

Partial left ventriculectomy: a retrospective study

Ventriculectomia parcial esquerda: uma análise retrospectiva

Marcelo Campos CHRISTO¹, Sérgio Figueiredo Campos CHRISTO², Liberato John Alphonse DI DIO³

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Abstract

Objective: To identify useful predictive data on chance mechanisms of postoperative outcome, the impact on symptoms of terminal heart failure — after partial left ventriculectomies (PLV) — was critically evaluated through the analysis of results, on accumulated descriptive data on reports, between 1995 and 1998.

Method: Available routine clinical data on surgical aspects and clinical outcomes were gathered and, when possible, validated for comparative analysis.

Results: PLV can provide a significant short to medium term amelioration in the quality-of-life in event-free survivors, but it was also watched out that in important proportion of them — as an evolutive sequence — PLV was incapable of changing the myocardial fibers leading tendency to conservatism of the preoperative vicious geometric dynamic pattern in late evolution. Importantly, the LV end-diastolic echocardiographic diameter of 7.5 cm (\pm 1.4 cm) was the steadiest quantitative significant numerical appeal to heart reduction surgery, in a setting of 465 patients, aged two to 74 years. And in a succession of individual reports of PLV results, whose mortality varied from 0 a 60%, survival after PLV showed a significant relationship with morphologic evolution

of cardiomyocytes, in postoperative, and augmented in absolute values in patients with progressive ventricular dysfunction, treated with the insertion of LVAD (Left Ventricular Assist Devices).

Conclusion: The material impossibility of identifying useful qualified predictors on chance mechanisms of postoperative outcome emerged as the crucial limitation for current usage of surgical reversal of left ventricular structural chamber dilation — to treat dilated cardiomyopathies — despite accumulated numerical values and clinical experiences.

Descriptors: Heart ventricle, surgery. Heart failure, surgery. Cardiomyopathy, congestive, surgery. Cardiac surgical procedures.

Resumo

Objetivo: Este estudo foi empreendido para identificar fatores que poderiam ter contribuído para o decréscimo da mortalidade e da morbidade em pacientes submetidos a ventriculectomia parcial esquerda (VPE) pela análise de dados descritores de resultados acumulados, a partir de diferentes publicações sequenciais, entre 1995 e 1998.

1 - Hospital Felício Rocho. Belo Horizonte, MG.

2 - Hospital Felício Rocho. Belo Horizonte, MG.

3 - Professor de Anatomia Cirúrgica, Universidade de Santo Amaro. São Paulo, SP.

Correspondence address:

Marcelo B. Campos Christo.

Rua Uberaba, 370, 10º andar.

CEP 30180-080.

Belo Horizonte, MG.

Método: Dados clínicos rotineiros, envolvendo aspectos cirúrgicos e resultados pós-operatórios, foram reunidos e, quando possível, selecionados para análise comparativa.

Resultados: A VPE foi capaz de possibilitar uma melhora muito significativa no desempenho da função sistólica e da qualidade de vida dos sobreviventes livres de eventos, mas também pode ser claramente verificado que, numa importante proporção destes sobreviventes — como uma seqüência evolutiva — a VPE foi incapaz de modificar a tendência das fibras miocárdicas de retornar ao modelo do pré-operatório, na evolução tardia. Além disso, o diâmetro diastólico final — ecocardiograficamente avaliado — de 7,5 cm ($\pm 1,4$ cm) foi o suporte quantitativo mais significativo para a indicação da VPE, em uma coorte de 465 pacientes de dois a 74 anos de idade. Em uma sucessão de resultados individuais nos quais a mortalidade pós-operatória oscilou de 0 a 60%, a

sobrevivência à VPE mostrou relação significativa com a evolução da morfologia da célula miocárdica no pós-operatório e aumentou exponencialmente, em termos absolutos, quando pacientes com aumento persistente da disfunção ventricular foram tratados com dispositivo de assistência ao ventrículo esquerdo. Left Ventricular Assist Device (LVAD).

Conclusão: A impossibilidade material de identificar preditores qualificados das chances de evolução pós-operatórias emergiu como uma limitação crucial para o uso rotineiro da reversão cirúrgica da dilatação da cavidade ventricular para tratar cardiomiopatias dilatadas, a despeito da experiência e dos valores numéricos acumulados.

Descritores: Ventrículo cardíaco, cirurgia. Insuficiência cardíaca, cirurgia. Miocardiopatia congestiva, cirurgia. Procedimentos cirúrgicos cardíacos.

INTRODUCTION

In dilated heart disease, the dilation of the left ventricular chamber is an early dominant characteristic, usually anticipating signs of heart failure. The increase of the myocardial mass, that is always present, is operatively incapable of compensating the progressive difficulty of shortening of the ventricular wall muscle fibers. The profile of structural dilation, evolving with the progressive increase of the cross-sectional diameter of the ventricular chamber, results in a predominantly ellipsoid shape of the left ventricle (LV), with individual variations originating from particular pathologic situations.

The “stroke” volume is maintained by a progressive increase in the ventricular chamber, in spite of the degeneration of the contractile capacity of the LV and of the reduction in the ejection fraction [1-6].

Independently of etiological factors, these morphologic and functional aberrations are adaptations characteristic of functional incompetence and the clinical result of the evolutionary course of a series of aggressions to the myocardium, involving myocardial muscle hypertrophy with segmental variations, in ventricular chamber walls in process of progressive dilation.

“Myocardial remodeling” has been the expression used to define these permanent and progressive alterations, involving muscular and non-muscular myocardial cells and evolving different periods of time for different cellular groups [1-10].

Clinical studies [7-9] and findings from autopsies have demonstrated that the quantitative relation of ventricular myocardial mass with the final diastolic volume of the LV has prognostic value and that lower survival can be

expected in patients with great dilations of the LV.

Surgical technique

Heart transplantation, “after hard-learned lessons” [11], became accepted as the best option for survival of patients with dilated heart disease and terminal heart failure. But the number of patients dying on waiting lists, awaiting hearts, started to grow exponentially, making the procedure an unattainable goal for the majority. Data from the “Single National Transplant Organ Network”, of the United States of America, show that an adult of the O blood group will wait, on average, 595 days to find an appropriate donor, and that half of these patients die before heart transplantation [12].

The scarcity of donors and the indecision of the society to invest in the treatment of terminal patients have been permanent challenges to the electiveness of heart transplantations as the preferred solution [13,14].

Cardiomyoplasty

Cardiomyoplasty, first used as a “myocardial reposition”, after traumatic injury [15] or the removal of malignancies [16], was proposed as an alternative to heart transplantation [17,18].

The procedure was well accepted and the results of important series were published [19,20]. But despite of aggregation of muscular mass by the electrically stimulated muscular voluminous patch LV, the consequences of the operation on the dynamics and morphology of the dilated ventricles were never well evidenced [21-24]. Post-operative evaluation of the LV function shows that the increase in ‘stroke’ volume and output were modest [20], not reflecting the active effect of the contraction of the

muscle patch [19,21]. Post-operative studies with the use of magnetic resonance [22] enabled the observation that the ventricular geometry continued unaltered and that the shape of the ventricular chamber is not modified with the contraction of the muscular patch.

On the other hand, the post-operative results make it very clear that there is an inverse relationship between the post-operative diameter of the ventricular chamber and the functional performance of the LV after cardiomyoplasty. Better results correspond to less dilated ventricles. Thus, cardiomyoplasty opened a new perspective for the surgical treatment of dilated heart disease: “the surgical reversal of the dilation of the ventricular chamber”.

Partial left ventriculectomy

Starting from the postulate that patients with dilated heart disease and terminal heart failure can have progressive contractile dysfunction of the myocardium, modified by a morphological rearrangement of the heart, partial left ventriculectomy (PLV) was imagined and performed. The operation reverting structural alterations, which corresponded to progressive functional disorders, corrects ventricular dysfunction, by allowing a better identified anatomic-quantitative relation with normality between post-load and contractile response of the LV.

One segment of viable muscle tissue is removed from the posterolateral wall of the LV, following the meridian axis of the chamber. Resection, starting at the point of the heart, is extended to the base of the papillary muscles (Figure 1).

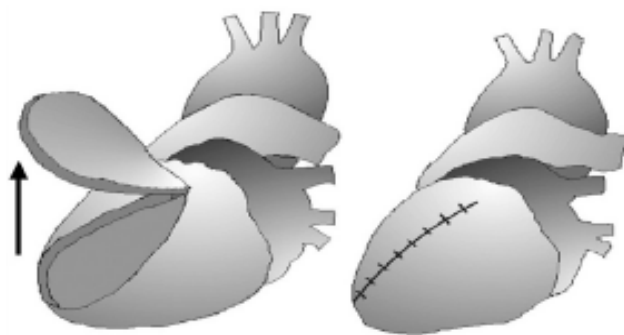


Fig. 1 – Partial left ventriculectomy

The operation results in the development of a concentric geometric shape of the LV, functioning as a procedure capable of removing an obstacle at ventricular systole, corresponding – morphologically and functionally – to a reversal of remodeling of the LV.

By altering the local distribution of ‘wall stress’, PLV corrects the most important geometric factor of ventricular

overload, reestablishing the balance of forces between the expansion and the functional restoration of the myocardial fibers. The theoretical expression of which is the mathematical equation of Laplace: $d = P \cdot R / 2Th$ (where d and P express ‘wall stress’ and inter-chamber pressure respectively and R and Th the radius of the chamber and the thickness of the wall).

The reducing ventriculectomy is followed by mitral valvuloplasty (or valve replacement) and by reconstruction of the ventricular wall using running sutures. The inventor of the technique, Randa Batista, refers to 103 patients operated on with a hospital mortality rate of 13%. Hospital mortality is mainly associated with the occurrences of hemorrhages and complications after infections. Whilst the late mortality was frequently associated with failure of the LV function or the occurrence of arrhythmias [25].

Patients

Between 1995 and 1998 sequential results, 465 PLVs were reported in 17 different works [25-41]. The total population of patients consisted of 72% of men and 28% of women, with ages between 2 and 74 years. The mean age was 50 ± 11 (Table 1).

Table 1. Partial left ventriculectomy: Clinical data analyzed among 465 operated patients

Partial left ventriculectomy	465 operated patients
Gender	Male = 72% Female = 28%
Age (years)	2 to 74 mean = 50 standard deviation = 11 95% of all the patients = 20 to 72
Functional class	Pre-operative = III-IV Post-operative = 97.0% ↗
Ejection fraction	Pre-operative = 0.18 Post-operative = 0.31
Quality of life	97% ↗
Deaths	Immediate = 25.0% (Hospital = 12.6%) Late = 36.1%
Associated procedures	Mitral = 74.0% Tricuspid = 10.5% Others = 58.3%
Selected patients	transplant waiting list = 50.0% Transplants contra-indicated = 50.0%
Etiology and indication (non-death proportional distribution)	idiopathic dilated heart disease, ischemic Chagasic, Valvar Alcoholic, Viral Peripartum, non-determined

Data collection

Demographic, hemodynamic and echocardiographic data, as well as other available data were collected for analysis and exhibited in tables and figures.

Clinical profile

The pre-operative clinical profile of the studied patients, in spite of the variety of sources of information, was basically the same: increase of the LV chamber, heart failure, functional class III and IV, all of the patients were maintained on 'maximum' medicinal treatment. The patients were referred for surgical treatment following different criteria. Starting from a global estimation: heart transplantation had been contraindicated in 50% of cases, including an important non-defined number of patients impeded from receiving transplants due to non-medical criteria. The other 50%, in a critical situation, were included on the "waiting list for heart transplant", and PLV was employed as a 'non-orthodox' alternative to improve the quality of life of these patients.

In spite of the imprecision of the obtained data, it seems without doubt that the etiological profile of the patients submitted to PLV did not differ from the etiologic profile of the patients submitted to heart transplantation [42,43].

The majority of these cases were suffering from non-ischemic myocardial disease but ischemic disease seems to have been the second most important cause of heart disease involved in this study, followed by a hard to define number of myocardial diseases, attributed to different causes, including Chagas' Disease.

Numerical and quantitative mechanical indices of LV

In the different experiments reported, each patient was submitted to evaluation by processing of images and numeric data, according to the adopted routines and supervening options. Data from bidimensional echocardiography were the most consistent and universally adopted to measure the ventricular function. From the point of view of measured numeric data, the dimension, around 70 mm (75 ± 14) final systolic diameter of the LV (FSDLV) was the pre-operative echocardiographic data most used for indication for PLV in the cohort of patients operated on between 2 and 74 years old (Figure 2). This parametric value, also recognized in large series of hearts in cadavers [6], seems to be a powerful predictor of the irreversibility of dilation, the loss of contractility and death.

The mass:volume ratio of the LV has been cited as a parameter for the indication of PLV in the works by Batista, but without numerical translation. Our patients were operated on with a mass:volume ratio of less than 0.9 (and a FSDLV greater than 70 mm) [35].

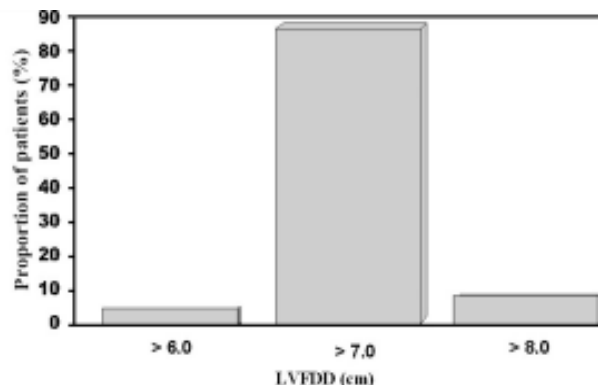


Fig. 2 - Accumulated distribution of FSDLV among 465 patients submitted to partial left ventriculectomy

The range of 'viable' myocardial mass (30-290 grams) removed in the experiments of Cleveland [31,32], or as was admitted as a parametric criterion in different works, seems to make the necessity of adequate planning for each case imperative. Post-operative failures of the LV, attributed to "insufficient reduction of ventricular mass" or to "redilation", remain as a serious clinical problem, apparently leading to depression of the systolic properties of the LV. Reoperations "to optimize the reduction in the ventricular mass" [25] have been reported, but criteria to prevent or correct these inconveniences have never been well defined.

Theoretical approaches demonstrated that the surgical reduction of myocardial mass is followed by "losses" in the diastolic properties of the LV [44-46]. Although "diastolic deaths" [47,48] have been exceptionally highlighted in the set of clinical evolutions of PLV, post-operative deaths, characterized by acute and serious elevations of "pressure and filling", have been incidentally commented on and attributed to a "super-reduction" [25] or to preponderance of intracardiac fibrosis [40]. An increased post-load of the left atrium, due to diastolic restriction in the filling of the LV, can result in a significant "retrograde flow" to the pulmonary veins. And the paradox of the "diastolic rigid heart" seems to represent a real clinical possibility after PLV [47].

Regional myocardial abnormalities

The surgical approach proposed by BATISTA [25] seems to correct the most frequent and recognized geometric obstacle of contractile dynamics of the LV in dilated heart disease very well: the functional incompetence of the apical myocardial segments [49].

Very little is known in respect to "regional differences" in the mobility or thickness of the ventricular wall, in apparently uniform diffuse and hypokinetic dilations of dilated heart diseases [6]. Among other publications, the same quantitative parameters for ventricular reduction were

indistinctly applied to all ventricular dilations [25] and the same “surgical reconstruction” model has been used for left ventricular reconstruction with different patterns of chamber dilation [6]. The predominance of diffuse or segmental anomalies in the distribution of the mass or the tolerance of the LV were never taken into account [25-41].

Ischemic disease and Chagas’ Disease, critical determinants of anomalies of contractility and of the load distribution on the LV, characterize peculiar geometric patterns and represent, with all probability, the least apt population to benefit from PLV [25-41].

On the other hand, the association of PLV with “aortic-coronary bypasses” was very frequently referred to, but fortuitous “restructuring approaches” consistent with myocardial revascularization, were never clearly explicated as alternatives to the ‘orthodox’ model of PLV.

In spite of the scarcity of specific information [25-41], it seems to be consensual that the worst clinical results can be expected in patients with dilations characterized by segmental anomalies of the LV wall.

Post-operative fatalities

It was impossible to obtain a very clear definition of the post-operative fatalities of PLV.

Estimates based on reported data point to a surgical and peri-surgical mortality rate of around 25% (Tables 1, 2 and 3), in a series of individual results whose mortality rates range from 0 to 60% [25-41].

In an important series [30,32,37,41], the one-year post-operative survival rate varied from 33 to 40%, to 57%, to 87% and it was perfectly clear that the use in the post-operative period, of “assist devices” (LVEAD) was a decisive factor in the reduction of mortality [32].

Table 2. Causes of post-operative deaths in 465 patients submitted to PLV

Cause	Death		Hospital	
	Nº	%	Incidence ¹	[IC 95%]
Hemorrhage	28	24%	6%	[3.8; 8.2]
Heart failure	30	26%	6%	[3.8; 8.2]
Arrhythmia	33	28%	7%	[4.7; 9.3]
Others	26	22%	6%	[3.8; 8.2]
Total	117	100%	25%	[21.1; 28.9]

¹ Incidence = number of deaths / 465 x 100;

² 95%: confidence interval for death;

³ A non-specific number of deaths was avoided by mechanical support of LV or heart transplantation;

⁴ A non-specific number of deaths was avoided by automatic implantable defibrillation.

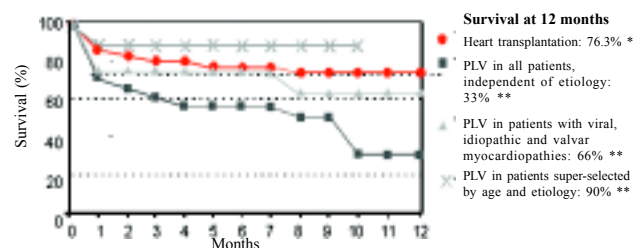
Table 3. Post-operative mortality (selected series)

Author	Nº of operated patients	Hospital	Late
Batista (1997)	120	22,0%	55,0%
McCarthy (1997)	53	2,0%	5,7%
Starling (1997)	17	0%	0%
Moreira (1998)	37	18,9%	43%

This agrees with our own estimates for a post-operative period from 30 days to two years, 36% (n = 125) of deaths were reported and the inexorable decline of the ventricular dysfunction was the most frequent cause of these deaths.

A critical analysis of death arising after PLV can be made by comparing it with death arising of a cohort of 186 patients, originally submitted to heart transplantation [11]. Both cohorts did not suffer losses in the immediate follow up. Whilst the survival at one year after transplantation was 76%, survival of patients of PLV was 33%, of all the patients operated on. When the PLV patients were selected by clinical criteria to only include patients with viral, valvular or idiopathic dilated heart disease of short-term evolution and excluding elderly patients, those with failure of other organs or patients operated on under emergency situations, the one-year survival rate after PLV was 60%. Finally a survival rate of 90% at one year was observed in a group of 10 super-selected PLV patients (Figure 3). The criteria involved in the selection of this special group of patients were not clearly defined, but careful consideration of “surgical experience” was stressed as a decisive factor [37].

Evidence of the post-operative functional decline has been better related with the long-term evolution than with the age of the patient and poor results can be expected in the presence of ischemia or interstitial fibrosis.



*Griffith BP Ann Surg 1986. [11]

**Batista JVR Ann Thorac Surg 1997. [37]:

Fig. 3 – Actuarial survival rate. Comparative analysis of the estimate of survival for heart transplant patients and patients submitted to partial left ventriculectomy

It is possible to speculate that a longer survival can be predicted for younger patients who recently entered in functional class IV, and those in which the myocardial cells may regress to their hypertrophy [41,50,51]. Anyway, there seems to be no doubt that an excellent immediate result is not a prediction of an excellent long-term result.

Associated surgical procedures

Although not always specifically referred to in the different publications, the correction of mitral insufficiency has been routine incidental to PLV. However, it has been frequently recognized that mitral valvuloplasty in isolation has not been able to persistently correct the performance of the mitral valve after PLV. On the other hand, the possible advantages of maintaining the “remodeled” mitral apparatus in a ventricular chamber that underwent a drastic reduction in its volume has been the subject of many debates. It seems to be very predictable that, after PLV, in contrast to the favorable effects of the reduction of ventricular mass on the systolic dynamics, the “hyperbolic” papillary muscles start to act as mechanical obstacles to the LV functional performance, reducing systolic output and favoring the local occurrence of infarctions.

To reduce these disadvantages, replacement of the mitral valve with removal of the papillary muscles has been defended by some [25,37] and, as an alternative, to preserve the mitral apparatus, with “transference” of the papillary muscles [32] and varying plastic procedures: orthodox and non-orthodox [32,37,40] have been proposed by others. No clinical evidence that enabled a clear choice has been found, whether for systematic valve replacement, or conservation of the mitral apparatus. The occurrence of some degree of mitral regurgitation in the post-operative period seems to have been a common event [25-41], not correlated with the degree of mitral insufficiency, nor with the pre-operative myocardial competence [52]. However, the post-operative progression of mitral insufficiency seems to have been an important marker of death [25-41].

Aspects of the association of PLV with plastic procedures of the tricuspid valve will be considered at another opportunity.

Although MR has been correctly referred to as an elective procedure associated to PLV on ventricular dilations, coexisting with ischemic myocardial disease, it was impossible to determine the real influence of this association in the results of PLV.

Other surgical procedures were circumstantially associated with PLV, but for sure, pacemakers, defibrillators, mechanical devices of ventricular assistance and heart transplantations were the associated procedures that changed the natural history of the evolution of many patients who had survived PLV [31-33].

Quality of life – functional class – ejection fraction – myocardial contractility

With an analysis of the clinical data (Table 1 and Figure 4) it is possible to conclude that the most significant and homogeneous impact of reduction surgery in event-free survivors was translated as an improvement in the “quality of life”. This was expressed according to the perspective of the patient and assessed by the increase in the “quality of life score”- physical performance, performance at work, psychological condition, sleep pattern, eating habits, perception of symptoms, quality of social interaction, feelings and expectations related to treatment [53]. These findings seem well consistent with the notion that in heart failure, morbidity progresses in parallel with the dilation of the LV chamber [54] and that the improvement in the quality of life might also be an indication for a change in the functional class [25-41]. However, after PLV, neither “re-qualification”, that is impossible to numerical express, which predominates in the evaluation of “quality of life”, nor increase in ejection indices that can, numerically, characterize improvement in functional performance, seem to be reliable predictors of a post-operative evolution with sustained functional improvements free of fatal arrhythmias.

The distribution of the numeric values of the reported results (Figure 4) clearly show that the extreme lack of uniformity of the indicator values of gains in the ejection fraction in the post-operative period, coincide with an extreme convergence in the gain of “quality of life”.

Assessment of the post-operative results of PLV have been wide-ranging, incorporating objectively obtained with post-operative values and subjectively perceived in clinical impressions with a large range of proportional distributions [25-41].

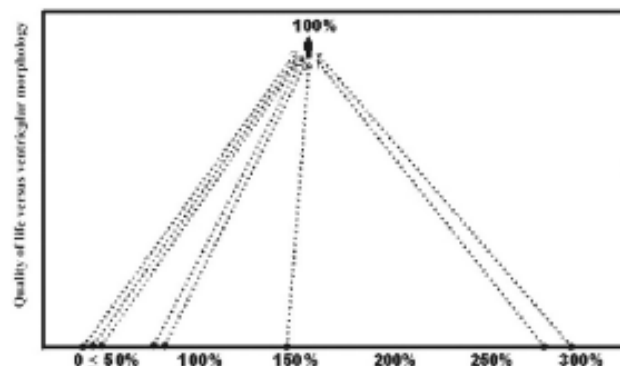


Figure 4 – Comparative analysis of the post-operative improvement in the physical, emotional and social well being and the quantitative estimate of the ejection fraction improvement after partial left ventriculectomy

Relevant data have been obtained by routine monitoring with “conductance” catheters in some post-operative evolution pilot studies [28,29,40,41]. This instrument, generating a continuous signal, is very sensitive to cavity volume variations and independent of possible distortions of the ventricular geometry, enabling immediate information about the systolic elastance and diastolic competence of the left ventricle “newborn babies” [45,55].

However, in routine clinical practice, no other evaluation, other than relief of symptoms and of ejection fraction alterations, both obtained by “non-invasive” procedures, seem to have had objective usefulness to measure therapeutic response of PLV, making the distinction between “ventricular chamber filling difficulties and myocardial function depression” problematic [25-41]. This seems to indicate that, to correctly evaluate the contractile function of the LV, an alternative for “load-dependant” indices – enabling an evaluation of myocardial contractility without influence of the “load” is potentially utilizable as a non-invasive clinical method – is a necessity [56].

In the late post-operative evolution, ejection fraction variations seem acceptable as markers of post-operative survival. A continuous decline of the ejection fraction – related with the time of evolution – seems well identified with the post-operative progression of initial myocardial disease and with deterioration in the functional performance of the LV.

Geometric preservation – Remodeling and apoptosis

The attraction for a single geometric model seems to be a dominant impulse in the evolutionary course of remodeling of the LV in myocardial failure [4,6].

Correcting the unorganized inter-relationship between ventricular chamber volume, wall thickness and myocardial mass, PLV surpasses a ventricular geometry model strongly related to the abnormal functional performance. And, a very significant functional performance improvement of the ventricular chamber has been widely recognized as an immediate result, in spite of the abnormality of the myocardial cells.

On the other hand, there is growing clinical and experimental evidence, that the abnormal hemodynamic overload triggers irreversible programs of cell death by apoptosis, in different cells of the myocardial divisions of the heart. Very little has been learnt about the progress of interruption of the events that signal and precipitate the triggering of such mechanisms of the cellular cycle [57-59]. Loss of contractile units, disorders of the conduction and lethal arrhythmias may represent different clinical translations of the evolutionary course of these programs,

apparently never influenced by PLV.

The clinical information available enables us to state that, in a significant number of patients, PLV has been unable to eliminate the “sensitive dependence” of the myocardial fibers of the pre-operative geometric-dynamic patterns. Despite the absence of definitive predictors, the return to the pre-operative morphology seems to be a real possibility of the late post-operative evolution [35,60,61]. On the other hand, by means of serial endomyocardial biopsies, a significant coincidence between the myocardial cellular morphology and prognosis of PLV was clearly demonstrated, independent of etiological factors [41,50,51].

Pulmonary hypertension – tricuspid regurgitation

There are few specific references about the influence of pulmonary hypertension in surgical results [30,36,37], but it has been clearly admitted that pulmonary hypertension is not a risk limitation in PLV. At least, differently to heart transplantation [42], a high mean pressure of the pulmonary artery has not been a criterion in the indication for PLV [30,36,37,41].

Apparently, it has been speculated that the drastic reduction in the final diastolic volume of the LV, combined with a “hyper-dimensioned” right ventricle after PLV, would result in a relative increase of the pulmonary vascular bed [62,63]. This would diminish the “operative opposition of the interventricular septum” to output of the LV, at systole [64].

Anyway, it was impossible to identify any clear relationship between the pre-operative elevation of pulmonary arterial pressure and post-operative mortality or morbidity, using the available clinical data [25-41].

On the other hand, there seems to be no doubt that the sustained increase of the systolic pressure in the pulmonary artery in the post-operative period, has been a powerful predictor of myocardial failure and death [52].

Tricuspid regurgitation after PLV started to be correctly assessed after the introduction of the echocardiogram in the per-operative routine of PLV, introducing a powerful instrumental evaluation for the optimization of its results [32].

In which circumstances tricuspid incompetence might be solved in the immediate post-operative period using medication or might become progressively more severe, transforming it in an obstacle to the systolic output of the LV, precipitating heart failure, is a question that remains without an answer.

In the same line, the advantages of making correction of tricuspid regurgitation a standard practice associated with PLV, suggested by some authors seems to be controversial [30].

Arrhythmias and sudden death

A series of communications about initial and late evolution of PLV have clearly demonstrated that “sudden death” always remains as an important threat to the post-operative evolution, in spite of indisputable functional gains (Figure 4). It has generally been admitted that the transmural scarring of LV, acting as arrhythmogenic dysplasia, may be associated with the post-operative deaths induced by tachyarrhythmia. “Sudden death” may be the result of an excellent post-operative clinical evolution after a complete clinical evaluation, including outpatient electrocardiographic monitoring [25-41].

The existence or not of “appearance of arrhythmogenic foci”, similar to “alterations in the cellular cycle” involving the conduction system [57,58], never deserved special attention. The possible role of arrhythmogenic events, induced by tensional modifications of the pulmonary vascular bed [59], was never taken into account. To associate PLV with automatic implantable cardioverter defibrillators (AICD) seems a justifiable proposal to reduce the high risk of spontaneous ventricular tachyarrhythmia in patients, in which a favorable surgical result can be predicted according to the hemodynamic parameters of the immediate post-operative period [65-67].

Possibilities and limitations

In spite of the limited evidence, this retrospective analysis enabled the elaboration of some postulated doctrines and the emergence of some aspects of the selection of patients and the post-operative manipulation that influenced the short- and long-term results.

1. Acting as an inhibiting mechanism of the geometric model of heart failure in dilated heart disease, PLV reestablishes the operational performance of the LV, reconfiguring the efficiency and the quantitative anatomy of the ventricular myocardium, defining a new strategy to modify the natural history of terminal heart failure.

2. The best results of PLV seem to be associated with dilated heart disease whose dominant clinical shape is the early expansion of the ventricular chamber, characterized by segmental thinning of the LV wall typical of Chagasic heart disease and ischemic disease. On the other hand independently of diverse etiologies, a limited association between the size of the myocardial cell and the results of PLV has been emphasized – the absence or regression of myocardial cellular hypertrophy is consistently associated with functional performance and to increased post-operative survival. This finding is, with all probability, the only independent predictor – scientifically proved – of “myocardial risk” of PLV, potentially more advantageous in patients without irreversible compromise of the myocardial cell.

3. The clinical syndrome of post-operative heart failure, with a “diastolic restriction” pattern of the LV, has been indicated as a bad result of PLV. “Diastolic deaths”, either attributed to the inability of LV relaxation, or to “super-reduction” of the ventricular mass, have been occasionally reported.

4. The association of aortic-coronary “bypasses” with PLV has been described, but “alternative reconfigurations” provoked by revascularization have never been clearly referred to.

5. In contrast to clinical and echocardiographic data that prove and give support to the importance of routine correction of mitral insufficiency in the post-operative period, the evaluation of tricuspid insufficiency seems to be seriously limited in its clinical value to command a per-operative correction routine of the tricuspid valve.

6. In spite of the elevated mortality and of the recognition that the long-term results of PLV are unpredictable, a clinical benefit is the short-term expectation of the operation. From an ethical point of view, PLV has been seen as a promising and acceptable therapeutic proposition, in the same proportions in which other non-orthodox therapeutic procedures have been ethically accepted to treat patients in advanced stages of dilated heart disease.

7. In the available clinical material, the volume of information involving patients who die after the operation seems to be much more decisive than the information about the clinical situation of those who survive.

8. Apparently, above the immediate functional results of PLV, structural alterations of the myocardium, advancing as non-controllable processes, continue following a series of courses effecting all the cellular cycle of the different myocardial cells.

9. A retrospective analysis of the post-operative evolution of patients who had been submitted to PLV revealed that, in a significant proportion of the cases, the reductive surgery was unable to overcome the dependency of the myocardial fiber to return to the intense model of the pre-operative period. This seems to function as an “intrinsic attracting model”, indifferently of the initial results and etiologic factors.

10. Although the progressive increase of the systolic pressure, in the post-operative period of PLV, has been highlighted as a powerful marker of irreversible heart failure, PLV seems to have been a totally acceptable alternative surgery when heart transplantation was contraindicated for pulmonary hypertension.

11. PLV cannot be accepted as a routine alternative for heart transplantations. But can be accepted as a matchless surgical intervention – ordinarily accessible and with reasonable chance of success over the short term – to

treat as outpatients, patients formerly confined to hospital with expectations of an evolution of heart transplant programs reducing the deaths and waiting lists.

CONCLUSIONS

1. Reversal surgery of structural dilation of LV by PLV is clearly correlated with improvement of the parameters of the functional performance of the heart, in a very significant proportion of surviving event-free patients.

2. The improvement in the quality of life, including physical and functional performance and emotional and social sensitivity, has been recognized as a more real expression of the results of PLV.

3. Of the reported quantitative numerical values, the final diastolic diameter of 75 mm (± 14 mm) was the best-correlated parametric datum with the indication of PLV, in a cohort of 465 patients, with ages ranging from 2 to 74 years.

4. The material impossibility to identify qualified predictors of the immediate post-operative evolution and long-term results emerges as a crucial limitation for the routine use of surgical reversal of structural dilation of the LV, as an elective treatment of dilated heart disease.

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