

A New Surgical Approach for Treating Dilated Cardiomyopathy with Mitral Regurgitation

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Objective - To evaluate the early outcome of mitral valve prostheses implantation and left ventricular remodeling in 23 patients with end-stage cardiomyopathy and secondary mitral regurgitation (NYHA class III and IV).

Methods - Mitral valvular prosthesis implantation with preservation of papillary muscles and chordae tendinae, and plasty of anterior cuspis for remodeling of the left ventricle.

Results - The surgery was performed in 23 patients, preoperative ejection fraction (echocardiography) varied from 13% to 44% (median: 30%). In 13 patients associated procedures were performed: myocardial revascularization (9), left ventricle plicature repair (3) and aortic prosthesis implantation (1). Early deaths (2) occurred on the 4th PO day (cardiogenic shock) and on the 20th PO day (upper gastrointestinal bleeding), and a late death in the second month PO (ventricular arrhythmia). Improvement occurred in NYHA class in 82.6% of the patients ($P < 0.0001$), with a survival rate of 86.9% (mean of 8.9 months of follow-up).

Conclusion - This technique offers a promising therapeutic alternative for the treatment of patients in refractory heart failure with cardiomyopathy and secondary mitral regurgitation.

Key words: mitral insufficiency, dilated cardiomyopathy, mitral implantation.

Heart failure has high incidence and prevalence¹ rates. Because statistics are not available in Brazil, North American data² are being used, which report that 400,000 new cases of heart failure are diagnosed annually. In spite of adequate drug administration, only 50% of these patients have a 5-year survival rate³. Forty percent of patients in functional class IV (NYHA), have a one-year survival rate³.

It is known that the treatment of choice for patients with congestive heart failure refractory to medical treatment, is heart transplantation. However, due to the reduced number of donors, indication for this procedure is restricted. Elderly patients, presence of other associated diseases, and an inadequate socio-economic environment, are some of the main factors that limit the use of transplantation, which is considered the gold standard¹. In this set of patients short-term outcome is not satisfactory having high mortality rates, because currently no effective surgical alternative to heart transplantation is available.

To comply with the above-mentioned restrictions to heart transplantation, and in the attempt to benefit a large number of patients, others surgical procedures have been suggested, mainly: cardiomyoplasty⁴⁻⁷, partial left ventriculotomy⁸⁻¹¹, mitral valve correction¹²⁻¹⁴ and myocardial revascularization itself¹⁵.

Mitral insufficiency is one of the factors causing severe hemodynamic changes in end-stage dilated cardiomyopathy. In addition to worsening of functional class, a higher number of hospital admissions, and a greater quantity of drugs administered to ameliorate symptoms, mitral regurgitation is also associated with a shorter survival of these patients¹⁶⁻¹⁹.

Mitral regurgitation appears late in advanced dilated cardiomyopathy and is caused by the interaction of the following factors: presence of segmentary alterations of ventricular contractility²⁰ papillary muscle dysfunction¹⁹, dilation of the mitral valve ring^{16,17,21}, and, in particularly, geometric alteration of the ventricular cavity¹⁸, which changes from an elliptic to as spherical shape, further deteriorating ventricular performance.

Reviewing our series of patients who underwent partial left ventriculotomy, as well as reports in the literature⁹,

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we noticed that significant improvement occurred in functional class, mainly in those patients who also underwent correction of mitral regurgitation. Based on these results, we decided to study, in patients with severe dilated cardiomyopathy the impact of mitral prosthesis implantation associated with procedures that determine remodeling of the left ventricle, through shortening of the left ventricle longitudinal axis.

Methods

Twenty-three patients with dilated cardiomyopathy

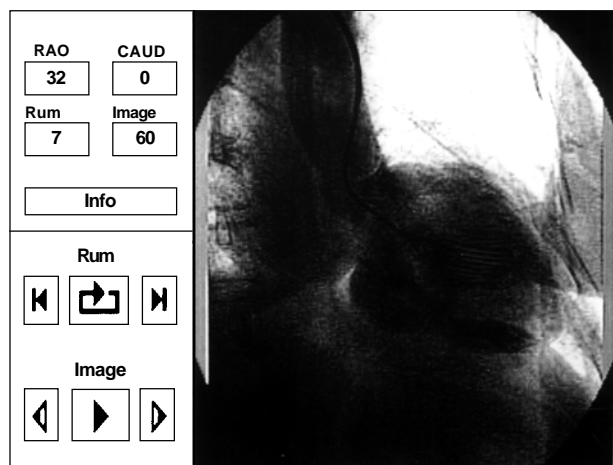


Fig. 1 - Left ventriculogram (OAD) in one of the patients showing left ventricle in systole and mitral regurgitation.

and moderate to severe mitral regurgitation underwent to this surgical procedure between December 1995 and December 1998. The cohort comprised 10 female and 13 male patients from 25 to 78 years of age (55±24). Sixteen patients were in NYHA class IV (72.7%) and 6 patients were in NYHA class III (27.3%). In spite of having received adequate and full medication, all patients had more than three hospital admissions in the prior 6 months. Six patients were in the intensive care unit dependent on inotropic support, and four were on an intraaortic counterpulsation balloon. Seven patients (30.4%) had undergone previous heart surgery (five for myocardial revascularization, one for mitral valve repair and one for partial left ventriculectomy). The preoperative ejection fraction (measured by transthoracic echocardiography) ranged from 13 to 44% (mean 30%) [fig. 1 (ventriculography)].

Regarding the causes of dilated cardiomyopathy, we observed that 11 cases (47.8%) due to ischemia; 6 (26%) were idiopathic or not diagnosed; 3 (13%) had valvular causes; 1 (4.3%) gestational, 1 (4.3%) viral and 1 (4.3%) was due to Chagas disease. Patient's clinical characteristics are detailed in table I.

Surgery was carried out in the usual way, by median sternotomy, with cannulation of the aorta and both vena cava. The mitral valve was approached through left longitudinal atriotomy. The prosthesis implantation preserved the valvular leaflets as well as the subvalvular system. The anterior leaflet was sectioned in the middle, its two margins being sutured separately at the level of the anterior com-

Table I – Distribution according to age, gender, cause, mitral insufficiency and evolution of functional classes

N	Age	Cause	Etiology Mitral	Insufficiency ECO	Functional Class Pre	Functional Class	Functional Class actual	Months of follow-up
1	68	M	Isc	M/S	III	II		39
2	72	M	I	S	IV	II	II	19
3	25	F	P	S	IV	II	II	18
4	54	M	I	M/S	III	II	II	15
5	36	M	I	S	IV	II/III	II/III	15
6	58	M	I	S	IV	II	II	15
7	75	F	Isc	M	IV	Death	Death	4° d PO
8	68	M	Isc	M	III	II	II	11
9	60	F	Isc	M	IV	II	II	11
10	50	F	I	S	III	II	IV	10
11	78	F	Isc	M	III	II		9
12	37	M	Isc	S	IV	II	III	9
13	38	M	Viral	S	IV	III	II/III	9
14	63	M	I	S	IV	Death	Death	20° d PO
15	40	F	I	S	IV	Death	Death	2° m PO
16	47	F	I	S	IV	II	II	7
17	52	M	Isc	S	IV	IV/III	II	7
18	49	F	I	S	III	II	II	7
19	61	M	Isc	S	IV	II	II	7
20	60	F	Chagas	S	III/IV	II	II	6
21	69	M	Isc	S	IV	II/III	II	6
22	72	F	I	S	III/IV	II		5
23	57	M	Isc	S	IV	III	II	3
Me	55,28							
SE	12,80							

Isc- ischemic; I- idiopathic; P- peripheral; M- moderate; S- severe.

missure and the other in the posterior commissure (fig. 2). This maneuver determines the shortening of the longitudinal axis of the left ventricle, in an equivalent distance between the free border of the cuspid and its insertion in the left atrioventricular ring. By traction of the base of the papillary muscle, we try to restore the ovoid shape of the left ventricle, which in conditions of end-stage cardiomyopathy tends to be spherical.

In most of the cases (85%), the prostheses used were biological with external diameters varying from 27 to 33mm. The procedure was carried out after approval of the institution and patients.

Based on the cause that led to the terminal cardiomyopathy, complementary surgical procedures were carried out, namely, myocardial revascularization with bypass (8 cases), and plicature of nonkinetic areas of the left ventricle (2 cases). None of these complementary procedures was, at preoperative assessment, of sufficient importance to justify refractory heart failure or surgery itself. They were carried out as coadjuvants to the main indication.

By definition, mitral regurgitation was not the cause of cardiomyopathy, but the consequence of it due to dilation of the mitral ring and the spherical shape of the left ventricle.

Only patients with moderate or severe mitral regurgitation evaluated by transesophageal echocardiography were considered for the procedure. They included patients with either significant regurgitation near the pulmonary veins or high "V" wave values of the pulmonary capillary pressure, or both.

The conventional transthoracic echocardiography and the left ventriculography carried out in the catheterization laboratory are not reliable because they underestimate the importance of mitral regurgitation in very low ejection fractions. Furthermore ejection fraction is overestimated due to lower afterload (mitral regurgitation).

All patients were followed up during the hospital stay and postoperative period by a member of the surgical team and a pre- and postoperative data comparison protocol was

carried out, analysing clinical outcome and echocardiographic findings.

Our central hypothesis was based on the assumption that clinical improvement would occur (using the patient's functional class as a parameter) correcting mitral regurgitation. Echocardiographic findings were compared basically at three stages: preoperative, postoperative during hospital stay before discharge and in the last evaluation. For this, analysis of variance using Friedman's test was carried out (using multiple comparison tests) to compare distributions of the pre- and postoperative functional class and the current class. The alpha error was established as $P < 0.01$.

Results

We observed a significant improvement in functional class in patients who underwent this procedure. At the last follow-up 17 (85%) were in functional class II, 2 (10%) in FC III and 1 (5%) in FC IV, even though no changes were made in the use of medication, when compared with the preoperative evaluation (fig. 3). The analysis of variance with Friedman's test resulted in a chi-square value of 24.11, with $P < 0.0001$, showing that the functional class improved significantly after surgery, and remained stable during follow-up (multiple comparison tests, with $P < 0.01$: preoperative > postoperative = follow-up).

Regarding ventricular function, which was measured by echocardiography, carried out by the same surgical team, it was observed that the ejection fraction that ranged between 13 and 44% with a mean of 28% prior to the operation, one month after ranged between 28 and 53% with a mean of 31%. Currently, the ejection fraction ranges between 10 and 60%, with a mean of 32% (fig. 4). Two patients merit special mention because their postoperative evolution substantiates the thesis advocated in this paper. Two months after the operation in one case and 3 months in the other, a severe worsening in functional class took place and was attributed to a considerable perivalvular leak. Its surgical repair, once again allowed restoration to functional class II.

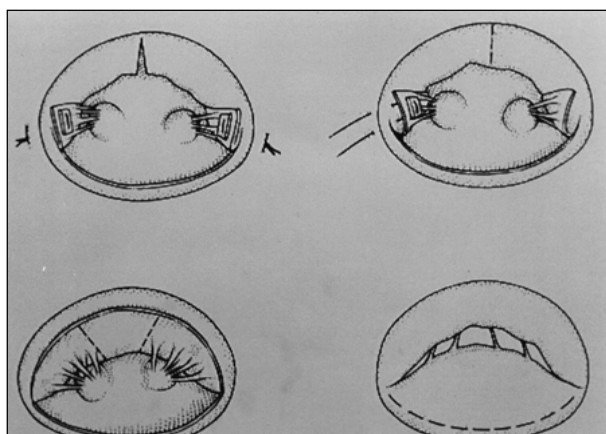


Fig. 2 - Technique of mitral prosthesis implantation preserving papillary muscles. The reduction in left ventricular longitudinal axis is equivalent to the distance between the leaflet free margin and the mitral annulus.

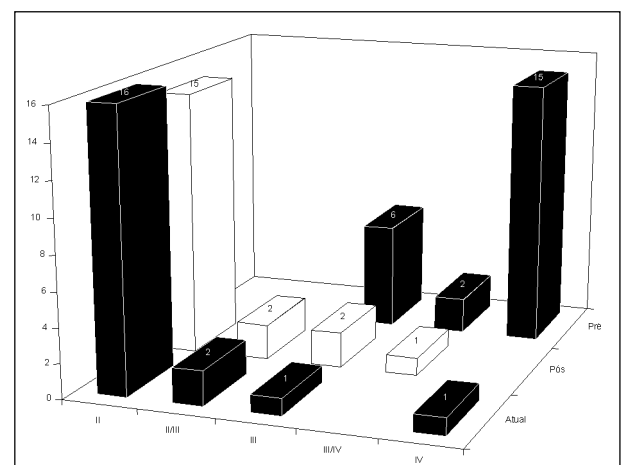


Fig. 3 - Functional classes pre, post (1st month) and present.

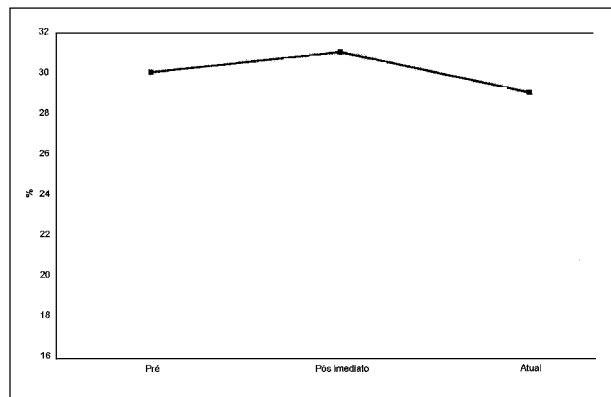


Fig. 4 - Ejection fraction before surgery, during first month, and at last evaluation.

Due to critical clinical conditions, intravenous vasoactive drugs were used in 6 patients and mechanical circulatory support (intraaortic counterpulsation balloon) was used in another before surgery. Of these, two had satisfactory postoperative evolution, and were in functional class II in the respective postoperative follow-up of 18 and 7 months.

With regard to valve disfunction during all surgical procedures, we observed that the valve escape was related to an inadequate coaptation of the valvular leaflets as well as to the dilatation of the mitral ring. In 13 patients, associated operations were performed (myocardial revascularization in 9 patients, ventricular aneurysm plicature repair in 3) and aortic prosthesis (1).

Bypass time ranged from 60 to 123min (mean 95min), and aortic clamping, varied from 0 to 80min (mean of 43min). Antegrade blood cardioplegic solution was used in 17 patients, and in 6 patients, no clamping was used. Only one patient needed intraaortic balloon counterpulsation after cardiopulmonary bypass.

During the evolution of the patients in the intensive care unit (ICU), we observed that six patients required vasoactive drugs for a period of over 72 hours, of which four were (3, 7, 9 and 15 months after the operation) in FC II and two (7 and 15 months after the operation) were in FC III.

Two early deaths occurred [2/23 (8.7%)]; one late death [1/21 (4.8%)] occurred at the two month follow-up. The cause was low cardiac output on the 4th postoperative day in one patient, and upper digestive tract hemorrhage on the 20th day in another who had significant concurrent diseases and had had a previous partial ventriculectomy 6 months earlier. The late death on the 60th postoperative day occurred in a patient with dilated cardiomyopathy who had satisfactory immediate evolution and had ventricular fibrillation during the emergency admission.

Four patients were re-admitted with heart failure, two undergoing the new surgical approach because of perivalvular leak, one patient due to bradyarrhythmia and another because of myocardial failure.

The postoperative evolution of functional class, ejection fraction, and ventricular diameters may be seen in tables I and II and figures 4 and 5. The actuarial survival curve is shown in figure 6. The follow-up times (in months) of each patient are reported in table I.

Discussion

Others than transplant surgeries selected for end-stage dilated cardiomyopathy^{4,8,9,11} refractory to conventional medical treatment have not met the expectations of improved performance in ventricular function. Most of the patients have not exhibited improved short-term survival rates or adequate quality of life^{7,9,11,15}. All these surgeries have thoroughly reported shortcomings, such as high mortality rates in patients who undergo partial left ventriculectomy⁹⁻¹¹, medium-term failure of the graft due to fatty degeneration of the skeletal fibers in cardiomyoplasty^{6,7} and incomplete resolution of heart failure with myocardial revascularization¹⁵. Considering the positive results reported in the literature¹²⁻¹⁴ concerning mitral valve reconstruction, the technique descri-

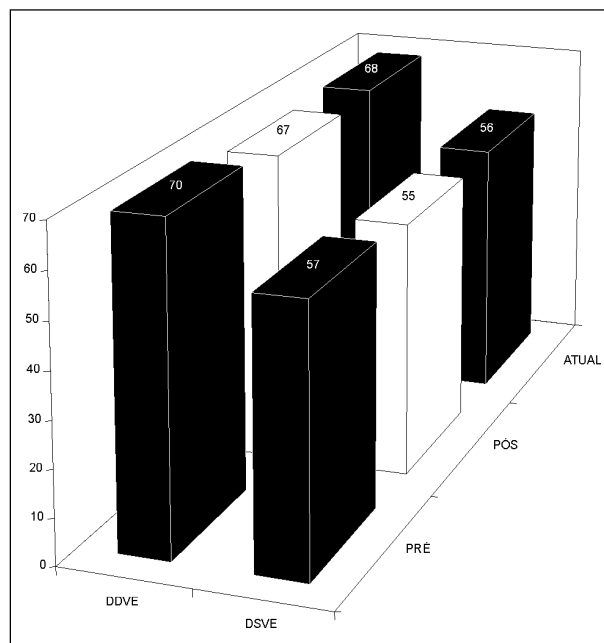


Fig. 5 - Comparison of diameters (systolic and diastolic) of left ventricle in three phases showing insignificant reduction in left ventricular chamber.

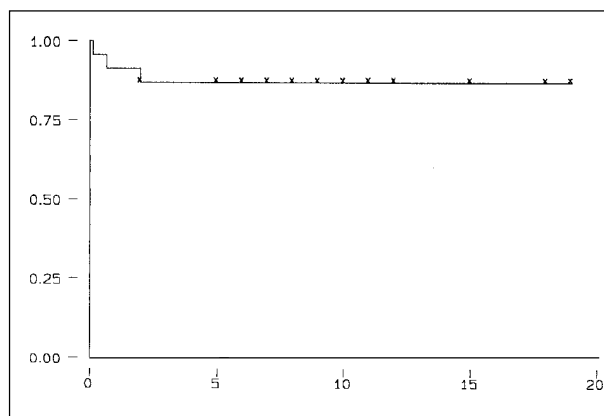


Fig. 6 - Actuarial survival curve of the 23 patients showing a stable life expectancy after the immediate risk of surgery.

Table II – Ejection fraction (transthoracic echocardiography) pre, 1 month, present and diastolic-systolic volumes of left ventricle.

Nº	Ejection fraction pre(%)	Ejection fraction I month	Ejection fraction present (%)	DDLV/SDLV pre	DDLV/SDLV post	DDLV/SDLV present
1	-	-	-			
2	44	47	43 (16° m PO)	61/44	60/49	60/47
3	43	32	45 (15° m PO)	68/49	60/46	60/46
4	35	35	39 (12° m PO)	60/42	58/40	57/39
5	28	42	28 (8° m PO)	104/90	98/86	95/89
6	32	28		88/80	72/59	
7	35	Death PO		42/26	-	
8	39	64	60 (8° m PO)	62/46	64/45	63/42
9	15	25	29 (6° m PO)	59/42	-	54/49
10	35	25	10 (6° m PO)	-	88/80	92/82
11	-	53		-	45/32	
12	20	28	28 (2° m PO)	63/50	60/48	71/64
13	19	31	24 (2° m PO)	73/63	68/58	80/70
14	15	Death		69/59	-	
15	23	28		80/73	74/68	
16	42	Late death	42 (2° m PO)	72/60	68/56	
17	36	36	36 (4° m PO)	65/56	-	67/56
18	28	23	45 (6° m PO)	80/69	74/68	60/46
19	30	34	34 (3° m PO)	88/77	-	78/65
20	13	15	20 (1° m PO)	64/54	65/55	65/55
21	32	31	28 (pós-alta)	65/55	62/55	62/53
22	15	20	20 (1° m PO)	63/52	61/42	61/42
23	19	19		70/59	64/55	
Med	28	32	33	70/57	67/55	68/56
DP	10	12	12	13/15	12/14	13/15

DDLV- diastolic diameter of left ventricle; SDLV- systolic diameter of left ventricle; PO- postoperative.

bed in this paper is different because it causes reduction in the longitudinal axis of the left ventricle bringing the top of the papillary muscles near the left atrioventricular annulus in an amount that is equivalent of the distance between the free margin of the mitral leaflets and the mitral annulus.

The physiopathology of the dilated cardiomyopathy gradually leads to a change in the geometry of the left ventricular cavity. Originally with an elliptical shape, it gradually becomes spherical¹⁸. Dilation of the mitral ring²¹ and impairment of the papillary muscles¹⁹ also occur, which cause segmentary alterations in ventricular contractility²⁰, determine noncoaptation of the valvular leaflets with subsequent valve escape. This is a factor obviously associated with clinical and hemodynamic worsening, as well as with a poor short-term prognosis²².

In this situation, the left ventricle begins to work with higher volume and pressure levels. Thus, tension in the wall increases in a ventricle that has already undergone chronic aggression. The outcome is a worsening of ventricular contraction. Batista et al.^{8,10} precisely propose reducing the diameter of the ventricle and consequently diminishing wall tension.

When we suggested implantation of a prosthesis in the mitral position, keeping all valve structures through displacement of the anterior leaflet together with preservation of the posterior leaflet²⁴, we aimed to stop the valve regurgitation and to remodel the ventricular cavity with the shortening of its longitudinal axis. We also maintained that the contracting function of the papillary muscles and the

wall tension is reduced without the need of ventricular resection. We believe that this technique adds a benefit to the performance of the left ventricle more than just simply correcting the mitral regurgitation through reconstruction.

This proposition to implant a prosthesis for the correction of mitral regurgitation, contrary to what was previously believed, causes improvement in cardiac output despite the same ejection fraction because it eliminates regurgitant flow with the totality of volumetric reduction used in ventricular systole.

Clinical and experimental studies have shown that preservation of the leaflets as well as of the subvalvular structures is beneficial to ventricular function²⁵, mainly in patients with severe heart failure. It is known that the mitral valve system is an integral part of the electromechanical mechanism of ventricular systole, although the mechanisms through which function of the mitral valve system changes and the geometry of the left ventricle is altered remain unknown.

By implanting a prosthesis, we were able maintain the entire valvular system with its leaflets, chordae tendinae and papillary muscles, as well as carry out reconstruction of the anterior leaflet, determine a remodeling of the left ventricle with enhanced ventricle performance, all of which resulted in clinical improvement, a fact observed in the cohort studied. The theoretical inconvenience of a prosthesis implantation is irrelevant if we consider that its life span will probably be greater than the patient's survival.

As limitation of our experience we admit is that the associated procedures carried out in 13 cases might influence

and perhaps overestimate the benefit of the procedure; however we point out, that none of these additional surgical procedures were the sole indication for surgery.

Despite the lack of records on long-term evolution, the surgical technique described in this paper is attractive, because the results obtained showed a survival rate of 86.9%

in the period under observation. We conclude that surgery was beneficial for 82.6% of the patients in the period of follow-up and resulted in a significant improvement in functional classes. Our data suggest that this technique may be a possible alternative for the treatment of severe cardiomyopathy with mitral regurgitation.

References

1. Evans RW, Orians CE, Ascher NL. The potential supply of donors. *JAMA* 1992; 267: 239-46.
2. Bassie BM, Parker M. Congestive heart failure current controversies in future projects. *Am J Cardiol* 1990; 66: 429-30.
3. Bourassa MG, Gurmé O, Brangdiwalasi N. Natural history and patterns of current practices in heart failure. *J Am Coll Cardiol* 1993; 22 S(A): 9-14.
4. Carpentier A, Chachques JC, Relland J, et al. Dynamic cardiomyoplasty at seven years. *J Thorac Cardiovasc Surg* 1993; 106: 42-54.
5. Patel HJ, Lankford EB, Polidore DD. Dynamic cardiomyoplasty: Its chronic and acute effects on the failing heart. *J Thorac Cardiovasc Surg* 1997; 114: 169-78.
6. El Oakley RM, Jarvis JC. Cardiomyoplasty- a critical review of experimental and clinical results. *Circulation* 1994; 90: 2085-90.
7. Moreira LF, Stolf NA, Braile DM, Jatene AD. Dynamic cardiomyoplasty in South America. *Ann Thorac Surg* 1996; 61: 408-12.
8. Batista RJV, Santos JLV, Takeshita N, Bocchino L, Lima PN, Cunha MA. Partial left ventriculectomy to improve left ventricular function in end-stage heart disease. *J Card Surg* 1996; 11: 967-75.
9. Mc Carthy PM, Starling RC, Wong J. Early results with partial left ventriculectomy. *J Thorac Cardiovasc Surg* 1997; 114: 755-65.
10. Batista RJV, Verde J, Nery P, et al. Partial left ventriculectomy to treat end-stage heart disease. *Ann Thorac Surg* 1997; 64: 634-8.
11. Replogle RL, Kaiser GC. Position paper: new technology assessment committee left ventricular reduction. *Ann Thorac Surg* 1997; 63: 909-10.
12. Bolling SF, Pagani FD, Bach DS. Intermediate outcome of mitral reconstruction in cardiomyopathy. *J Thorac Cardiovasc Surg* 1998; 115: 381-8.
13. Bach DS, Bolling SF. Early improvement in congestive heart failure after correction of secondary mitral regurgitation. *Am Heart J* 1995; 129: 1165-70.
14. Bolling SF, Deeb MG, Brunsting LA, Bach DS. Early outcome of mitral reconstruction in patients with end-stage cardiomyopathy. *J Thorac Cardiovasc Surg* 1995; 109: 676-83.
15. Dreyfus GD, Duboc D, Blasco A, et al. Myocardial viability assessment in ischemic cardiomyopathy: benefits of coronary revascularization. *Ann Thorac Surg* 1994; 57: 1402-8.
16. Blondheim DS, Jacobs LC, Kother MN. Dilated cardiomyopathy with mitral regurgitation: decreased survival despite a low frequency of ventricular thrombus. *Am Heart J* 1991; 122: 763.
17. Boltwood CM, Tei C, Wong M, Shah PM. Quantitative echocardiography of the mitral complex in dilated cardiomyopathy: The mechanism of functional mitral regurgitation. *Circulation* 1987; 76: 777-85.
18. Kono T, Sabbah HN, Rosman H, Alam M, Goldstein J. Left ventricular shape is the primary determinant of functional mitral regurgitation in heart failure. *J Am Coll Cardiol* 1992; 20: 1594-8.
19. Goldley RW, Wann S, Rogers EW, Feigenbaum H, Weyman AE. Incomplete mitral leaflet closure in patients with papillary muscle dysfunction. *Circulation* 1981; 63: 565-71.
20. Gorman JH, Gorman RC, Plappert T. Infarct Size and location determined development of mitral regurgitation in sheep model. *J Thorac Cardiovasc Surg* 1998; 115: 615-22.
21. Chandraratna PAN, Aronow WS. Mitral valve ring vs dilated ventricle. *Chest* 1987; 79: 2.
22. Romeo F, Pelliccia F, Cianfrocca C, Gallo M, Barilla F, Cristofani R. Determinants of end-stage idiopathic dilated cardiomyopathy: a multivariate analysis of 104 patients. *Clin Cardiol* 1989; 12: 387-92.
23. Hansen DE, Cahill PE, de Campli WM, Harrison D, Miller G. Valvular-ventricular interaction: the importance of the mitral apparatus in canine left ventricular systolic performance. *Circulation* 1986; 73: 1310-20.
24. Dickstein MA, Spotnitz HM, Rose EA, Burkhoff. Heart reduction surgery: an analysis of the impact on cardiac function. *J Thorac Cardiovasc Surg* 1997; 113: 1032-40.
25. Lillehei CW, Levy MJ, Bonnabeau RC. Mitral valve replacement with preservation of papillary muscles and chordae tendinae. *J Thorac Cardiovasc Surg* 1964; 47: 532-43.