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Surgical Treatment of Heart Failure in Patients with Primary and Ischemic Dilated Cardiomyopathy

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ABSTRACT

Objective: The aim of this study was to show hemodynamic and clinical improvement of heart failure after reductive annuloplasty of double (mitral and tricuspid) orifices (RADO) in the treatment of ischemic (IDCM) and primary (PDCM) dilated cardiomyopathy.

Materials and Methods: From November 1986 to July 15, 2002, 341 patients underwent operations for dilated cardiomyopathy. The IDCM group consisted of 231 patients (68%) with a mean ejection fraction (EF) of 23.3%. From July 1991 to July 15, 2002, the 110 patients in the PDCM group (mean EF, 22.9%) underwent such operations.

Results: The postoperative 30-day mortality rate was 5.9% for the entire patient population, 7.3% for the IDCM group, and 2.7% for the PDCM group. Follow-up survival rates were 61.5% ± 4.0% at 5 years and 38.2% ± 8.0% at 14 years for the IDCM group and 43.9% ± 5.6% at 5 years and 21.3% ± 8.5% at 10 years for the PDCM group.

Conclusion: RADO corrects remodeling of the fibrous skeleton of the heart, changes the spherical geometry of the left ventricle, improves the hemodynamic action of the left and right ventricles, and slows down the progression of heart failure. We recommend the RADO procedure as an important associated procedure in the surgical treatment of IDCM and as a new surgical alternative for treating the early stage of PDCM immediately after the first decompensation.

INTRODUCTION

Ischemic mitral regurgitation is a common complication in end-stage ischemic dilated cardiomyopathy (IDCM). There are many reasons and factors that increase the operative risk: the poor preoperative hemodynamic conditions of these patients and technical difficulties in surgical procedures involving associated lesions such as diffuse and distal coronary artery disease, left ventricle aneurysm, and long aortic cross-clamping times.

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Primary dilated cardiomyopathy (PDCM) has been defined by the World Health Organization as the dilatation of the left, right, or both ventricles with impaired systolic function of unknown cause [Richardson 1996]. Patients with PDCM, particularly in the end stage, present a special clinical problem because of PDCM's refractoriness to medical treatment. Even with aggressive medical therapy, heart failure is associated with a high mortality rate and a poor prognosis.

Mitral and tricuspid regurgitation are unavoidable complications of end-stage dilated cardiomyopathy that significantly contribute to cardiac failure and predict an early lethal outcome. This poor prognosis has led to increasing interest in treating heart failure with different surgical procedures: conventional heart transplantation, cardiomyoplasty [Moreira 1990], partial ventriculectomy, and mitral and tricuspid valve repair [Bolling 1995, Radovanovic 1995]. Heart transplantation provides good short-term and long-term results, but the limited donor heart pool and contraindications for heart transplantation have stimulated an interest in alternative surgical options.

Our investigations of patients with IDCM and PDCM show severe remodeling of the left ventricle, compared with a control group of patients with normal hearts (Table 1).

IDCM and PDCM show similarities in cardiomegaly, in the size and shape of the left ventricle, and in the presence of mitral and tricuspid annulus dilatation and regurgitation (Table 2). The exception is that the mitral valve area is significantly larger in PDCM.

The first cardiac transplantation in Serbia was performed at our clinic in 1989. In 1991, we had to stop our heart transplantation program because of the civil war in the former Yugoslavia. The patients with PDCM who were waiting for heart transplantation have presented with end-stage severe mitral and tricuspid regurgitation. Encouraged by our experience since 1986 in the surgical treatment of patients with IDCM, we began in July 1991 the surgical correction of mitral and tricuspid regurgitation in those patients to improve their hemodynamic status. In August 1993, during the XVth Congress of the European Society of Cardiology in Nice, France, we first described the results in the first 17 patients with our new surgical method for treating end-stage heart failure of PDCM with reductive annuloplasty of double (mitral and tricuspid) orifices (RADO) [Radovanovic 1993].

The aim of this report is to describe our 16 years of experience in the surgical treatment of patients with IDCM and

Table 1. Transesophageal Echocardiographic Study: Left and Right Ventricular Remodeling in the IDCM and PDCM Groups*

	IDCM	*P*	Control Group	*P*	PDCM
MR, °	3.4 ± 0.5	<.001	0	<.001	3.7 ± 0.4
TR, °	2.3 ± 0.7	<.001	0	<.001	3.3 ± 0.7
SI	78.0 ± 11.0	<.001	48.9 ± 7.8	<.001	84.8 ± 11.9
MAA, cm^2	9.8 ± 2.1	<.001	6.2 ± 0.6	<.001	10.0 ± 2.7
MVA, cm^2	3.7 ± 0.7	.14	3.9 ± 0.3	<.001	4.5 ± 0.9
TAA, cm^2	11.6 ± 2.9	<.001	7.77 ± 0.81	<.001	14.0 ± 3.7
LVIDs, cm	6.2 ± 0.9	<.001	3.2 ± 0.4	<.001	6.1 ± 1.0
LVIDd, cm	8.1 ± 1.0	<.001	4.8 ± 0.5	<.001	7.2 ± 0.9
FS, %	15.7 ± 4.5	<.001	34.4 ± 6.3	<.001	15.9 ± 5.7
ESV, mL	196 ± 63.0	<.001	41.4 ± 12.4	<.001	211 ± 77.2
EDV, mL	274 ± 81	<.001	111 ± 27	<.001	285 ± 84.4
EF, %	26.0 ± 5.6	<.001	60.8 ± 6.0	<.001	27.4 ± 6.6
WALLS, kPa/cm^2	84.1 ± 17.6	<.001	43.2 ± 4.49	<.001	82.4 ± 18.7

*IDCM indicates ischemic dilated cardiomyopathy; PDCM, primary dilated cardiomyopathy; MR, mitral regurgitation; TR, tricuspid regurgitation; SI, sphericity index; MAA, mitral annulus area; MVA, mitral valve area; TAA, tricuspid annulus area; LVIDs, left ventricular systolic internal diameter; LVIDd, left ventricular diastolic internal diameter; FS, left ventricular fractional shortening; ESV, end-systolic volume; EDV, end-diastolic volume; EF, ejection fraction; WALLS, wall stress.

our 11 years of experience in the surgical treatment of patients in the terminal stage of PDCM.

MATERIALS AND METHODS

Between 1986 and 2002, 341 patients underwent operations for end-stage dilated cardiomyopathy at the University Clinic of Cardiovascular Surgery in Novi Sad, Yugoslavia. Of these patients, 249 (73%) exhibited signs of congestive heart failure before their operation. Most of the patients (281, 82%) had previously experienced myocardial infarction. Twenty-seven percent of these infarctions were posteroinferior, 48% were anterolateral, and 24% were posteroinferior and anterolateral. The mean time from myocardial infarction to operation was 31 months.

From November 1986 through July 15, 2002, 231 patients with ejection fractions (EF) below 30% underwent surgical intervention for IDCM (IDCM group). This patient group consisted of 210 men (91%) and 21 women (9%) aged 31 to 72 years (mean, 55.7 years). The mean preoperative EF was 23.3% ± 5.3%. The distribution of patients in the IDCM group by EF was as follows: 10% to 15%, 32 patients; 15% to 20%, 70 patients; 20% to 25%, 52 patients; and 25% to 30%, 77 patients. Preoperative investigations of mitral and tricuspid regurgitation registered by transthoracic Doppler echocardiography showed that the majority of patients had degree IV mitral regurgitation (120 patients, 52%) and degree III tricuspid insufficiency (118 patients, 51%). Preoperative hemodynamic data for the IDCM patient group were as follows: mean cardiac output (CO), 4.08 L/min (range, 2.0-6.6 L/min); mean cardiac index (CI), 2.26 L/min per m^2 (range, 1.2-4.6 L/min per m^2); mean central venous pressure (CVP), 7.19 mm Hg (range, 2.0-20.0 mm Hg); mean pulmonary artery pressure (PAP), 40.38 mm Hg (range 12.0-70.0 mm Hg); mean pulmonary capillary wedge pressure (PCW), 25.30 mm Hg (range, 7.0-38.0 mm Hg); mean pulmonary vascular resistance (PVR), 648.7 $dyn \cdot s \cdot cm^{-5}$ (range, 107-2000 $dyn \cdot s \cdot cm^{-5}$).

The PDCM group consisted of 110 patients, 83 men (75%) and 27 women (25%), aged 14 to 66 years (mean, 49.4 years), who underwent operations between July 1991 and July 15, 2002, for end-stage PDCM. All patients were in New York Heart Association classes III (29 patients, 26%) and IV (81 patients, 74%). The average duration of illness was 24.5 months, and the average number of preoperative decompensation events was 2.9 (range, 1-18) per patient.

Preoperative medical therapy in the PDCM group included digoxin (91.8%, 101 patients), diuretic drugs (100%, 110 patients), acute coronary event inhibitors (37.3%, 41 patients), and antiarrhythmic drugs (79.1%, 87 patients). Twenty patients (18.2%) were dependent on inotropic agent stimulation (dopamine, intravenous administration of adrenaline)

Table 2. Transesophageal Echocardiographic Study: Comparison of Echocardiographic Parameters in the IDCM and PDCM Groups*

	IDCM	*P*	PDCM
MAA, cm^2	9.8 ± 2.1	.38	10.0 ± 2.7
MVA, cm^2	3.7 ± 0.7	<.01	4.5 ± 0.9
LVIDs, cm	6.2 ± 0.9	.91	6.1 ± 1.0
LVIDd, cm	8.1 ± 1.0	.29	7.2 ± 0.9
EDV, mL	274 ± 81	.23	285 ± 84.4
ESV, mL	196 ± 63.0	.15	211 ± 77.2
SV, mL	78.2 ± 28.0	.73	80.4 ± 27.0
FS, %	15.7 ± 4.5	.95	15.9 ± 5.7
EF, %	26.0 ± 5.6	.26	27.4 ± 6.6
WALLS, kPa/cm^2	84.1 ± 7.6	.80	82.4 ± 18.7

*SV indicates stroke volume; other abbreviations are expanded in the footnote to Table 1.

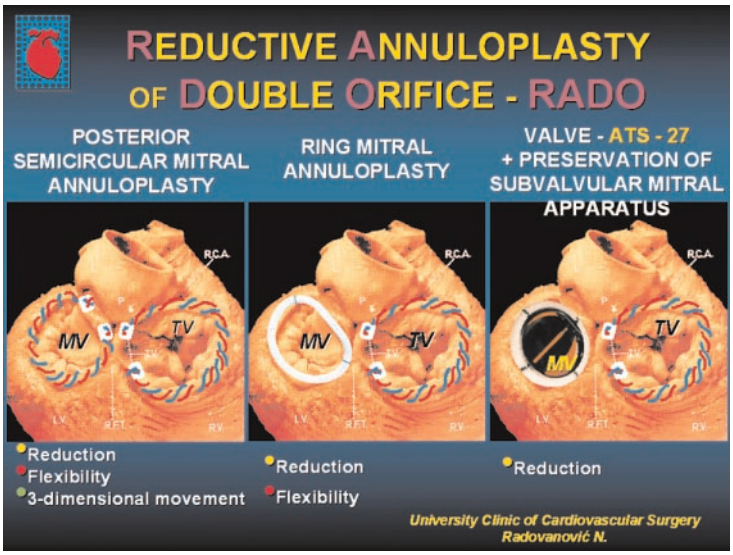

Figure 1. Reductive annuloplasty of double mitral and tricuspid orifices (RADO). RCA indicates right coronary artery; MV, mitral valve; TV, tricuspid valve.

at the time of operation. The mean preoperative EF in the PDCM group was 24.2% ± 5.0%. The distribution of patients according to EF was as follows: 10% to 15%, 6 patients; 15% to 20%, 33 patients; 20% to 25%, 33 patients; 25% to 30%, 29 patients; and 30% to 35%, 9 patients. Preoperative investigations of mitral and tricuspid regurgitation registered by transthoracic Doppler echocardiography showed that the majority of PDCM group patients had degree IV mitral regurgitation (89 patients, 79%) and degree III tricuspid insufficiency (53 patients, 48%). Preoperative hemodynamic data for PDCM group patients were as follows: mean CO, 3.7 L/min (range, 2.1-6.0 L/min); mean CI, 1.9 L/min per m^2 (range, 1.2-3.0 L/min per m^2); mean CVP, 8.1 mm Hg (range, 2.0-19.0 mm Hg); mean PAP, 35.4 mm Hg (range, 16.0-70.0 mm Hg); mean PCW, 22.7 mm Hg (range, 4.0-40.0 mm Hg); and mean PVR, 585 $dyn \cdot s \cdot cm^{-5}$ (range, 80-1829 $dyn \cdot s \cdot cm^{-5}$).

For both groups, operations were performed with the standard technique of cardiopulmonary bypass (CPB), hemodilution, mild systemic hypothermia, and membrane oxygenator. Myocardial protection was performed with cold crystalloid St. Thomas cardioplegic solution, topical hypothermia, and our own procedure.

The transseptal approach to the mitral valve was routine because of better valve exposure, even in the presence of a small left atrium, and because of the possibility of direct exploration and correction of the tricuspid annulus and a reduction in aortic cross-clamping time. In the IDCM group, reconstructive surgery was performed with mitral ring annuloplasty in 40 patients (17.3%) according to the procedure of Carpentier, and surgery was performed in 184 patients (79.6%) with posterior semicircular mitral annuloplasty with the use of Ethibond 2-0 suture (Ethicon, Somerville, NJ, USA) supported by polytetrafluoroethylene (Teflon) pledgets. This technique provides reduction and remodulation of the mitral annulus but preserves the normal flexibility, shape, and 3-dimensional movement of the mitral valve annulus (Figure 1).

In the IDCM group, 7 patients (3%) received a no. 27 bileaflet mitral prosthesis implant with the preservation of the mitral subvalvular apparatus. Tricuspid annuloplasty according to our own modification of De Vega annuloplasty (Figure 1) was performed in 223 patients (96.5%). Left ventricle aneurysmectomy was carried out in 19 patients (8.2%).

All IDCM patients underwent myocardial revascularization. The mean number of grafts was 2.3 per patient. Ninety-eight patients (43%) had diffuse coronary disease, and coronary endarterectomy had to be performed on one or more vessels.

In the PDCM group, mitral ring annuloplasty was performed according to the technique of A. Carpentier in 9 patients (8.1%), posterior semicircular mitral annuloplasty was carried out in 85 patients (77.2%), and a bileaflet mitral prosthesis was implanted in 16 patients (14.5%). All patients underwent tricuspid annuloplasty according to our own modification of the De Vega procedure.

Intraoperative transesophageal echocardiography (TEE) was used in both groups to recognize the morphology and the function of the mitral valve and left ventricle before and after RADO. TEE was performed with the Toshiba SSH-140A 5.0 MHz transesophageal ultrasound probe (Toshiba Medical Systems, Tokyo, Japan) before and after CPB.

The mitral annulus area (MAA) was calculated according to the Goldberg formula: $(\pi/4)x^2$, where x is mitral valve annulus diameter. Mitral valve area was calculated by the Doppler pressure half-time method. Meridian wall stress was calculated with a simplified Laplace formula, $P(R/Th)$, where P is the systolic pressure, R is the radius of the left ventricle, and Th is the thickness of the left ventricular wall.

To determine which dimension of the MAA should be reduced, we investigated the correlation of MAA with body

Table 3. Transesophageal Echocardiographic Study in the IDCM Group*

	Before Operation	After CPB	Normal Physiological Value	*P*
MAA, cm^2	9.8 ± 2.1	4.2 ± 0.9	4.5-7.0	<.01
MVA, cm^2	3.7 ± 0.7	3.6 ± 0.7	4.2 ± 0.9	NS
ESA, cm^2	27.5 ± 8.5	22.7 ± 6.0	—	<.01
EDA, cm^2	37.7 ± 7.6	33.1 ± 6.4	9.8-16.3	<.01
FAC, %	28.8 ± 10.3	34.9 ± 10.1	36-64	<.01
LVIDs, cm	6.2 ± 0.9	5.4 ± 0.9	2.7-3.7	<.01
LVIDd, cm	8.1 ± 1.0	6.5 ± 1.2	3.7-5.6	<.01
FS, %	15.7 ± 4.5	21.3 ± 5.8	36 ± 6	<.01
ESV, mL	196 ± 63.0	146 ± 53	33-98	<.01
EDV, mL	274 ± 81	243 ± 80	96-157	<.01
SV, mL	76.4 ± 28.7	103 ± 38.7	55-98	NS
EF, %	26.0 ± 5.6	39.5 ± 8.7	49-70	<.01
WALLS, kPa/cm^2	84.1 ± 17.6	58.0 ± 14.7	37.3 ± 7.1	<.01
% PLVWT	20.2 ± 5.1	26.6 ± 9.6	—	<.01

*CPB indicates cardiopulmonary bypass; NS, not significant; ESA, end-systolic area; EDA, end-diastolic area; FAC, left ventricular fractional area change; SV, stroke volume; PLVWT, posterior left ventricular wall thickness. All other abbreviations are expanded in the footnote to Table 1.

surface area by 2-dimensional multiplane TEE analysis in patients with a normal heart [Radovanovic 2002a]. The normal heart mean MAA was 6.2 cm^2 (range, 4.9-7.0 cm^2). A correlation between MAA and body surface area does exist and is positive, very high, and highly statistically significant ($P < .01$). The reduction in MAA is of essential importance in this operation, because the goal of reduction is not only to eliminate mitral regurgitation but also to change the spherical geometry of the left ventricle.

Continuous hemodynamic monitoring by a flow-directed Swan-Ganz pulmonary catheter and thermodilution allowed the measurement of CO, CI, CVP, PCW, and PVR before CPB, immediately after the operation, and during the next 24 to 48 postoperative hours.

All patients were contacted by telephone, and all examinations were done in the University Clinic of Cardiovascular Surgery, Novi Sad.

All data are presented as the mean ± SD. The Student *t* test was used for statistical analyses. Survival analyses were performed by the Kaplan-Meier method. A *P* value of less than .05 was considered statistically significant.

RESULTS

Immediate Postoperative Results

The analysis of hemodynamic parameters by Swan-Ganz catheter and thermodilution data for the IDCM group indicated significant improvements in the early postoperative period after myocardial revascularization and RADO. The mean CO increased immediately after CPB from 3.2 ± 1.1 L/min to 6.6 ± 1.7 L/min ($P < .01$) and remained at the physiological level in the observed period. The mean CI increased from 1.7 ± 0.6 L/min per m^2 before to 3.4 ± 0.9 L/min per m^2 immediately after CPB ($P < .01$). The mean PCW decreased after CPB from 18.7 mm Hg to 12.6 mm Hg. The means of all other hemodynamic parameters (CVP, PAP, and PVR) after CPB and during the postoperative observation period showed statistically significant improvements.

Significant improvements in hemodynamic and morphologic variables, as well as the elimination of mitral and tricuspid regurgitation, were registered by TEE for the IDCM group in the early postoperative period after RADO. Mitral regurgitation and tricuspid regurgitation were significantly reduced from 3.4° ± 0.5° to 0.1° ± 0.2° and from grade 2.3° ± 0.7° to 0.03° ± 0.2°, respectively. Changes in other variables obtained by TEE are presented in Table 3.

Perioperative hemodynamic monitoring also showed a significant improvement in the PDCM group after RADO. The mean CI increased from 2.0 ± 0.5 L/min per m^2 before CPB to 4.0 ± 0.6 L/min per m^2 immediately after CPB ($P < .05$). The mean PAP decreased from 32.5 ± 8.4 mm Hg to 19.8 ± 3.8 mm Hg, and the mean PCW decreased from 19.4 ± 4.0 mm Hg to 10.8 ± 4.4 mm Hg. The means of all other hemodynamic parameters (CO, CVP, and PVR) after CPB and during the postoperative observation period showed statistically significant improvements.

The mean mitral regurgitation decreased significantly from 3.7° ± 0.4° to 0.5° ± 0.7° ($P < .001$), and the mean tricuspid regurgitation decreased from 3.3° ± 0.7° to 0.6° ± 0.8°. Table 4 shows TEE parameter values before and after RADO.

The 30-day postoperative mortality rate for the patients of both groups was 5.9% (20/341). The rate for the IDCM group was 7.3% (17/231), and the rate for the PDCM group was 2.7% (3/110).

Follow-up Study

The closing date of the follow-up study for both groups was September 15, 2001. The follow-up rate for the IDCM group was 94%. The average time of follow-up was 42 months (range, 0-14 years). Survival rates at 5 and 10 years after the operation were 61.5% ± 4.0% and 38.2% ± 8.0%, respectively (Figure 2). The follow-up rate for the PDCM group was

Table 4. Transesophageal Echocardiographic Study in the PDCM Group*

	Before Operation	After CPB	Normal Physiological Value	*P*
SI, %	86.2 ± 74	71.7 ± 8.4	60	<.001
MAA, cm^2	10.0 ± 2.7	4.7 ± 1.2	4.5-7.0	<.01
MVA, cm^2	4.5 ± 0.9	3.7 ± 0.7	4.2 ± 0.9	<.01
ESA, cm^2	29.6 ± 9.9	24.3 ± 7.4	—	<.01
EDA, cm^2	40.9 ± 10.0	35.8 ± 8.5	9.8-16.3	<.01
FAC, %	28.7 ± 11.5	32 ± 7.8	36-64	<.01
LVIDs, cm	6.1 ± 1.0	5.5 ± 0.9	2.7-3.7	<.01
LVIDd, cm	7.2 ± 0.9	6.7 ± 0.8	3.7-5.6	<.01
FS, %	15.9 ± 5.7	21.2 ± 5.7	36 ± 6	<.01
ESV, mL	211 ± 77.2	160 ± 54.4	33-98	<.01
EDV, mL	285 ± 84.4	249 ± 61.0	96-157	<.01
SV, mL	78.2 ± 26.8	92.0 ± 27.5	55-98	<.01
EF, %	27.4 ± 6.6	38.1 ± 8.8	49-70	<.01
WALLS, kPa/cm^2	82.4 ± 18.7	62.4 ± 13.4	37.3 ± 7.1	<.01
% PLVWT	19.2 ± 6.4	22.8 ± 5.5	—	<.01

*CPB indicates cardiopulmonary bypass; ESA, end-systolic area; EDA, end-diastolic area; FAC, left ventricular fractional area change; SV, stroke volume; PLVWT, posterior left ventricular wall thickness. All other abbreviations are expanded in the footnote to Table 1.

100%. The average time of follow-up was 32 months (range, 0-10 years). Survival rates at 5 and 10 years were 43.9% ± 5.6% and 21.3% ± 8.5%, respectively (Figure 3).

DISCUSSION

Patients with IDCM have been difficult to manage, and clinical results in most series have been disappointing. In 1997, Hausmann et al, reporting a postoperative mortality rate of 50% for mitral valve replacement and 42.9% for mitral valve repair for a group of patients with EFs of <30%, maintained that heart transplantation was the only alternative [Hausmann 1997].

Many techniques of surgical treatment have been described. Conventional mitral valve replacement may impair left ventricular function because of the destruction of the mitral apparatus. To preserve normal mitral-papillary continuity during valve replacement, surgeons have developed many other techniques. Lillehei [Lillehei 1995] suggested maintaining papillary muscle–annulus continuity, at least posteriorly. Indications for mitral valve replacement are severely deformed leaflets, primary abnormalities of the leaflets, and calcification.

A better solution is mitral valve repair, which includes different procedures such as simple resection and suture of the mitral leaflets [McGoon 1960] and mitral annuloplasty [Kay 1986, Carpentier 1990, Oury 1994]. Clinical reports have shown that results are better in patients with mitral valve repair than in those with a mitral valve replacement. Flexible annuloplasty is better than a rigid ring [David 1989]. This advantage is in part due to the preservation of the subvalvular apparatus and the restoration of the normal shape of the mitral annulus, resulting in a better left ventricular function after valve repair [Rankin 1988]. Other known benefits of reconstructive procedures are a lower risk of thromboembolic and anticoagulant-related complications, a lower risk of infective endocarditis, and a lower reoperation rate because of the greater durability of the reconstructed mitral valve. Most often, the entire mitral apparatus appears normal, but an annulus dilatation is present. In all such cases, mitral annuloplasty is possible. Our procedure of first choice is a posterior semicircular mitral annuloplasty. This simple maneuver reduces and restores the normal shape of the mitral valve, avoids the rigidity of the mitral annulus that exists after Carpentier ring annuloplasty, and permits 3-dimensional movement. Other advantages of our technique are a fast procedure, a suture under less tension than with a rigid ring, and lower cost. Intraoperative TEE is mandatory, because it immediately demonstrates the quality of the repair and it is of utmost importance for improving the results of the surgery [Sheikh 1991].

Dilatation of the mitral and tricuspid annuli in end-stage ischemic cardiomyopathy presents a remodeling of the fibrous skeleton of the heart, and an important part is the remodeling of the left ventricle. Ventricular remodeling refers to the structural and shape changes associated with myocardial damage [Jugdutt 1995]. Myocardial infarction is associated with the remodeling of both the infarcted and the

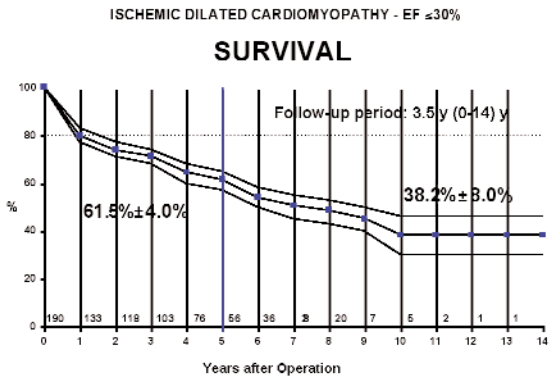

Figure 2. Ischemic dilated cardiomyopathy—survival at 5 and 10 years. EF indicates ejection fraction.

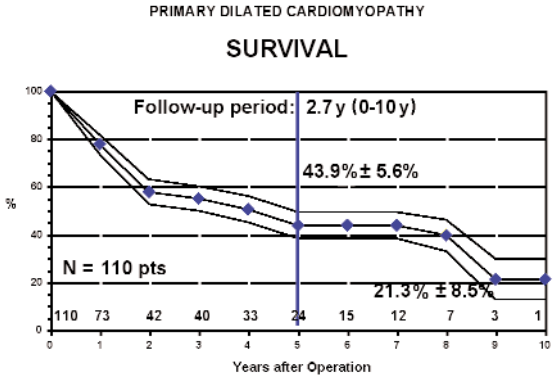

Figure 3. Primary dilated cardiomyopathy—survival at 5 and 10 years.

noninfarcted myocardium over a period of weeks to months that results in progressive ventricular dilatation, hypertrophy, and dysfunction. Early in the process, it is possible to prevent or reverse ventricular remodeling with unloading therapy [Cohn 1995]. Late in the process, progressive global left ventricular dilatation occurs with chronic mitral insufficiency. The structural remodeling of the myocardium shows an increase in myocyte length, a reduction in the number of myocytes per unit of ventricular wall thickness, and myocyte slippage. Fibrosis occurs in both ventricles in the segmental or perivascular interstices with increased levels of collagenase activity [Weber 1995].

Our IDCM patients had a dilated ventricle, and the mean left ventricular diastolic internal diameter was 8.1 ± 1.0 cm. The mean value of MAA in our patients was 9.8 ± 2.1 cm^2. Dilatation of the mitral and tricuspid annuli changes the geometry of the left ventricle, increases sphericity, and presents an independent factor of heart failure. RADO reverses the remodeling of the heart and improves the hemodynamic function of the left ventricle.

The operative risk in our series of 231 IDCM patients was acceptable—7.3 %. The explanation for this result is not only the good myocardial protection and operative technique and tactics used in the mitral valve surgery but also the optimal myocardial revascularization. Most of the patients had triple-vessel coronary artery disease, and the number of grafts was 2.3 per patient. In 98 patients (43%), we had to perform a coronary endarterectomy because of diffuse and distal coronary disease. However, coronary revascularization alone in this group of patients with severe remodeling of the heart would not have reduced mitral regurgitation [Fuchs 1998].

According to our results, significant hemodynamic and clinical improvement was attained after RADO in the patients with end-stage PDCM. This procedure is safe and has a low operative risk. In our series of 110 patients who underwent this operation, the 30-day postoperative mortality rate was 2.7%.

Aside from the similarities at the cellular level in the shapes and lengths of the myocytes, IDCM and PDCM are two different diseases. The possibilities for improvement with surgical treatment are better in IDCM cases, because the myocardial revascularization restores ventricular function in the viable but stunned or hibernating myocardium [Rahimtoola 1989]. Reconstructive procedures on the left ventricle presenting with a left ventricle aneurysm or akinesia improve the hemodynamic function of the left ventricle [Dor 1997].

In the terminal stages of IDCM and PDCM, tricuspid regurgitation is significantly increased because of the dilatation of the tricuspid annulus. Pulmonary hypertension is the consequence of left ventricle failure and the cause of the dilatation of the right ventricle and the tricuspid annulus, ie, the cause of right ventricle failure. Patients in end-stage heart failure experience many cardiac decompensations and die of left and right heart failure. In our experience, tricuspid annuloplasty is a mandatory procedure for improving right ventricle function in these patients.

In a review of recent publications concerning Batista's operation (partial left ventriculectomy), we found that the best results were obtained in patients in whom mitral valve repair was combined with partial left ventriculectomy. This fact confirms that the reduction of the mitral annulus is of the most importance for the improvement of left ventricle function [Bolling 1998].

At the beginning of our experience, we were of the opinion that only with the elimination of mitral and tricuspid regurgitation could we improve the function of the heart; however, the results we achieved were much better. We found the explanation for this fact in the improved geometry of the left ventricle. All values of parameters of left ventricle function obtained in the hemodynamic and TEE studies showed significant improvement [Radovanovic 2002b].

The perioperative morphologic analysis showed that apoptosis, bcl-2 expression, the proliferative activity of myocytes, the myofibrillar volume fraction, and the volume density of interstitial tissue may be useful in predicting the prognosis (progressive versus nonprogressive form) of patients with heart failure due to dilated cardiomyopathy [Zorc 2003]. RADO is a simple, fast, and inexpensive surgical procedure that reverses the remodeling of the fibrous skeleton of the heart and left ventricle. By using the RADO procedure, we can help patients who are candidates for heart transplantation by providing them with a low operative risk, better prospects for longer survival, and a better quality of life. This procedure may also be a bridge to heart transplantation, but for some patients in whom transplantation is impossible or contraindicated, RADO is a new strategy and the only alternative.

CONCLUSION

We consider remodeling of the fibrous skeleton of the heart with dilatation of the mitral and tricuspid annuli to be an independent factor for causing heart failure, especially if the MAA is greater than 7 cm^2.

RADO may reverse the remodeling of the fibrous skeleton of the heart, change the spherical geometry of the left ventricle, improve the hemodynamic action of the left and right ventricle, and slow down the progression of heart failure. We recommend the RADO procedure in the early stage of PDCM immediately after the first decompensation and as an important associated procedure during myocardial revascularization in patients with IDCM.

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REVIEW AND COMMENTARY

Invited Commentary from Mark Levinson, MD, Hutchinson Hospital, Hutchinson, Kansas, USA:

This report by Radovanovic et al from Novi Sad, Yugoslavia, is both a landmark publication and a remarkable achievement in surgical technique documented by late follow-up data. The authors are to be commended for logical development of a new surgical approach, clear explanation of their operative strategy, and continued follow-up of their results. In modern times, there is a tendency to publish early conceptual work. The excitement typical of new discoveries is shared by us all, but equal importance must be placed on documenting success over time. This report by Radovanovic et al is strengthened by the authors' persistence in acquiring their 94% late follow-up data. Because the RADO procedure is aimed at reducing the consequences of heart failure, it is acceptable to base conclusions on raw survival data to determine the effectiveness of the procedure.

One of the unique features of this report is the necessity for the authors to devise an alternative to transplantation as a treatment for end-stage dilated cardiomyopathy. In more affluent countries, transplantation has become the standard for such patients. Because of the constraints imposed by civil war in the authors' home country and the limitation of both economic resources and donor hearts, alternatives were sought. The authors developed a new and systematic approach based on a global understanding of the pathophysiology of heart failure and mitral regurgitation that incorporated all of the major advances of Carpentier, Bolling, Dor, Batista, Lillihei, and others in their surgical plan (with correct referencing of these prior accomplishments). The logic of their approach is clearly outlined, and their results and long-term follow-up data provide proof of concept.

Several aspects of this report are noteworthy. All patients were well chosen for the intended operation. No patient

with an EF greater than 35% was included. More than 79% of the patients had echocardiographic grade IV mitral regurgitation, and 48% had grade III tricuspid regurgitation. Nearly every patient was on digoxin and diuretics. The relatively low frequency of acute coronary event inhibitor use (37.3%) was likely due to the expense of these drugs rather than an intended undertreatment. The final report contains a large series of patients (N = 341), and all age ranges were included. The authors correctly broke out their results into ischemic and nonischemic subgroups, a division that is very important to the practicing surgeon. Unlike most publications, virtually every studied parameter of left ventricular function showed statistically significant improvement after the operation. A minor criticism is the absence of any explanation of how the authors chose their control population described in Table 1; however, the control data do appear to represent normal ventricles, so the comparison with their heart failure population is still valid.

My only concern is the long-term structural integrity of purse-string–style annuloplasties. In almost 80% of their cases, the authors used a modified De Vega annuloplasty on both the mitral and tricuspid valves. It has been shown in past studies that the recurrence rate for tricuspid regurgitation is higher with the De Vega technique than with a prosthetic ring annuloplasty. Long-term follow-up in this report was mostly by telephone interview, which is adequate to determine raw survival but not for late recurrence of mitral or tricuspid regurgitation. It would be important to know the frequency of recurrent mitral or tricuspid regurgitation before the widespread adoption of this technique. However, I agree that suture-based annuloplasty is a nonrigid reconstruction with some theoretical advantages over rigid or even semirigid prosthetic rings.

It is paramount that surgeons become more involved in the treatment of congestive heart failure. As more patients survive coronary ischemic events and live longer, the population of congestive heart failure patients is increasing. Heart failure is one of the leading causes of hospital admissions and financial drains on society, as well as a debilitating chronic illness causing tremendous suffering. Until the era arrives when myocyte proliferation by gene therapy is accomplished in vivo, the best hope for surgical treatment of congestive heart failure is myocyte and ventricular remodeling. Radovanovic et al have made an excellent case for a simple, inexpensive, and comprehensive procedure that is a major step forward in this field.