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# Left ventricular surgical remodelling: is it a matter of shape or volume?<sup>†</sup>

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#### Abstract

**OBJECTIVE**: Left ventricular surgical remodelling (LVSR) can be targeted to volume reduction (VR), (independently of the final shape) or to conical shape (CS). The aim of this study was to evaluate the long-term clinical and echocardiographic results of these two surgical strategies.

**METHODS**: From January 1988 to December 2012, 401 patients underwent LVSR: 107 in Group VR (1988–2001) and 294 in Group CS (1998–2012). The latter group of patients had lower ejection fraction (EF) and higher mitral and tricuspid regurgitation grade, with higher incidence of pulmonary hypertension. A propensity score model was built to adjust long-term results for preoperative and operative profiles.

**RESULTS**: Thirty-day mortality was 6.0%. Median follow-up interval time was 100 (3–300) months. Overall 20-year and event-free survival were  $36.1 \pm 7.8$  and  $19.4 \pm 7.2$ , respectively. No differences were found regarding 10-year survival (Group VR:  $55.1 \pm 4.8$  vs Group CS:  $64.2 \pm 4.2$ , P = 0.16) and event-free survival (Group VR:  $41.1 \pm 4.8$  vs Group CS:  $50.5 \pm 4.8$ , P = 0.12). However, Group CS provided better 10-year freedom from cardiac deaths ( $74.5 \pm 3.7$  vs  $60.4 \pm 4.8$ , P = 0.03) and from cardiac events ( $55.6 \pm 5.0$  vs  $45.0 \pm 4.9$ , P = 0.04). After propensity score adjustment, all the main outcomes were significantly better in Group CS. Multivariate Cox analysis confirmed this result; furthermore, to avoid any bias related to improved experience, 30-day mortality being higher in Group VR, we excluded the first month from Cox analysis: left ventricle VR (independently of the final shape) was still confirmed as the wrong approach. At the follow-up, Group CS showed significant improvement in EF (+18 vs +8%), end-systolic volume index (-35 vs -20%) and sphericity index (-6 vs +9%).

CONCLUSIONS: LVSR should aim to provide a more physiological shape (conical) rather than simple VR.

Keywords: Left ventricular reshaping • Surgical ventricular restoration • Akinesia • Left ventricular remodelling

# INTRODUCTION

The change of healthy myocardium into scar tissue after acute myocardial infarction (AMI) causes deep changes in morphology and function of the left ventricle (LV), especially after anterior AMI. The chamber dilates, the wall stress increases and the pump efficiency reduces, all these changes being at the basis of heart failure symptoms.

Surgical treatment of ventricular dilatation, which follows AMI, started in the 1950s, but till now is not a widely accepted form of treatment. In particular, there is no agreement as to whether the purpose of surgery has to be only volume reduction (VR) or to be the recovery of a more conical shape (CS), in addition to VR.

<sup>†</sup>Presented at the 27th Annual Meeting of the European Association for Cardio-Thoracic Surgery, Vienna, Austria, 5-9 October 2013. Recently, the STICH trial, based on a technique aimed for VR [1], questioned the benefit of adding left ventricular surgical remodelling (LVSR) to coronary artery bypass grafting in patients who had a previous anterior AMI [2]. Even if widely criticized (the eligibility criteria were changed during the study, and in 2003 the heart failure symptoms were abolished, the LV volume was not anymore an eligibility criterion, only ejection fraction (EF) of  $\leq$ 35% was kept and so on), the conclusions of the trial cannot be ignored. The debate, in our opinion, has not to defend preconceived hypothesis, but has to identify a better patient selection, proposing a new algorithm for the surgical treatment of this complication of AMI.

In this study, we evaluate our experience, started in 1988, in order to report the long-term results of two different strategies that had, as their targets, restoration of ventricular volume, as in the STICH trial, or of ventricular shape.

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### MATERIALS AND METHODS

## **Patient population**

From January 1988 to December 2012, 401 patients underwent LVSR; the entire cohort was split into two groups: the first one included patients in whom the target was LV VR, independently of the final LV shape (Group VR, n = 107, from 1988 to 2001); the second group included patients in whom the target was, together with VR, a final CS (Group CS, n = 294, from 1998 to 2012). The latter group of patients had lower EF, higher grade of mitral and tricuspid regurgitation and higher incidence of pulmonary hypertension. Retrospective analysis of our database was approved by the Institutional Review Board, which waived patient consent. Echocardiographic transthoracic assessment was performed preoperatively, at discharge from the hospital and during follow-up. Echocardiographic methods have already been reported [3]. Table 1 summarizes some clinical and echocardiographic characteristics.

# Surgical techniques

The Dor procedure (Group VR) was performed as previously reported [3]. The modified Guilmet procedure (Group CS) has been already described [4]. The septal reshaping (Group CS) was used when the septum was more involved than the anterior wall [5]. The septo-apical Dor procedure (Group CS) was used only in the case of scars that involved the distal portion of the septum and the apex. Mitral valve prosthesis was inserted inside the native mitral valve when coaptation depth was >10 mm, whereas in the remaining cases the mitral valve was repaired.

# **Definition of terms**

Early mortality was defined as any death that occurred in the first 30 days from surgery and late mortality as any mortality that occurred during the follow-up. Cardiac death was defined as

#### Table 1: Clinical, echocardiographic and surgical data

|                             | Group VR          | Group CS          | P-value |  |
|-----------------------------|-------------------|-------------------|---------|--|
|                             | ( <i>n</i> = 107) | ( <i>n</i> = 294) |         |  |
| Age (vears)                 | 62 ± 10           | 65 ± 9            | 0.001   |  |
| Female                      | 20 (19%)          | 48 (16%)          | 0.44    |  |
| Diabetes                    | 24 (22%)          | 86 (29%)          | 0.17    |  |
| Hypertension                | 45 (42%)          | 138 (47%)         | 0.20    |  |
| Dyslipidaemia               | 48 (45%)          | 129 (44%)         | 0.85    |  |
| ECV                         | 23 (21%)          | 74 (25%)          | 0.44    |  |
| Angina<br>NYHA              | 57 (53%)          | 162 (54%)         | 0.85    |  |
| Ш                           | 49 (46%)          | 118 (40%)         | 0.38    |  |
| 111                         | 42 (39%)          | 135 (46%)         |         |  |
| IV                          | 16 (15%)          | 41 (14%)          |         |  |
| EDV (ml/m <sup>2</sup> )    | 112 ± 42          | 117 ± 41          | 0.294   |  |
| ESV (ml/m <sup>2</sup> )    | 73 ± 33           | 80 ± 34           | 0.067   |  |
| EF (%)                      | 39 ± 10           | 32 ± 11           | < 0.001 |  |
| MR (0 to 4+)                |                   |                   |         |  |
| 0                           | 56 (53%)          | 71 (24%)          | < 0.001 |  |
| 1+                          | 24 (22%)          | 74 (25%)          |         |  |
| 2+                          | 11 (10%)          | 79 (27%)          |         |  |
| 3+                          | 14 (13%)          | 35 (12%)          |         |  |
| 4+                          | 2 (2%)            | 35 (12%)          |         |  |
| TR (1+ to 4+)               |                   |                   |         |  |
| 0                           | 87 (81%)          | 132 (45%)         |         |  |
| 1+                          | 16 (15%)          | 97 (33%)          | <0.001  |  |
| 2+                          | 2 (2%)            | 44 (15%)          |         |  |
| 3+                          | 2 (2%)            | 15 (5%)           |         |  |
| 4+                          | 0                 | 6 (2%)            |         |  |
| sPAP (mmHg)                 | 35 ± 11           | 45 ± 13           | <0.001  |  |
| Akinesia/dyskinesia<br>LVSR | 32/75             | 230/64            | <0.001  |  |
| Dor                         | 107 (100%)        | 40 (13.6%)        |         |  |
| Guilmet                     | -                 | 38 (12.9%)        |         |  |
| SR                          | -                 | 216 (73.5%)       |         |  |
| CABG                        | 97 (91%)          | 226 (77%)         | 0.002   |  |
| MVS                         | 24 (22%)          | 178 (61%)         | <0.001  |  |
| Repair                      | 21 (20%)          | 144 (48%)         | <0.001  |  |
| Replacement                 | 3 (2%)            | 34 (11%)          | 0.008   |  |
| TVA                         | 4 (4%)            | 94 (32%)          | <0.001  |  |
| DeVega                      | 4 (4%)            | 16 (54%)          | <0.001  |  |
| Band                        | 0                 | 78 (26%)          | <0.001  |  |

ECV: extracardiac vasculopathy; NYHA: New York Heart Association; ED: end-diastolic volume; ESV: end-systolic volume; EF: ejection fraction; MR: mitral regurgitation; TR: tricuspid regurgitation; sPAP: systolic pulmonary artery pressure; LVSR: left ventricular surgical remodelling; SR: septal reshaping; CABG: coronary artery bypass grafting; MVS: mitral valve surgery; TVA: tricuspid valve annuloplasty.

any death due to cardiac causes; patients who experienced sudden death or unexplained death were considered as having cardiac death. Cardiac events were defined as cardiac death, cardiac reoperation, hospitalization for heart failure, heart transplant and New York Heart Association (NYHA) Class III/IV. The term 'any event' was defined as all of the above events, including all deaths and any cause.

# Follow-up

All patients were clinically followed up: the most recent information was obtained by calling the patient or the referring cardiologists. Follow-up was 100% complete and ended in March 2013. In 197 patients, an echocardiographic control was collected.

### Statistical analysis

Results are expressed as mean (±standard deviation) and median value. Categorical variables were reported as counts and percentages. Differences between the two groups were evaluated by means of independent *t*-test (continuous variables) and  $\chi^2$  test (categorical variables). A saturate logistic regression model was used to obtain the propensity score using Group A as reference (goodness-of-fit c-statistic 0.83). Different parametric models were used to assess changing of hazard function across time; in all cases, hazard risk peaked at 1 month (early phase). Hence, risk factors for early mortality were investigated by means of stepwise binary logistic regression, entering into the initial model all variables already reported [3]. The results were reported as odds ratio, 95% confidence limits (CLs) and P-value. Ten-year survival curves were obtained with the Kaplan-Meier method and adjusted using the propensity score; significant difference was evaluated with the log-rank test. Time-to-event analysis was performed by a multivariable Cox proportional-hazard regression (see stepwise logistic regression). The results of Cox analysis were reported as hazard ratio (HR), 95% CI and P-value. Changes in LV volumes and EF from preoperative to follow-up period have been evaluated by means of longitudinal linear mixed-model regression for repeated measurements. Changes in NYHA class and mitral regurgitation (MR) grade across time have been evaluated by means of longitudinal ordinal logistic regression for repeated measurements. The propensity score was forced in all the regression analyses to adjust all the models for preoperative and operative differences. For all tests, a *P*-value of <0.05 was significant. The SPSS software (SPSS, Inc., Chicago, IL, USA) was used.

### RESULTS

Table 1 reports the clinical, echocardiographic and surgical data of the two groups. All patients had a Q-wave anteroseptal myocardial infarction of different extents. Operative mortality was 6.0% (24 cases), significantly higher in Group VR (12, 11.2 vs 12, 4.0%, P = 0.014). Causes of death were cardiac in 19 cases (low cardiac output in 17 and intractable arrhythmias in 2) and non-cardiac in the remaining 5 cases (pneumonia in 3 and stroke in 2).

Overall 20-year and event-free survival were 36.1 ± 7.8 and 19.4 ± 7.2, respectively. No differences were found regarding 10-year unadjusted survival (Group VR: 55.1 ± 4.8 vs Group CS: 64.2  $\pm$  4.2, P = 0.16) and event-free survival (Group VR: 41.1  $\pm$  4.8 vs Group CS:  $50.5 \pm 4.8$ , P = 0.12). However, Group CS provided better unadjusted 10-year freedom from cardiac deaths (Group VR:  $60.4 \pm 4.8$  vs Group CS:  $74.5 \pm 3.7$ , P = 0.03) and from cardiac events (Group VR: 45.0 ± 4.9 vs Group CS: 55.6 ± 5.0, P = 0.04); after propensity score adjustment, all the main outcomes were significantly better in Group CS than in Group VR (Table 2, and Figs 1 and 2). Stepwise logistic regression and Cox analyses adjusted for propensity score showed that simple LV VR (independent of the final shape) was a poor choice for both early and longterm cardiac outcomes (Table 3). To avoid any bias related to improved experience, 30-day mortality being higher in Group VR, we excluded the first month from Cox analysis: LV VR (independent of the final shape) was still confirmed as the less effective approach.

After a median follow-up of 63 (IQR = 19–103) months, 96 patients died, 74 of cardiac and 22 of non-cardiac causes. Further cardiac procedures were performed in 9 cases (4 heart transplants, 1 for MR recurrence, 2 for worsening untreated MR and 2 for LV assist device implant); 84 of 294 patients surviving first perioperative months were readmitted into the hospital due to new onset of heart failure, 41 in Group VR and 43 in Group CS.

At the end of follow-up, 281 survived, with a median follow-up of 100 months (minimum 3 months and maximum 300 months), 48 in Group VR and 233 in Group CS. Among them, 241 (85.8%) were in NYHA Class I or II (38, 79.2% in Group VR and 203, 87.1%

|                             | Group VR   | Group CS   | P-value |
|-----------------------------|------------|------------|---------|
|                             | (n = 107)  | (11 = 294) |         |
| Survival                    |            |            |         |
| Unadjusted                  | 55.1 ± 4.8 | 64.2 ± 4.2 | 0.16    |
| Propensity score-adjusted   | 48.3 ± 4.5 | 69.6 ± 4.0 | 0.003   |
| Freedom from cardiac deaths |            |            |         |
| Unadjusted                  | 60.4 ± 4.8 | 74.5 ± 3.7 | 0.032   |
| Propensity score-adjusted   | 54.8 ± 4.6 | 78.2 ± 3.9 | 0.002   |
| Freedom from cardiac events |            |            |         |
| Unadjusted                  | 45.0 ± 4.9 | 55.6 ± 5.0 | 0.042   |
| Propensity score-adjusted   | 39.2 ± 4.5 | 61.2 ± 4.8 | 0.025   |
| Event-free survival         |            |            |         |
| Unadjusted                  | 41.1 ± 4.8 | 50.5 ± 4.8 | 0.12    |
| Propensity score-adjusted   | 34.7 ± 4.5 | 56.3 ± 4.5 | 0.003   |

Table 2: Ten-year unadjusted and adjusted results

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Figure 2: Long-term event-free survival adjusted for propensity score. Group CS (solid line); Group VR (dashed line).

in Group CS). LV reshaping rather than VR was associated with NYHA class improving across time (coefficient  $-0.80 \pm 0.21$ , P = 0.002).

A postoperative echocardiogram, within 15 years from surgery, was obtained in 201 (70%) cases, 48 in Group VR and 153 in Group CS. Median follow-up was 67 months. Figure 3 shows a higher improvement in LV EF (VR: +5% vs CS: +24%), end-systolic volume (-15 vs -32%) and sphericity index (+9 vs -4%) in Group CS than in Group VR. Final sphericity index was 0.75 ± 0.13 (more spherical) in Group VR compared with Group CS (0.67 ± 0.10), P < 0.001 (Table 4).

# DISCUSSION

Ventricular remodelling after AMI includes changes in shape and, more importantly, in volume, together with a reduction of the systolic function, measured as EF or other parameters. The LV dilatation and thinning, which follows AMI, increase wall stress and, due to the inability of the remaining myocardium to compensate, cause further LV remodelling. Moreover, extensive ventricular 
 Table 3:
 Stepwise logistic regression and Cox analyses, adjusted for the propensity score

|                        | OR (95% CL)      | P-value |
|------------------------|------------------|---------|
| 30-dav mortality       |                  |         |
| LV volume reduction    | 3.3 (1.3-8.5)    | 0.013   |
| MVS                    | 3.2 (1.2-8.4)    | 0.018   |
| Age                    | 1.06 (1.01–1.12) | 0.031   |
| NYHA class             | 3.1 (1.6-6.5)    | 0.001   |
|                        | HR (95% CL)      | P-value |
| 10-year any deaths     |                  |         |
| LV volume reduction    | 1.3 (1.1–2.1)    | 0.033   |
| MVS                    | 2.7 (1.4–3.3)    | 0.000   |
| 10-year cardiac deaths |                  |         |
| LV volume reduction    | 1.8 (1.2–2.9)    | 0.002   |
| MVS                    | 2.6 (1.7-4.0)    | 0.000   |
| 10-year cardiac events |                  |         |
| LV volume reduction    | 2.0 (1.4–2.3)    | 0.001   |
| MVS                    | 2.3 (1.6-3.4)    | 0.000   |
| 10-year any events     |                  |         |
| LV volume reduction    | 1.7 (1.1–2.6)    | 0.003   |
| MVS                    | 2.6 (1.8–3.5)    | 0.000   |
|                        |                  |         |

LV: left ventricle; MVS: mitral valve surgery; NYHA: New York Heart Association; OR: odds ratio; HR: hazard ratio.

dilatation limits the benefits of isolated myocardial revascularization [6, 7], as patients with larger ventricular sizes show no improvement in the systolic function and higher incidence of cardiac death, myocardial infarction and hospitalization for heart failure.

LVSR was conceived to reverse symptoms of heart failure by reducing LV size, improving, as a consequence, the systolic function in patients with remodelled heart after a myocardial infarction in the left anterior descending (LAD) territory. On these theoretical bases, LVSR was applied since decades with different technical approaches. In the late 1980s, Dor reported a technique that remained, with some modifications (addition of the Fontan stitch and introduction of a shaper [8]), the most diffuse procedure for LVSR [9]. The aim of the Dor technique is reduction in the LV volume, irrespective of the shape obtained.

In the same decade, Guilmet et al. [10] published another technique, which combined the reduction in volume with a more CS, the 'overcoat technique'. This procedure remained obsolete for many years until our group, in search of more physiological solutions for surgical LV remodelling, rediscovered this elegant solution in 1998 and, with some modifications, started its clinical application [4]. The linear suture (anterior wall to septum) was later changed into an oval patch of different sizes (to obtain a reasonable end-diastolic volume), tailored in such a way that the new apex was as distal as possible, to maintain a sphericity index as low as possible. This evolution was called septal reshaping [5] and was one of the surgical strategies our group used to maintain a postoperative CS. In fact, the normal LV shape is a prolate ellipsoid with its long axis directed from apex to base, and its inflow and outflow being in continuity. LV ejection and filling is a function of systolic twisting and diastolic untwisting, which depends on the angular orientation of the oblique muscular fibres that are unique to the LV [11]. A minor change of 5-10° in the fibre orientation, as happens in more spherical heart, affects ventricular torsion and myocardial performance [12]. A change in sphericity seriously

affects the LV function. A myofibril contraction of 15% in a ventricle with a normal sphericity index (0.5, ellipsoid shape) generates an EF of 62%. At the same, with 15% fibre contraction, the EF falls below 40% if the sphericity index approaches 1 (spherical shape) and goes up to  $\geq$ 80% if the sphericity index approaches 0 (extreme ellipsoid) [13]. Thus, maintaining a sphericity index as low as possible must be one of the aims of LVSR.

The benefit of ventricular reduction in patients with postinfarctual akinesia or diskinesia of the anteroseptal wall was challenged by the STICH trial [2], which demonstrated that there was no survival or clinical advantage in patients who had LVSR and coronary artery bypass grafting (CABG) when compared with equivalent patients who had CABG without LVSR. Results of this trial caused a strong debate, as inclusion criteria were changed during the recruitment of patients and, more importantly, because of the lack of demonstration of left ventricular end-systolic volume  $\geq 60$  ml/ m<sup>2</sup> and akinesia  $\geq$ 35% in the LAD territory. Nevertheless, the conclusions of the STICH trial, based exclusively on the Dor procedure [1], are focusing the core problem of ventricular reduction surgery: the benefits anticipated with surgical reduction in LV volume are counterbalanced by a reduction in diastolic distensibility [2].

The pathophysiological effects of the Dor procedure have been extensively studied. Even if, after surgery, the wall stress was reduced in the remote zone, in the border zone and in the infarct zone, this reduction was not able to restore the wall stress to



Figure 3: Echocardiographic evolution expressed as percentage variation between preoperative and follow-up values. Ejection fraction (red column), end-systolic volume index (blue column), end-diastolic volume index (green column) and sphericity index (violet column). \*P < 0.05 (evaluated with longitudinal linear mixed-model regression for repeated measurements).

normal values [14]. Wall stress reduction, furthermore, does not improve contractility in the border zone, as myocytes in that region are in most cases irreversibly damaged. Globally, it seems that reduction in wall stress cannot improve symptoms or increase survival, as suggested by the STICH trial.

The Dor procedure causes an increase in the EF, which is not a valid surrogate of the improvement in the systolic function, as LVSR, reducing by definition the end-diastolic volume, causes an increase in EF to different extents, independently of the effective stroke volume. Reduction in mechanical dyssynchrony [15, 16] improves mechanical efficiency and global LV performance through an improved synergic distribution of regional stress during the isovolumic contraction and relaxation phases. Globally, we can agree that the Dor procedure improves systolic function and mechanical efficiency by reducing LV wall stress and mechanical dyssynchrony, and increasing EF. Revascularization of dysfunctional myocardium contributes to improved systolic performance; the impact of the single procedure on global improvement is then impossible to quantify.

The most common negative effect of the Dor procedure is failure to restore a conical LV shape, as demonstrated by a sphericity index increase [14, 17], which can be at the basis of postoperative diastolic dysfunction. This effect is independent of the use of a ventricular shaper [14, 17]. The regional shape shows no significant changes, as indexes of curvature increase, but not to normal values, failing to optimize the ventricular shape. The remaining distortion may cause non-optimal filling and diastolic dysfunction [14]. Choi et al. [18] showed that a low curvature wall was characterized by a maximum in the transmural fibre stress and strain in the mid-wall region, while a steep subendocardial transmural gradient was present in a high curvature wall. In a clinical setting, end-diastolic elastance increases after surgery, showing a worsening diastolic function [19], insensitive to the enddiastolic LV pressure [17]. The diastolic dysfunction causes the Starling relationship to be depressed in most of the patients. Of 12 patients studied by Lee et al. [17], the predicted Starling relationship remained unchanged in 3, worsened in 8 and improved only in 1. Other studies reported similar findings [16]. This is reflective of reduced stroke volume at rest [14, 20] and failure to increase adequately the cardiac output under effort. In general, the improvement in systolic function is counterbalanced, in most cases, by worsened diastolic function.

Other important information was provided by a fluid dynamic model of the normal LV and of a LV after the Dor procedure, described by Doenst *et al.* [21]. They found that, in the normal heart, the fluid dynamic was such to remove blood from the apex

|                          | Group VR    |             |       | Group CS    |             |       | $P^1$ |
|--------------------------|-------------|-------------|-------|-------------|-------------|-------|-------|
|                          | Pre (48)    | Post (48)   | Р     | Pre (153)   | Post (153)  | Р     |       |
| EDV (ml/m <sup>2</sup> ) | 105 ± 34    | 95 ± 29     | 0.040 | 110 ± 34    | 86 ± 27     | 0.000 | 0.049 |
| ESV (ml/m <sup>2</sup> ) | 67 ± 28     | 57 ± 19     | 0.043 | 73 ± 34     | 50 ± 23     | 0.000 | 0.058 |
| EF (%)                   | 39 ± 11     | 41 ± 10     | 0.216 | 33 ± 10     | 41 ± 11     | 0.000 | 1.00  |
| Sphericity index         | 0.69 ± 0.10 | 0.75 ± 0.13 | 0.010 | 0.70 ± 0.10 | 0.67 ± 0.10 | 0.009 | 0.000 |
| MR ,                     | 1.0 ± 0.9   | 0.8 ± 0.6   | 0.156 | 1.8 ± 1.1   | 0.7 ± 0.6   | 0.000 | 0.31  |

#### Table 4: Ecocardiographic results

EDV: end-diastolic volume; ESV: end-systolic volume; EF: ejection fraction; SI: sphericity index; MR: mitral regurgitation; P: pre versus post; P<sup>1</sup>: post group VR versus post group CS.

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of the LV and to generate a vortex, which redirects the blood towards the outlet. Only 2% of the blood that entered the LV during diastole remains in the cavity after four cycles. After surgery, LV geometry became apple-shaped and the fluid dynamics were completely changed. Blood stagnated into the apex and was no longer redirected towards the outflow tract. As a consequence, 39% of the blood entering LV in diastole is still present in the cavity after four cardiac cycles. Blood washout was in any case similar before and after surgery.

Even with these potential disadvantages, the outcome of patients who underwent the Dor operation is satisfying. Survival at 5 years ranges from 61.5 to 72.9% [3, 22]. In this study, our 10- and 20-year freedom from death of any cause have been 60 and 36.1%, respectively.

Both survival and clinical results seem to be better according to the end-systolic volume index (ESVI) obtained after surgery. The best results are obtained when the postoperative values are <60 ml/  $m^2$ , with values  $\geq 60 \text{ ml/m}^2$  representing a risk factor for lower survival [23]. Patients who reach an ESVI of <60 ml/m<sup>2</sup> have a better preoperative profile, with higher EF and lower volumes. As a consequence, VR has to be less extensive (-26 ml/m<sup>2</sup>, from 85 to 59 compared with -50 ml/m<sup>2</sup>, from 109 to 50 [23]). Preoperative ESVI is important as well, as values higher than 70 ml/m<sup>2</sup> (73 [23] or 80 [7]) are necessary to obtain a favourable reverse remodelling of the LV cavity. However, whereas the EDV can be reduced as much as the surgeon considers necessary, the final ESV will depend not only on the EDV obtained. The systolic contraction will depend on the residual wall stress, on the curvature of the myocardial wall and on the amount of contractile recovery of the remote area, which will depend as well on the amount of fibrosis present preoperatively. Most of these variables are not foreseeable and the final outcome can be unpredictable.

Only a few papers compared volume-related techniques with shape-related techniques. Isomura *et al.* [24] reported a 7-year survival of 61.5 vs 72.1%, respectively (P = 0.041), and our group showed improved freedom from cardiac death in patients who underwent shape-related LVSR (86.6 vs 76.3%, P = 0.032) [3]. In this study, we confirmed that, after 10 years from surgery, clinical results still favour techniques aimed to obtain a more CS together with a reduction in volume. We recently started to apply again the Guilmet principle, as modified by us, in large ventricles, since elimination, when possible, of the akinetic area represented by a patch can be related to a improved stroke volume [25].

#### Limitations of the study

This study has several limitations. It is a retrospective analysis of many patients undergoing operations over a long period of time, when techniques, strategies and experience progressively increased and improved, and the retrospective nature of this study causes, by definition, a selection bias. Moreover, this article reports the evolution of a concept across time, starting from the VR to septal reshaping, and therefore although its retrospective nature represents a limitation, we believe that a retrospective study is the only chance we had to report our experience. However, even if propensity score could reduce the selection bias, it cannot protect against the time bias due to the fact that enrolment was made in different decades. Finally, being retrospective, some preoperative and follow-up data are missing or incomplete, and therefore, a complete vision of the preoperative status of the patients was not possible in some cases: Echocardiographic data

were obtained for 70% of the entire surviving population. Other preoperative data such as renal function and chronic obstructive pulmonary disease were incomplete (only 55 and 63%, respectively) and so not reported. Aortic atherosclerosis, neoplasms, smoking and provocative test data to assess myocardial viability were completely lacking.

Most descriptions of the effect of shape on function are only experimental or rely on assumptions never validated in human diseased hearts.

#### Conclusion

Our long-lasting experience with LVSR shows that long-term outcome is satisfying and confirms the validity of this procedure. Results at 10 years seem to be better when VR combined with CS, rather than a mere VR, is pursued, and there are many physiopathological considerations in favour of this concept. We believe that, as surgery must address different anatomical features, it is not possible to apply to every patient the same procedure. Surgeons must be ready to adapt to each patient the technique necessary to reach the best correction for the specific case, rather than attempting to adapt the patient to the technique they are able to master. Definitively, surgical flexibility is the key point to achieve an optimal treatment of these complex patients.

Conflict of interest: none declared.

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# APPENDIX. CONFERENCE DISCUSSION

**Dr R. Klautz** (Leiden, Netherlands): Your elegant presentation points to the fact that reshaping the ventricle is probably more important than reducing the volume. Many people have already said that, but you just gave us the proof that it has not only direct but also long-term consequences. But as you've found that the shape of the ventricle is so important, not every reshaping is the same. Have you tried to make it more quantifiable – for example, that patients with a higher conical index or sphericity index have a better outcome? Was there a

difference - not just that those with reshaping had better outcome - but was there a difference between patients with severe and patients with more limited sphericity index change?

Dr Di Mauro: We had patients with poorer sphericity index who in the long term probably had worse outcome. But when we started to change our minds and to reshape the ventricle, we no longer found sphericity index to be a problem because we addressed the issue of LV sphericity with a very long patch (median 60 cm) to create a very long longitudinal axis of the left ventricle and to reduce the transverse diameter of the ventricle. Using this technique, we are able to improve the sphericity index. So the sphericity index probably now is no longer an issue.

**Dr Klautz:** Did you use any measurement to decide how to reshape the ventricle? All of us have used the Mannequin which directs the volume, but it also directs the shape where the new apex needs to come.

**Dr Di Mauro**: No. We don't use the Mannequin because the Mannequin is set on the physiological LV volume, but the "new" ventricle has a wall which is completely akinetic which could result in the repaired ventricle being restrictive. So we prefer to use a long patch from deep septum to the apex with the aim of obtaining a more conical chamber. We don't use any particular measurement. We just set the 6 cm patch for 2 cm or for 3 cm depending on the volume of the ventricle. This is enough to obtain a conical chamber.

**Dr Klautz**: I have a final technical question. How do you deal with the remainder of the scar you leave in the ventricle? We usually plicate that.

**Dr Di Mauro**: The remaining scar is sutured on the new apex. Thus we have a third chamber that clots after surgery. We found in the new echocardiographic control that all the third excluded chambers were clotted inside.

Dr E. Mostafa (Cairo, Egypt): Considering myself as a student in the school of Foch working with Daniel Guilmet, I myself am inclined to use the technique of Guilmet that you used in about 15% of cases, as shown in your slide.

**Dr Di Mauro**: Yes, we used a modified Guilmet technique in 40 cases before the use of a patch. I think the overcoat technique is a good technique, but we preferred to move to the septal reshaping technique believing that it is more comfortable for the surgeon to place the patch and to obtain a very long longitudinal diameter.

Dr Mostafa: Yes, I completely agree because it's not considered to be the best approach in large aneurysms. I have two questions. Firstly, have you found a difference between the Guilmet technique and the Dor technique in your physiological shaping rather than anatomical shaping? My second question is about the incidence of ventricular dysrhythmias in these types of patients and how you dealt with them.

**Dr** Di Mauro: We have already published a paper comparing the Dor and Guilmet techniques before the advent of this new technique, and we found that probably the overcoat technique, the Guilmet technique, should be applied when we have a large ventricle with huge involvement of the septum so that we can obtain a very conical shape. When we use the Dor, we had some difficulties in excluding all the septum. Sometimes we had some restrictive chambers and these patients died because of a restrictive syndrome. So probably it's not a matter of comparison between two techniques but two different approaches. We should tailor the technique to the patient.

Dr R. Przybylski (Zabrze, Poland): Did you check the diastolic dysfunction? In our series we found that diastolic dysfunction correlates with volume rather than shape.

**Dr Di Mauro**: We are now moving to the second part of this paper to investigate diastolic dysfunction. But diastolic dysfunction is not easy to investigate because we do not have a lot of tools to define it. Since we started with this new technique, we have had no significant diastolic dysfunction with this new approach.