR and 1 with degenerative mitral disease whereby they obtained good representative models of MV leaflet and annulus geometry using 3D echocardiographic printing. The authors note that this automated imaging method is still in its early stages and requires refinement to better describe the other elements of the MV apparatus. Mahmood *et al.* [21] also developed physical models of mitral annuli in 5 patients and found them helpful to understand the disease and the postrepair changes. Its clinical use is still limited, but we think that 3D echocardiography in the next few years will add value to disease assessment and to the treatment of other concomitant lesions.

The operative mortality rate of patients in the degenerative subgroup who had re-repairs was 2.5%; these results were similar to those of Anyanwu *et al.* [12] (28.3% of their cohort had non-degenerative disease) and of Dumont *et al.* [13]. Anyanwu *et al.* [12] reported 90% survival at 5 years. Dumont *et al.* [13] reported 81% survival at 12 years. We had a worse survival rate than these authors, but it is noteworthy that in our cohort, 20% of the patients presented with acute active endocarditis, and there was a significant number of associated procedures. Our results for freedom from reoperation are also similar to those of Dumont *et al.* [13].

## Mitral valve re-repair: general considerations

In our experience, redo repair is not associated with higher mortality and morbidity rates than MVr. Surgical indications for re-repair are the same as for repair. In our opinion, the surgery should not be delayed in order to prevent the potential risk of damaging the valve. The ring is removed systematically because there is always pannus coming from the ring and invading the leaflet, inducing its restriction. By removing the ring, we give the valve more mobility, which allows better assessment of the mechanism of failure. We think that it is mandatory in late redo surgery to start by removing the ring. In some cases of early redo surgery, when the ring has not yet induced a fibrous reaction, re-repair might be performed without removing the ring.

## Limitations

The population of this retrospective study is very heterogeneous, with many small subgroups that are difficult to analyse correctly. We did not perform a comparison of replacement results in patients judged difficult to re-repair. The 1st repair was not done in our department for 41% of patients, and in many cases (14 patients), the exact repair technique used at the 1st procedure was not clearly described. For these patients, the only way to determine the repair techniques used at the 1st procedure was to review the pre-re-repair TOE scans and the perioperative surgical evaluation during re-repair surgery.

## CONCLUSION

MV re-repair is feasible and has good mid-term results in patients with degenerative disease. Rheumatic disease is associated with a certain risk of failure over time; nevertheless, these patients show excellent survival after re-repair. Regardless of the aetiology, re-repair should always be considered. Good exposure of the MV, removal of the ring, thorough assessment of the tissue and application of standard, appropriate repair techniques are the keys to successful repair.

**Conflict of interest:** none declared.

## REFERENCES

[1] Vahanian A, Alfieri O, Andreotti F, Antunes MJ, Baron-Esquivias G, Baumgartner H *et al.* Guidelines on the management of valvular heart disease (2012): the Joint Task Force on the Management of Valvular Heart Disease of the European Society of Cardiology (ESC) and the European Association for Cardio-Thoracic Surgery (EACTS). Eur J Cardiothorac Surg 2012;42:S1–44.

[2] Gillinov AM, Cosgrove DM. Current status of mitral valve repair. Am Heart Hosp J 2003;1:47–54.

[3] Mohty D, Orszulak TA, Schaff HV, Avierinos J-F, Tajik JA, Enriquez-Sarano M. Very long-term survival and durability of mitral valve repair for mitral valve prolapse. Circulation 2001;104(Suppl 1):I1–17.

[4] Lazam S, Vanoverschelde J-L, Tribouilloy C, Grigioni F, Suri RM, Avierinos J-F *et al.* Twenty-year outcome after mitral repair versus replacement for severe degenerative mitral regurgitation: analysis of a large, prospective, multicenter, international registry. Circulation 2017; 135:410–22.

[5] Carpentier A. Cardiac valve surgery—the 'French correction'. J Thorac Cardiovasc Surg 1983;86:323–37.

[6] Salvador L, Mirone S, Bianchini R, Regesta T, Patelli F, Minniti G *et al.* A 20-year experience with mitral valve repair with artificial chordae in 608 patients. J Thorac Cardiovasc Surg 2008;135:1280–87.e1.

[7] David TE, Armstrong S, Ivanov J. Chordal replacement with polytetrafluoroethylene sutures for mitral valve repair: a 25-year experience. J Thorac Cardiovasc Surg 2013;145:1563–9.

[8] Uchimuro T, Fukui T, Shimizu A, Takanashi S. Mitral valve surgery in patients with severe mitral annular calcification. Ann Thorac Surg 2016; 101:889–95.

[9] De Kerchove L, Price J, Tamer S, Glineur D, Momeni M, Noirhomme P *et al.* Extending the scope of mitral valve repair in active endocarditis. J Thorac Cardiovasc Surg 2012;143:S91.

[10] Piciché M, El Khoury G, D'udekem Y, Noirhomme P. Surgical repair for degenerative and rheumatic mitral valve disease. Operative and mid-term results. J Cardiovasc Surg 2002;43:327–35.

[11] Anyanwu AC, Adams DH. Why do mitral valve repairs fail? J Am Soc Echocardiogr 2009;22:1265–8.

[12] Anyanwu AC, Itagaki S, Varghese R, Castillo J, Chikwe J, Adams DH. Re-repair of the mitral valve as a primary strategy for early and late failures of mitral valve repair. Eur J Cardiothorac Surg 2014;45:352–8.

[13] Dumont E, Gillinov AM, Blackstone EH, Sabik JF, Svensson LG, Mihaljevic T *et al.* Reoperation after mitral valve repair for degenerative disease. Ann Thorac Surg 2007;84:444–50.

[14] Suri RM, Schaff HV, Dearani JA, Sundt TM, Daly RC, Mullany CJ *et al.* Recurrent mitral regurgitation after repair: should the mitral valve be re-repaired? J Thorac Cardiovasc Surg 2006;132:1390–7.

[15] Zegdi R, Sleilaty G, Latrémouille C, Berrebi A, Carpentier A, Deloche A *et al.* Reoperation for failure of mitral valve repair in degenerative disease: a single-center experience. Ann Thorac Surg 2008;86:1480–4.

[16] Akins CW, Miller DC, Turina MI, Kouchoukos NT, Blackstone EH, Grunkemeier GL *et al.* Guidelines for reporting mortality and morbidity after cardiac valve interventions. J Thorac Cardiovasc Surg 2008;135: 732–8.

[17] Kwedar K, McNeely C, Vassileva CM *et al.* Outcomes of early mitral valve reoperation in the medicare population. Ann Thorac Surg 2017;4975: 30670–7.

[18] Nishida H, Fukui T, Kasegawa H, Kin H, Yamazaki M, Takanashi S. Causes of repair failure for degenerative mitral valve disease and reoperation outcomes. Eur J Cardiothorac Surg 2018;53: 1244–50.

[19] Alfakhouri A, Heidrich FM, Sveric KM, Pfluecke C, Kvakan H, Quick S *et al.* Three-dimensional transoesophageal echocardiography is crucial for valid assessment of mitral valve leaflet morphology in severe mitral regurgitation prior to interventional repair. Acta Cardiol 2017;5:1–9.

[20] Witschey WRT, Pouch AM, McGarvey JR, Ikeuchi K, Contijoch F, Levack MM *et al.* Three-dimensional ultrasound-derived physical mitral valve modeling. Ann Thorac Surg 2014;98:691–4.

[21] Mahmood F, Owais K, Montealegre-Gallegos M, Matyal R, Panzica P, Maslow A *et al.* Echocardiography derived three-dimensional printing of normal and abnormal mitral annuli. Ann Card Anaesth 2014;17:279.