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Reshaping the remodelled left ventricle: a new concept

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Abstract

Objective: Based on the law of Laplace, transventricular tension members were designed to diminish wall stress by changing the left ventricle (LV) globular shape to a bilobular one, thus reducing the ventricular wall radius of curvature. This concept was tested in a model of congestive heart failure. **Methods**: Seven calves were used for the study (74.3 ± 4.2 kg). Treatment efficacy was assessed with sonomicrometric wall motion analysis coupled with intraventricular pressure measurement. Preload increase was applied stepwise with tension members in released and tightened position. **Results**: Tightening of the tension members improved systolic function for CVP > 10 mmHg (dP/dt: 828 ± 122 vs. 895 ± 112 mmHg/s, P = 0.019, for baseline and 20% stress level reduction respectively; wall thickening: 11.6 ± 1.5 vs. 13.3 ± 1.7%, P < 0.001) and diastolic function (LV end-diastolic pressure: 15.9 ± 4.8 vs. 13.6 ± 2.7 mmHg, P < 0.001, for CVP > 10 mmHg; peak rate of wall thinning: -12.2 ± 2.2 vs. -14 ± 2.3 cm²/s, P < 0.001 and logistic time constant of isovolumic relaxation: 48.4 ± 10.9 vs. 39.8 ± 9.6 ms, P < 0.001, for CVP > 5 mmHg). **Conclusions**: This less aggressive LV reduction method significantly improves contractility and relaxation parameters in this model of congestive heart failure. © 2001 Elsevier Science B.V. All rights reserved.

Keywords: Heart failure; Congestive; Remodelling; Volume reduction

1. Introduction

Congestive heart failure is a major and growing public health problem. Currently it affects 1% of people under 55 years and 9% of those over 80 [1]. In this chronic progressive disease, the central element involves remodelling of the cardiac chamber with ventricular dilatation. The degree of left ventricular dilatation, as well as its more spherical shape have been both shown to be independent risk factors of poor outcome [2,3].

Heart transplantation is the current treatment of choice for selected patients with end-stage heart failure. Unfortunately, the shortage of donor hearts and the complications of immunosuppression and rejection limit its benefits to a minority of patients with advanced heart failure [4]. The number and proportion of potential recipients who die awaiting the procedure are increasing every year. This situation has promoted ongoing efforts to develop other alternatives. These treatments are designed to arrest or substantially reverse the remodelling by assisting or resting the myocardium. Mechanical ventricular assist devices and cardiomyoplasty are intended to limit cardiac dilatation and ultimately to reverse the remodelling. More recently, partial left ventriculectomy, involving the removal of a portion of the lateral wall, has been introduced. Based on the law of Laplace (wall tension = (intraventricular pressure × left ventricular radius)/(2 × wall thickness)), it directly addresses the remodelling process.

However, today, there are no procedures available to consistently treat a broad based patient population for chronic heart failure at reasonable cost. A new concept of reshaping the left ventricle (LV) with transventricular splints, providing a smaller radius of curvature has been developed to address this significant need. The effects of this system on myocardial contraction and relaxation properties are tested here in a model of progressive volume loading.

2. Material and methods

The protocols described herein were reviewed and approved by the Committee on Animal Care, Office Vétérinaire Cantonal, Lausanne. All animals received care in compliance with the 'Principles of Laboratory Animal Care' formulated by the National Society for Medical Research and the 'Guide for the Care and Use of Laboratory Animals' prepared by the National Academy of Sciences

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2.1. Animals preparation

This study was conducted in seven calves, with a mean bodyweight of 74.3 ± 4.2 kg (standard deviation). The animals were premedicated with xylazine (0.15 mg/kg, given intramuscularly). General anesthesia was started with thiopentone sodium (10 mg/kg, given intravenously) and maintained thereafter with volatile anaesthetic (N2O and Halothane) mixed with oxygen-enriched air. The animals were equipped with a jugular central venous catheter and a femoral arterial catheter for hemodynamic monitoring. Respiratory rate and stroke volume were adjusted to maintain arterial blood gases within the normal physiologic range. Three ECG leads were installed. An arterial line was inserted into the right femoral arterial catheter for hemodynamical monitoring. A venous catheter was inserted through the right jugular vein into the right atrium to measure filling pressures. A median sternotomy was performed. The pericardium was opened and reflected to form a cradle for suspending the heart. A 8F high-fidelity micromanometer tip catheter (Millar Instruments Inc, Houston, TX, USA) and an intravascular ultrasound probe (IVUS, Boston Scientific Corporation, Sunnyvale, CA, USA) were inserted within the left ventricle (LV) through the apex of the heart. In order to measure wall thickness, a pair of ultrasonic crystals (Sonometrics, London, Canada) was placed on the antero-lateral wall of the LV at a point anticipated to be at equidistance between the Myosplints extremities. Heparin (Liquemin, 300 U/kg body weight, F. Hoffmann-La Roche & Co., Basle, Switzerland), was given systemically and the activated clotting time (ACT, Hemochron, International Technidyne corp., Edison N.J.) was kept above 400s throughout the experiment. A 24 French venous cannula was introduced into the left jugular vein for volume loading.

2.2. The device

The MyocorTM Myosplint TM system (Myocor Inc., Maple Grove, MN, USA) is a passive implantable device. The Myosplint consists of two epicardial pads and a transventricular tension member. The two pads are located on the surface of the heart with the load bearing tension member passing through the ventricle, connecting the pads and drawing the ventricular walls toward one another. Three Myosplints are placed to create the appropriate shape change along the length of the LV. Implantation of the Myosplint is performed as part of an open chest cardiac procedure on the beating heart.

The implant procedure includes three distinct phases. They are comprised of (i) identification and marking, (ii) tension member placement and (iii) tension member tightening. The Myosplints are equally spaced along a line from the apex to the base, perpendicular to the LV long axis (Fig. 1). This orientation promotes bissection of the LV while avoiding both the external and the internal critical structures of the heart. During the identification and marking phase, the coronary vasculature is visually identified on the exterior of the heart. Echocardiography is used to observe the inside of the chamber. To provide a more accurate view, a probe has been designed to slightly indent the surface. This indentation is used to correlate the internal anatomy and external position of the pads. In particular, care should be taken to avoid the mitral valve apparatus. All three device locations are identified and marked prior to tension member placement.

Placement of the tension member is facilitated via a 'Cdevice' stabilizer. The C-Device is placed on the heart and serves as a guide to connect the two marked locations on the opposite sides of the heart. Once the 'C-device' has been placed, a needle and a stylet are inserted through a guide tube on the 'C-device' and the ventricular chamber at the points identified on the surface of the heart. The stylet is then removed from the 'C-device' and the needle is fully advanced through the ventricular chamber. The 'C-device' is then removed from the chest cavity. The needle creates a passage through the LV chamber connecting the two marked points for placement of the tension member. The tension member is then inserted through the needle tube. The needle tube is then removed and the tension member is drawn through the chamber until the fixed pad comes in contact with the outside of the ventricular wall. This process is repeated for placement of the other tension members.

The adjustable pads are then threaded onto the leaders of the tension members. A specially designed 'Measurement and Tightening Device' is then loaded onto the tension member and advanced until snug to the outside of the heart. Based on the epicardial distance, the device applies a simple ratio calculation to determine a defined percentage stress reduction setting. The tension member is then tightened with the device to the appropriate calculated level. After tightening all three tension members, the pads are locked in place, and the excess portion of the tension members are cut using a cautery. The ventricle will thus have a bilobular appearance in the short axis (Fig. 1), once the Myosplints have been delivered, tightened and locked in place.

2.3. Study protocol

After placement of the tension Myosplints under ultrasound control, a period of 30 min was allowed for stabilization. Baseline hemodynamic data as well as contractility and relaxation parameters were recorded. Two litres of crystalloid solution (NaCl 104 mmol/l, KCl 5.4 mmol/l, CaCl₂ 1.6 mmol/l, MgCl₂ 1 mmol/l, Na Lactate 27 mmol/l, Na Bicarbonate 50 mmol/l) were infused over 5 min through the left jugular cannula. After a stabilisation period of 5 min, the haemodynamic, contractility and relaxation parameters were recorded. Then, the tension members were tightened to a 10% stress reduction level. After a stabilisation period



Fig. 1. Drawing of the dilated left ventricle before (right) and after (left) the Myosplint insertion and tightening. (A) LV cavity radius before the Myosplint insertion; (B) LV cavity radius with the Myosplint.

of 5 min, the haemodynamic, contractility and relaxation parameters were recorded. Then, the tension members were tightened to a 20% stress reduction level. After a stabilisation period of 5 min, the haemodynamic, contractility and relaxation parameters were recorded again. The overall duration of the procedure was 30 min, including the Myosplints manipulation and the data recording. This procedure was repeated stepwise until a CVP above 20 mmHg was reached.

2.4. Data analysis

Data were recorded digitally on-line with a sampling rate of 250 Hz/channel (Transceiver unit TRx 001, Sonometrics, London, Canada). Mathematical analysis of the data was performed off-line with a software package for cardiovascular analysis (Sonosoft version 3.1.3., Sonometrics, London, Canada). Three beats were averaged for the calculation of systolic and diastolic function indices. End-diastole was defined as the time corresponding to 5% of maximal dP/dt, and end-systole as the point of maximal systolic excursion of the wall thickness, occurring at or within 20 ms before peak negative dP/dt. The haemodynamic data included heart rate (HR), central venous pressure (CVP), left ventricular end diastolic pressure (LVDEP) and mean arterial pressure (MAP). The contractility parameters included the first derivative of the LV pressure (dP/dt), and wall thickening. Diastolic function was evaluated with the peak rate of wall thinning and the logistic time constant of isolovolumic relaxation (Tau).

2.5. Calculations

Peak rate of wall thinning was calculated as -dh/dt/dh [5], where *h* is the difference between maximal and minimal wall thickness, and *t* is the time.

The logistic model for the isovolumic relaxation pressure was given by:

$$LV P(t) = (P_A/1 + e\frac{t}{T_L}) + P_B$$

where LV P(*t*) is the isovolumic relaxation pressure of the left ventricle, P_B is a non-zero asymptote, P_A is an amplitude constant, *t* is time, and T_L is the time constant of the exponent (Tau) [6].

2.6. Statistical analysis

Mean and standard deviation were derived for each parameter analyzed. For the statistical analysis, each hemodynamic, contractility and relaxation variable was classified according to CVP values, which were categorized into intervals of 5 mmHg. For simplification of the results, interval 1

Table 1 Contractility indices categorized according to CVP intervals of 5 mmHg^a

CVP (mmHg)		1–5			5.1–10			10.1–15			15.1–20			> 20		
		Base	10%	20%	Base	10%	20%	Base	10%	20%	Base	10%	20%	Base	10%	20%
dP/dt	Mean	1059	1077	1108	976	984	1031	916	931	966	799	827	894	713	735	782
(mmHg/s)	SD	146	219	142	102	109	140	110	80	77	78	110	100	60	65	79
	Ρ		0.447			0.163			0.036			0.029			0.011	
Wall thickening	Mean	13.5	13.6	13.7	14.1	14.1	14.3	12.6	13.3	14.3	11.5	12.5	13.2	9.9	10.9	11.9
(%)	SD	0.4	1.7	1.5	0.8	1.3	1.2	1.1	1.2	1.5	1.1	1.7	1.6	1.3	1.2	0.8
	Р		0.868			0.463			< 0.001	l			< 0.001		< 0.001	

^a Base, baseline value; 10%, value when the Myosplints are tightened to a 10% stress reduction level; 20%, value when the Myosplints are tightened to a 20% stress reduction level; *P*, *P* value of ANOVA for each interval.

corresponds to 1–5 mmHg, interval 2 to 5.1–10 mmHg, interval 3 to 10.1–15 mmHg, interval 4 to 15.1–20 mmHg, and interval 5 to >20 mmHg. Analysis of variance for repeated measures were used for determination of statistical significance. Student *t* test was used for comparison of baseline values between different intervals. Statistical significance was reached for *P*values <0.05.

3. Results

All the Myosplints could be implanted without problem in all the animals. There were no persistent hemodynamic or rhythmic instability. No damage to the epicardial vessels or the mitral valve apparatus occurred. The procedure could be performed up to the targeted CVP in all the animals. The mean duration of the experiment, from the first baseline measurements, was 5 h \pm 24 min, range 4 h 30 min to 5 h 30 min. The mean volume of infusion was 20 \pm 1.6 l, range 18–22 l.

During the experiment, baseline MAP did not vary significantly with a values of 93 ± 15 mmHg at the beginning and 86 ± 10 mmHg at the end of the protocol. Moreover, when the Myosplints were tightened, MAP did not vary significantly at any time interval. The same holds true for HR, with

values of 89 ± 12 beats/min at the beginning and 82 ± 7 beats/min at the end of the protocol.

Baseline dP/dt values (Table 1) differed significantly between the 1st and the 2nd interval (P = 0.025), between the 3rd and the 4th interval (P = 0.007), and between the 4th and the 5th interval (P = 0.016). When the Myosplints were tightened, dP/dt increased significantly at the 3rd, 4th and 5th interval (Table 1).

Baseline wall thickening values (Table 1) differed significantly between the 2nd and the 3rd interval (P = 0.001), between the 3rd and the 4th interval (P = 0.002), and between the 4th and the 5th interval (P < 0.001). When the Myosplints were tightened, wall thickening increased significantly at the 2nd, 3rd, 4th and 5th interval (Table 1).

Baseline LVDEP values (Table 2) differed significantly between the 1st and 2nd interval (P = 0.003), between the 2nd and the 3rd interval (P = 0.031), between the 3rd and the 4th interval (P = 0.042), and between the 4th and the 5th interval (P < 0.001). When the Myosplints were tightened, LVDEP decreased significantly at the 3rd, 4th and 5th interval (Table 2).

Baseline peak rate of wall thinning values (Table 2) differed significantly between the 1st and the 2nd interval (P = 0.016), between the 2nd and the 3rd interval (P = 0.045), between the 3rd and the 4th interval

Table 2

Relaxation indices	categorized	according to	CVP	intervals	of 5	mmHgʻ
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CVP (mmHg)		1–5			5.1–10			10.1–15			15.1–20			> 20		
		Base	10%	20%	Base	10%	20%	Base	10%	20%	Base	10%	20%	Base	10%	20%
LVDEP	Mean	10.5	10.5	10.3	11.9	12.1	12	14.1	12.7	11.7	16.1	15	14	19.7	17.9	15.9
(mmHg)	SD	2	1.8	1.9	2.2	2.1	2.2	6.8	2.5	2.5	2.2	1.91	2	2.7	2.4	2.8
	Р		0.67			0.344			0.006			< 0.001			< 0.001	
Peak rate of wall	Mean	-15.3	-15.1	-15.5	-13.7	-14.4	-15.5	-12.5	-13.1	-13.9	-10.8	-11.8	-13.1	-9.2	-10.2	-11.3
Thinning (cm ² /s)	SD	1.44	1.8	0.9	2.2	2.5	2.7	1.5	1.8	1.7	1.3	0.9	0.9	1.4	1	0.9
	Р		0.717			< 0.001			< 0.001			< 0.001			< 0.001	
Tau	Mean	39.8	38.4	35.9	43.2	38.9	33.8	48.8	46.1	39.4	53.7	48.2	43.6	55.3	50.7	45
(ms)	SD	11.9	11.2	13.5	11.6	9.5	8.1	8	6.1	7.4	5.1	6.3	10.1	10.1	10.2	9.4
	Р		0.852			< 0.001			< 0.001			0.002			< 0.001	

^a Base, baseline value; 10%, value when the Myosplints are tightened to a 10% stress reduction level; 20%, value when the Myosplints are tightened to a 20% stress reduction level; *P*, *P* value of ANOVA for each interval.

(P = 0.005), and between the 4th and the 5th interval (P = 0.026). When the Myosplints were tightened, peak rate of wall thinning decreased significantly at the 2nd, 3rd, 4th and 5th interval (Table 2).

Baseline Tau (Table 2) differed significantly between the 1st and the 2nd interval (P = 0.037), between the 2nd and the 3rd interval (P = 0.033), and between the 3rd and the 4th interval (P = 0.013). When the Myosplints were tightened, Tau decreased significantly at the 2nd, 3rd, 4th and 5th interval (Table 2).

4. Discussion

The term 'ventricular remodelling' is used to address the changes in ventricular geometry, volume, mass and myocellular structure in response to myocardial injury or alteration in loading conditions. This process can follow myocardial infarction and mechanical overload such as in hypertension or valvular heart disease, and can also occur in inflammation and dilated cardiomyopathy. Despite these various etiologies, the remodelling process involves similar alterations of the biology and structure of the myocyte, the non-myocyte component as well as the architecture and chamber geometry of the heart [7]. This process may be considered initially as an adaptative mechanism following the initial damage to the myocardium, with a stroke volume maintained through the Frank-Starling mechanism. However, progressive ventricular remodelling is ultimately a maladaptative process, as the further increase in ventricular size occurs at the expense of increasing wall stress, according to the law of Laplace, which in turn promotes further dilatation and reduced pump function [8].

In this experimental set-up of volume loading, the systolic function is improved by the Myosplint as evidenced by significant increase of dP/dt and wall thickening for CVP values above 10 mmHg. The positive effect of the Myosplint for higher loading conditions may be related to the profile of the Starling's curve. This curve, relating end-diastolic LV volume to stroke volume, shows an ascending limb of the stroke volume for low LV volume up to a certain point, beyond which further increase in end-diastolic LV volume is accompanied by a decrease in the stroke volume (descending limb). The mechanical energy delivered during the contraction depends on the area of chemically active surfaces, i.e. on the length of muscle fibres represented by the end-diastolic LV volume [9]. In this model, the optimal end-diastolic LV volume is reached at the 3rd interval (CVP value between 10.1 and 15 mmHg).

The Myosplint has a positive impact on the diastolic function too, with a decrease of LVDEP for CVP values above 10 mmHg, as well as a decrease of the peak rate of wall thinning and the logistic time constant of isolovolumic relaxation, for CVP values above 5 mmHg. This difference of onset of improvement may be due to the high sensitivity of both last parameters to diastolic function alteration [5,6].

Of note, baseline Tau is dependent on the loading conditions in this model as reported by Matsubara et al. [5] in a canine model.

Other parameters of the systolic and diastolic function measurable with crystal technology, such as short axis shortening, wall stress, ejection fraction or cavity enlargement were not recorded because they involve crystals placed on opposite walls of the LV [6,8]. With the bilobular shape generated by the Myosplint, comparisons of such measurements with those at baseline, while the LV has a globular shape, cannot be performed. Therefore, the parameters were limited to those provided by transmural pair of crystals placed on the antero–lateral wall of the LV, at equidistance between the two rows of Myosplint pads in order to obtain comparable data.

Because cardiac transplantation is available to a minority of heart failure patients, cardiomyoplasty and partial left ventriculectomy have been developed for those who do not improve with optimal medical therapy. These recent surgical treatments may derive benefit by their ability to arrest or substantially reverse the remodelling process. Dynamic cardiomyoplasty has been shown to improve LV function in selected patients [10,11]. While systolic squeezing assist effect may play a role in some patients, others do not require this effect to achieve benefits from the procedure. In this subgroup, cardiomyoplasty has been suggested to act more passively like an elastic girdle around the heart [12]. Partial left ventriculectomy directly reverses remodelling by removing a portion of the lateral wall [13]. While some patients benefit from this procedure, early failures have precluded its widened application [14,15]. Therefore, selection criteria appear to play a role in predicting the outcomes with these two procedures [16]. Performance may be improved by geometric alteration when the intrinsic integrity of the myocyte contractile function is preserved [17]. However, tools to determine the integrity of the myocyte function are imprecise and, as of today, there is no reliable mean to predict a beneficial effect of such techniques.

In comparison with these procedures, the Myosplint appears as a simpler and potentially reversible alternative. Unlike cardiomyoplasty, its implantation does not require an extensive operation, and no prolonged delay is expected before a clinical improvement. Unlike partial left ventriculectomy, neither cardiopulmonary bypass, nor damage to the myocardial muscle, nor ventricular sutures is required. Moreover, because of its concept, the Myosplint can be removed and the original geometry of the LV may be restored in case of failure. This appears to be a substantial advantage in view of the absence of reliable outcome predictors of the other techniques aiming at reversing the remodelling process.

Importantly, none of the systolic or diastolic parameters ever worsened during the experiment at any CVP interval. This could be achieved by careful insertion of the Myosplint under echocardiographic control in order to avoid any damage or involvement of the mitral papillary muscle, which could have caused valvular distortion with subsequent regurgitation and worsening of the heart function.

One potential limitation of this study lies in the fact that the experiment was performed on a normal functioning heart. However, while this is true for the initial part of the protocol, in the later stages, which are the most relevant for the clinical application of the Myosplint, the hearts were in a failing state because of the important volume loading over a prolonged period. This is clearly demonstrated by the baseline values of the hemodynamical as well as the contractility and relaxation parameters.

In conclusion, the Myosplint has shown to improve the systolic and diastolic function in this experimental set-up. This simple device has the potential to be reversible, and does not require an extensive surgical procedure, nor the use of cardiopulmonary bypass. The results of a clinical trial which has started in Europe will help to determine whether these findings pertain to the clinical setting.

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