Management of mitral regurgitation during left ventricular reconstruction for ischemic heart failure†

Patrick Klein*a, Jerry Braunb, Eduard R. Holmanc, Michel I.M. Versteeghd, Harriette F. Verweyf, Robert A.E. Diona, Jeroen J. Baxc and Robert J.M. Klautza

*a Department of Cardiothoracic Surgery, Leiden University Medical Center, Leiden, The Netherlands
b Department of Cardiology, Leiden University Medical Center, Leiden, The Netherlands
c Corresponding author. Tel: +31-71-5264022; fax: +31-71-5266965; e-mail: p.klein@lumc.nl (P. Klein).

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Abstract

OBJECTIVE: Remodeling of the left ventricle (LV) in ischemic cardiomyopathy frequently leads to functional mitral regurgitation (MR). The indication for correcting MR in patients undergoing LV reconstruction (LVR) is unclear. In this study, we evaluated our strategy of correcting MR ≥ grade 2+ by restrictive mitral annuloplasty (RMA) during LVR.

METHODS: We studied 92 consecutive patients (76 men, mean age 61 ± 10 years) who underwent LVR for ischemic heart failure (IHF). RMA was performed in all patients with MR ≥ grade 2+ on preoperative echocardiography and in patients who showed increased MR to ≥ grade 2+ immediately after LVR. Patients were attributed to a RMA and no-RMA group, depending on whether or not concomitant RMA had been performed. Mean clinical and structured echocardiographic follow-up was 47 ± 20 months and was 100% complete.

RESULTS: In 38 out of 40 patients (95%) with preoperative MR ≥ grade 2+, concomitant RMA was planned and performed. In 17 out of 52 patients (33%) with MR < grade 2+ preoperatively, MR increased after LVR to ≥ grade 2+ leading to additional RMA during a second period of aortic cross-clamping. Early mortality in the RMA group (n = 55) was 12.7% and survival at 36 months 78.2 ± 11.2%. Early mortality in the no-RMA group (n = 37) was 5.4% and survival at 36 months 81.1 ± 12.8%. Patients in the RMA group had significantly more reduced LV function with greater LV dimensions and volumes preoperatively. Echocardiography demonstrated sustained improvement in LVEF with reduction of LV volumes in both patient groups. Recurrence of MR at late follow-up was observed in 2 patients (1 patient per group).

CONCLUSIONS: Patients with IHF eligible for LV reconstruction have MR ≥ grade 2+ in 44% of cases. In one-third of IHF patients with MR < grade 2+ preoperatively, MR increases to ≥ grade 2+ after LVR. Concomitant mitral valve repair for MR ≥ grade 2+, on either preoperative echocardiography or immediately after LVR, results in favorable late clinical and echocardiographic outcome that proved to be similar to patients without concomitant mitral valve repair, despite more advanced disease.

Keywords: Left ventricular reconstruction (LVR) • Dor procedure • Mitral regurgitation • Restrictive mitral annuloplasty (RMA) • Ischemic heart failure (IHF)

INTRODUCTION

Remodeling of the left ventricle (LV) in ischemic cardiomyopathy leads to systolic and diastolic dysfunction, and frequently to functional mitral regurgitation (MR) as a secondary phenomenon [1–5]. Surgical ventricular restoration or left ventricular reconstruction (LVR) restores LV shape, reduces LV volume, and improves pump function in patients with ischemic cardiomyopathy [6,7]. The impact of LVR on MR – both early and late – is unclear, as is the indication for concomitant correction of the MR during LVR. Our management of MR in patients undergoing LVR encompasses performing a restrictive mitral annuloplasty (RMA) when MR ≥ grade 2+, established either preoperatively or immediately post-LVR. In this study, we evaluated the results of this strategy in patients with ischemic heart failure (IHF), who underwent LVR, with or without concomitant RMA, with a focus on late clinical and echocardiographic outcome.

MATERIALS AND METHODS

Ninety-two consecutive patients with ischemic cardiomyopathy and heart failure (NYHA class III or IV and LV ejection fraction ≤ 35%) underwent LVR between April 2002 and April 2007. Patients were considered eligible for LV reconstructive surgery when they had LV dilatation following an antero-septal myocardial infarction with an echocardiographically derived Wall

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Motion Score Index (WMSI) ≤ 2.5, or with evidence of contractile reserve when WMSI exceeded 2.5, as described earlier [8]. Patients were attributed to an RMA and no-RMA group, depending on whether or not concomitant RMA had been performed.

Patient characteristics

There were 76 men and mean age was 61 ± 10 years. All patients presented with IHF, 76 patients (83%) were in NYHA class III. Mean LVEF was 25 ± 7% (range 12–35%). Median interval after myocardial infarction was 36 months (range 1–360). Logistic EuroSCORE averaged 10 (range 3–42). All patients underwent elective surgery. Preoperative moderate to severe (≥ grade 2+) MR was present in 40 patients (43.7%) on transthoracic echocardiography (TTE). Patient characteristics are summarized in Table 1.

Preoperative echocardiography

A transthoracic echocardiogram was performed within 5 days prior to surgery. When significant mitral and/or tricuspid regurgitation was demonstrated on TTE, transesophageal echocardiography (TEE) was additionally performed to further evaluate the severity and mechanism of the regurgitation. The severity of mitral and/or tricuspid regurgitation was graded semi-quantitatively from color-flow Doppler acquisitions in the conventional parasternal long-axis and apical four-chamber images. Mitral and tricuspid regurgitation was characterized as: mild, 1+ (jet area/leaf or right atrial area <10%); moderate, 2+ (jet area/
ment in all patients is shown in Fig. 1.

transmitral diastolic gradient were assessed.

fraction, LV dimensions and volumes, presence of MR, and

at the outpatient clinic. From these examinations, LV ejection

ectograms were performed after surgery, starting just

toms before surgery and at annual follow-up. Serial transthoracic

the NYHA classi

heart failure after surgery. Functional status was assessed using

Patients were maintained on optimal medical treatment for

procedure performed. A flowchart demonstrating MR manage-

Clinical and echocardiographic follow-up

Patients were maintained on optimal medical treatment for

heart failure after surgery. Functional status was assessed using

the NYHA classification for symptoms of heart failure. An inde-

pendent physician at the outpatient clinic evaluated the symp-

toms before surgery and at annual follow-up. Serial transthoracic

echocardiograms were performed after surgery, starting just

prior to hospital discharge and followed by annual examinations

at the outpatient clinic. From these examinations, LV ejection

fraction, LV dimensions and volumes, presence of MR, and

transmitral diastolic gradient were assessed.

Statistical analysis

Statistical analysis was performed using SPSS 16.0 statistical

software (SPSS Inc., Chicago, IL, USA). Categorical variables are

described as frequencies and percentages and compared using the

chi-square test with Yates’ correction. Continuous data are

expressed as mean ± standard deviation (SD) or median with

ranges and compared using the Student’s t-test for paired data.

The Kaplan-Meier method was used to model survival. Survival

between two groups was compared by the Mantel-Cox log rank

test. A P-value < 0.05 was considered significant.

RESULTS

Intraoperative management of MR

Preoperative TTE demonstrated MR ≥ grade 2+ in 40 patients. In

38 patients (95%), concomitant RMA was performed. RMA was

not performed in two patients, because of a completely calcified

posterior mitral annulus in one patient and a complicated pro-

cedure in another patient, making additional mitral surgery in-

appropriate. Fifty-two patients had preoperative MR < grade 2+.

Eight patients had no MR preoperatively; in these patients

MR did not appear after LVR. A total of 17 patients with MR

grade 1+ on preoperative examination showed increasing MR to

≥ grade 2+ immediately after LVR and underwent subsequent

RMA. In the remaining 35 patients, MR stayed < grade 2+ imme-

diately after LVR. The flowchart of MR management is shown in

Fig. 1.

None of the patients had primary organic valvular disease; in

all patients the mechanism underlying MR was systolic restriction

of both leaflets with annular dilatation. Median RMA ring size

was 26 (range 24–32). Apart from the patient with the accepted

MR grade 2+, intraoperative TEE demonstrated absent or mild

MR in all patients. In patients who had undergone concomitant

RMA, mean length of leaflet coaptation after mitral valve repair

was 8 ± 2 mm and mean transmitral diastolic gradient was

2.9 ± 1.7 mmHg.

Comparison of baseline echocardiographic

characteristics between RMA and no-RMA group

Based on above-mentioned criteria for mitral valve repair, 55

patients were attributed to the RMA group and 37 to the

no-RMA group. Comparing preoperative TTE data, WMSI in

the RMA group proved to be significantly higher than in the

no-RMA group (2.6 ± 0.5 vs 2.3 ± 0.5, P < 0.01), indicating more

and/or more severe regional LV wall-motion abnormalities and

hence an overall greater deterioration of LV function. In addition,

LV volumes and dimensions were significantly larger in the RMA

group (P < 0.01 for left ventricular end-systolic volume (LVESV),

left ventricular end-diastolic volume (LVEDV), left ventricular

end-systolic diameter (LVESD), and left ventricular end-diastolic
diameter (LVEDD)). These data are summarized in Table 3.

Early outcome

In-hospital mortality in the RMA group and no-RMA group

was 12.7% (seven patients) and 5.4% (two patients),

Table 2: Surgical data (n = 92)

<table>
<thead>
<tr>
<th></th>
<th>RMA group (n = 55)</th>
<th>No-RMA group (n = 37)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVR with patch (n, %)</td>
<td>53 (96.4%)</td>
<td>36 (97.3%)</td>
</tr>
<tr>
<td>Patch size (cm²) (mean ± SD)</td>
<td>13 ± 7</td>
<td>12 ± 8</td>
</tr>
<tr>
<td>Inferior wall plication (n, %)</td>
<td>8 (14.5%)</td>
<td>4 (10.8%)</td>
</tr>
<tr>
<td>Balloon/shaper size (ml) (mean ± SD)</td>
<td>109 ± 13</td>
<td>110 ± 11</td>
</tr>
<tr>
<td>Mitral valve annuloplasty (n, %)</td>
<td>55 (100%)</td>
<td>0</td>
</tr>
<tr>
<td>Median ring size (range)</td>
<td>26 (24–32)</td>
<td>-</td>
</tr>
<tr>
<td>Tricuspid valve annuloplasty (n, %)</td>
<td>20 (36.4%)</td>
<td>0</td>
</tr>
<tr>
<td>Median ring size (range)</td>
<td>28 (26–38)</td>
<td>-</td>
</tr>
<tr>
<td>CABG (n, %)</td>
<td>32 (58.2%)</td>
<td>26 (70.3%)</td>
</tr>
<tr>
<td>No. of distal anastomoses/patient (mean ± SD)</td>
<td>2 ± 1</td>
<td>3 ± 1</td>
</tr>
</tbody>
</table>

LVR: left ventricular restoration; CABG: coronary artery bypass grafting; LIMA: left internal mammary artery; RIMA: right internal mammary artery; BIMA: bilateral internal mammary artery; LV: left ventricle; ECC: extracorporeal circulation; IABP: intra aortic balloon pump.

![Figure 1](https://academic.oup.com/ejcts/article-abstract/41/1/74/484198/fig1)

The management chart of MR during LVR. MR: mitral regurgitation; RMA: restrictive mitral annuloplasty; no-RMA: no restrictive mitral annuloplasty; LVR: left ventricular restoration.
Table 3: Transthoracic echocardiographic data

<table>
<thead>
<tr>
<th></th>
<th>RMA group</th>
<th>No-RMA group</th>
<th>RMA group</th>
<th>No-RMA group</th>
<th>RMA group</th>
<th>No-RMA group</th>
<th>RMA group</th>
<th>No-RMA group</th>
<th>RMA group</th>
<th>No-RMA group</th>
<th>RMA group</th>
<th>No-RMA group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>Early postop.</td>
<td>P-value baseline versus early postop.</td>
<td>1-year FU</td>
<td>P-value baseline versus 1-year FU</td>
<td>2-year FU</td>
<td>P-value baseline versus 2-year FU</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>EF (%)</td>
<td>24 ± 7</td>
<td>35 ± 8</td>
<td>&lt;0.01</td>
<td>33 ± 12</td>
<td>&lt;0.01</td>
<td>30 ± 10</td>
<td>&lt;0.01</td>
<td>27 ± 7</td>
<td>39 ± 9</td>
<td>&lt;0.01</td>
<td>40 ± 6</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>LVESV (ml)</td>
<td>190 ± 88</td>
<td>150 ± 47</td>
<td>&lt;0.01</td>
<td>155 ± 56</td>
<td>&lt;0.01</td>
<td>116 ± 41</td>
<td>&lt;0.01</td>
<td>186 ± 61</td>
<td>136 ± 43</td>
<td>&lt;0.01</td>
<td>144 ± 38</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>LVEDV (ml)</td>
<td>249 ± 96</td>
<td>249 ± 86</td>
<td>NS</td>
<td>155 ± 56</td>
<td>0.01</td>
<td>116 ± 41</td>
<td>&lt;0.01</td>
<td>249 ± 96</td>
<td>136 ± 43</td>
<td>&lt;0.01</td>
<td>144 ± 38</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>LVESD (cm)</td>
<td>5.3 ± 1.1</td>
<td>5.0 ± 0.9</td>
<td>NS</td>
<td>4.9 ± 1.1</td>
<td>0.01</td>
<td>4.9 ± 1.1</td>
<td>NS</td>
<td>5.3 ± 1.1</td>
<td>4.9 ± 1.1</td>
<td>NS</td>
<td>4.9 ± 1.1</td>
<td>NS</td>
</tr>
<tr>
<td>LVEDD (cm)</td>
<td>6.8 ± 0.9</td>
<td>6.2 ± 0.9</td>
<td>&lt;0.01</td>
<td>6.2 ± 0.9</td>
<td>&lt;0.01</td>
<td>6.1 ± 1.1</td>
<td>&lt;0.01</td>
<td>6.8 ± 0.9</td>
<td>5.7 ± 0.8</td>
<td>&lt;0.01</td>
<td>6.2 ± 0.5</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

EF: left ventricular ejection fraction; LVEDV: left ventricular end-diastolic volume; LVESV: left ventricular end-systolic volume; LVEDD: left ventricular end-diastolic diameter; LVESD: left ventricular end-systolic diameter; WMSI: wall motion score index; postop.: postoperative; FU: follow-up; SD: standard deviation; NS: not significant.
systolic restriction of both leaflets with limited coaptation. The patient in the no-RMA group was in NYHA functional class II and showed grade 3+ recurrent MR (with severe pulmonary hypertension) due to progressive tethering of the mitral valve leaflets with systolic restriction on TTE. LV volumes and dimensions in this patient were still smaller than preoperatively, but showed slight progression after the initial surgically induced reduction. Preoperatively, this patient had MR grade 1+ which remained stable after LVR. At discharge MR was still grade 3+. Despite increased dosages of diuretics and ace inhibitors, MR remained stable grade 3+ at late follow-up.

Survival analysis was also performed comparing 36 months survival between patients with and patients without concomitant RMA. RMA: restrictive mitral annuloplasty; no-RMA: no restrictive mitral annuloplasty.

**DISCUSSION**

Functional MR in patients with ischemic cardiomyopathy is a secondary phenomenon caused by remodeling of the LV [1–5]. MR is related to LV dilatation and is caused by geometrical changes at the annular, subannular, and ventricular level. Annular dilatation, increased distance between annulus and papillary muscles, and increased distance between the papillary muscles alter and reduce coaptation of the mitral valve leaflets [12]. MR leads to volume overload that promotes further LV remodeling and carries an excess mortality in post-infarction patients, which is unrelated to the underlying degree of LV dysfunction [13–16]. The presence of MR has been shown to be an independent marker of excess mortality, even when the potential artificial increase in LVEF was taken into account. LVR restores LV shape, reduces LV volume, and improves pump function in patients with ischemic cardiomyopathy [6,7]. Correcting functional MR by RMA results in excellent and durable results, as we have published before [23].
The impact of LVR on MR, both immediately and during longer follow-up, remains unclear, as is the indication for concomitant correction of MR during LVR. On the one hand, immediate decrease of LV volumes and diameters, with the reduction of the distances between annulus and papillary muscle and between the papillary muscles, can lead to improved mitral valve leaflet coaptation [12,18]. Reduction of wall stress by the decrease in LV volumes and dimensions contributes to improvement in ventricular and papillary muscle function [9]. On the other hand, it is possible that LVR leads to a distortion of the geometry of the LV and subvalvular apparatus, causing an increase in MR. Moreover, possible further LV remodeling over time with gradual increase of LV volumes and diameters might lead to the appearance or recurrence of MR at midterm follow-up if MR is left untreated [9].

There is little debate to treat functional MR when it is moderate-severe or severe (MR grade 3+ or 4+). However, there is no consensus on how to treat mild or moderate MR (MR grade 1+ and 2+). Di Donato et al. propose to leave MR grade 2+ untreated. They demonstrated an excellent survival; however, a substantial percentage of patients (29%) was found to have at least a moderate degree of MR (grade 2+) at follow-up [18]. Prucz et al. demonstrated an overall reduction in MR grade with good functional results and excellent survival in a group of patients who underwent LVR with untreated moderate MR. However, 76% of the patients still had MR > grade 2+ at follow-up [12]. As such, a conservative approach to functional MR grade 2+ will leave a significant proportion of patients at risk for the potentially deleterious effects of MR, which are further LV remodeling and increased mortality. As has been demonstrated, a moderate degree of MR proves to be of hemodynamic importance in patients with reduced LV function and imposes significant clinical implications in post-infarction patients, even in those with minimal symptoms [15,25]. In the setting of ischemic MR, even a regurgitant volume as little as 30 ml is associated with a limited 5-year survival of 47%.

A conservative approach to functional MR grade 2+ might be related to the idea of an increased perioperative mortality caused by the additional intervention on the valve. In our study, perioperative mortality and morbidity were indeed higher in the RMA group, but it should be noted that patients in that group had more advanced disease, as demonstrated by the higher preoperative WMSI (more wall-motion abnormalities) and larger LV volumes and dimensions. MR should be regarded as the result of ongoing LV remodeling, and the increased perioperative risk should be interpreted against that background and, in addition, be weighed against the increased complication rate at longer follow-up associated with untreated MR. It has also been shown by others that concomitant mitral annuloplasty does not add by itself to the risk of the operation [9,20].

Aggressive correction of MR ≥ grade 2+ by RMA during LVR results in excellent functional improvement, favorable 36 months survival, and very low recurrence of MR. Moreover, elimination of MR leads to a similar functional improvement and equal survival comparing patients with and without preoperative MR ≥ grade 2+ (mean NYHA class at late follow-up 1.8 ± 0.9 and 1.7 ± 0.8 in the RMA and no-RMA groups, respectively, P = NS; 3-year survival 78.2% vs 80.7%, P = NS). This comparable outcome occurs despite the fact that patients with MR ≥ grade 2+ undergoing LVR have a more severely damaged LV, as also reflected by the higher early mortality and more frequent need of IABP support. Similar results were found by Athanasuleas and the RESTORE group, who demonstrated an increased 30-day mortality by twofold from 4% to 8.7%, but the 5-year survival after LVR was not influenced [7,21]. In our previously published meta-analysis, we found however that concomitant mitral valve surgery was associated with both an increased risk for early (RR = 1.57, P = 0.001) and late mortality (RR = 4.28, P < 0.001) [22]. The discrepancy in late outcome may be explained by the fact that concomitant mitral valve surgery – in the studies that were entered into the meta-analysis – comprises both mitral valve repairs and replacements. Mitral valve repair is associated with a better survival than mitral valve replacement (especially without preservation of the subvalvular apparatus) because of better preservation of ventricular contraction and fewer complications related to prosthetic deterioration, malfunction, or hypocoagulation [24]. Moreover, patient selection, surgical techniques (myocardial protection), and peri-operative management have improved over time.

LV reverse remodeling in IHF is also influenced by myocardial revascularization. Revascularization of viable but dysfunctional myocardium because of ischemia may resolve functional MR; however, this has proved to be very unpredictable [19]. The recently published STICH-trial, reporting over 1000 patients, randomized for either coronary artery bypass grafting (CABG, n = 501) and CABG and LVR (n = 499), did not demonstrate any benefit of LVR over CABG [17]. Since patients with severe post-infarction heart failure were not included in this trial (only 49% of patients were in NYHA class III or IV), and patients who would clearly benefit from LVR were not randomized, we do not consider that study representative for the patients evaluated in the current study. Moreover, both the reduction in LV volume (19% in the STICH-trial vs 60–69% (LVEDV) in our study) and the type of LV reconstruction (in 59% of the LVR patients in the STICH-trial, an endoventricular patch was used compared to 96–97% of the patients in this study) were different. Finally, it should be noted that in our study 42% of the patients in RMA group did not have coronary vessels suitable for revascularization and thus could not benefit from revascularization alone.

As published by our group recently, the recurrence rate of MR in patients who underwent RMA for MR ≥ grade 2+ in ischemic and non-ischemic cardiomyopathy and heart failure was 19% at a mean follow-up of 2.6 year [16]. These patients had similarly dilated LVs and reduced LVEF as the patients in the current study. The combination of reduction in LV volumes and reduction in wall stress by LVR with RMA probably contributed to the low recurrence rate of MR in these patients.

The long-term clinical and echocardiographic results of this study support our strategy of managing MR in patients undergoing LVR: when MR is absent preoperatively, neither appearance of MR directly after LVR or at late follow-up is observed. Rightfully, no concomitant RMA is performed in these patients. In patients with preoperatively MR ≥ grade 2+ and in patients showing increase of MR ≥ grade 2+ immediately after LVR, concomitant RMA is performed with excellent functional improvement, favorable 36 months survival, and very low recurrence of MR. In patients with MR < 2+ after LVR, concomitant RMA is not performed, which is justified by the low occurrence rate of MR at late follow-up.

CONCLUSIONS

Patients with IHF eligible for LV reconstruction have MR ≥ grade 2+ in 44% of cases. In one-third of IHF patients with MR < grade...
2+ preoperatively, MR increases to ≥ grade 2+ after LVR. Concomitant mitral valve repair for MR ≥ grade 2+, on either preoperative echocardiography or immediately after LVR, results in favorable late clinical and echocardiographic outcome that proved to be similar to patients without concomitant mitral valve repair, despite more advanced disease.

LIMITATIONS

Although the present study includes a relatively large sample size, more patients need to be studied to confirm the current results. Also, longer follow-up data are needed to evaluate the long-term results. Possibly, in some patients MR would have decreased after LVR and CABG alone. Our proven strategy of treating functional or ischemic MR ≥ grade 2+ by RMA, however, precludes any conclusions on this potential effect.

Conflict of interest: none declared.

REFERENCES


APPENDIX A. CONFERENCE DISCUSSION

Dr L. Menicanti (Milan, Italy): This paper deals with a very tough group of patients with mitral regurgitation after an acute myocardial infarction, low ejection fraction, and a large left ventricle, the type of patient that presents a very high mortality in all published series. The results you reported are different in one way, and you report the same survival in the two groups of patients with and without mitral regurgitation before the procedure. So it seems that with your techniques, you put a zero on the impact of the bad ventricle that is normally present with mitral regurgitation. I have two questions for you.

You have an incredibly low rate of recurrence of mitral regurgitation, around 2%, and I would like to ask if you have the same recurrence in the patients with mitral regurgitation that are treated, irrespective of the cause, ischemic or not, with the same dilatation of the ventricle?

Dr Klein: In a recently published paper in JACC in August of this year, we showed that the predictors of recurrence of MR in patients with ischemic and
non-ischemic cardiomyopathy at 2.6 years is around 19%. So probably the left ventricular reconstruction combined with restrictive mitral annuloplasty, by the reduction of left ventricular volumes and reduction in wall stress, is the cause of the low recurrence rate of MR.

Dr Menicanti: And the other thing, in your manuscript you described a group of patients in whom, after the procedures, some degree of mitral regurgitation is still present, and in this group of patients you went back onto extracorporeal circulation and you corrected the mitral regur. So I would like to ask you if this group of patients presents a more difficult postoperative period, higher mortality? How is it in the follow-up period?

Dr Klein: Mortality in this group of 17 patients is only one patient. He died of a sepsis in the ICU. So it is a low mortality of 5.4%. And both functional improvement and follow-up are essentially the same as in the other group of patients. So concomitant restrictive mitral annuloplasty in this patient group did not add to the surgical risk and did not pose a risk of reduced survival.

Dr Menicanti: Because we are always afraid to go back onto extracorporeal circulation with this type of patient, but it seems that there is no danger at all.

Dr Klein: We need a little bit more balloon pumping, of course, in these patients, but functional class improvement is the same, survival is the same, and mortality is low.

Dr M. Deja (Katowice, Poland): Your paper is very interesting, and I absolutely agree with the results you are presenting. I have, however, two questions to ask. Your group, and Professor Dion in particular, was always teaching that you should never assess mitral regurgitation while under anesthesia in the operating theatre. So how are you judging when it is appropriate to go back and do a repair on the patient that you actually did SVR on a minute ago? That is the first question.

And the other is less a question and more a remark. Although I agree with the results you are showing and I believe they are true, some kind of control group is missing. You are just making the assumption that if they both fail the same way, too.

Dr Menicanti: Not after the reconstruction, no. And to answer your second question, you are right, of course, there is no control group, but our previous results in both ischemic and non-ischemic patients demonstrating the efficiency of restrictive mitral annuloplasty made it standard practice in our hospital. So we performed restrictive mitral annuloplasty in this group of patients. But of course you are right, I cannot draw any conclusions as to whether the MR has decreased in a certain small group of patients.

Dr S. Bolling (Ann Arbor, MI): I have a question for you to reflect on Dr Menicanti’s comments. Clearly you thought those that needed annuloplasty and those that did not need annuloplasty were very different groups of patients, but in the ‘did not need annuloplasty’ group of those 52 patients, you had to go back on 17 or 33% of those. One question. Did that make you unhappy? And two, did you change your institutional policy of perhaps being more aggressive in performing an annuloplasty with lesser preoperative MR?

Dr Klein: Yes, you are right. First, we are very aggressive in performing restrictive mitral annuloplasty in these patients. We don’t do restrictive mitral annuloplasty for grade 1 MR, because it is not supposed to influence the left ventricular function and outcome in the future.

And you also wanted to know —

Dr Bolling: Did it make you unhappy to have to go back on bypass one-third of the time? That would make me unhappy. That seems like a high rate.

Dr Klein: It is all about the end results. You have to give a good treatment to these patients, and we know that leaving moderate MR or more in these patients results in a suboptimal outcome. So you have to go back and repair the valve.

Dr Bolling: I agree.

Dr K. Vural (Ankara, Turkey): Do your Kaplan—Meier curves and the subsequent survival comparison include operative mortality? Otherwise the perception of the diagram may be misleading, and, in my opinion, the legend or footnote of the diagram should contain this information. As far as I could see from your slides, there was a considerable difference between the mortalities of the mitral intervention group and the other group.

Dr Klein: Of course, in our Kaplan—Meier curve operative mortality is included, and in the first part of the graph you see a sharp drop that shows the operative mortality. And, yes, both groups are different. The patients in the RMA group have a more severe degree of disease, they have much more enlarged ventricles, and they therefore have a higher or a different mortality rate.