Acute and Short-Term Effects of Partial Left Ventriclectomy in Dilated Cardiomyopathy

Assessment by Pressure-Volume Loops

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OBJECTIVES

The aim of this study was to evaluate the short-term effects of partial left ventriculectomy (PLV) on left ventricular (LV) pressure-volume (P-V) loops, wall stress, and the synchrony of LV segmental volume motions in patients with dilated cardiomyopathy.

BACKGROUND

Surgical LV volume reduction is under investigation as an alternative for, or bridge to, heart transplantation for patients with end-stage dilated cardiomyopathy.

METHODS

We measured P-V loops in eight patients with dilated cardiomyopathy before, during and two to five days after PLV. The conductance catheter technique was used to measure LV volume instantaneously.

RESULTS

The PLV reduced end-diastolic volume (EDV) acutely from 141 ± 27 to 68 ± 16 ml/m² (p < 0.001) and to 65 ± 6 ml/m² (p < 0.001) at two to five days postoperatively (P-V loops, wall stress, and the synchrony of LV segmental volume motions in patients with dilated cardiomyopathy. Often, PLV is combined with mitral valve and tricuspid valve reconstruction (1–5). The procedure aims to restore the normal relationship between ventricular mass and dimension to normalize wall stress throughout the cardiac cycle. From a multiple compartment elastance model study, in which the effects of PLV were simulated, it was concluded that PLV leads to a reduction in wall stress (WS) for any level of LV pressure (6). This implies an improvement of the efficiency by which WS is transferred to intraventricular pressure.

In patients with dilated cardiomyopathy, impairment of cardiac performance is generally associated with impaired LV relaxation and diastolic and systolic wall motion abnormalities (7–9). Nonuniformity in wall motion reduces the mechanical efficiency of ventricular ejection and contributes to regional diastolic abnormalities (10). In a previous study we observed marked LV nonuniformity in patients with dilated cardiomyopathy before undergoing cardiomyoplasty. Six months after cardiomyoplasty, WS was decreased by the wrapped latissimus dorsi muscle and the decreased LVEDV, whereas LV nonuniformity was markedly decreased (7). Because PLV should lead to a decrease in WS, PLV should also improve the mechanical efficiency of LV ejection by reducing LV nonuniformity.

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Jan J. Schreuder, MD, † Department of Cardiology, Leiden University Medical Center, Leiden, The Netherlands. This study was funded by the Cardiovascular Research Institute Maastricht, Maastricht, The Netherlands; and the Department of Cardiology, Leiden University Medical Center, Leiden, The Netherlands. This study was funded by the Cardiovascular Research Institute Maastricht, Maastricht, The Netherlands.
The conductance catheter was used to study LV nonuniformity by measuring regional synchrony of contraction and relaxation. With this technique, the volume changes occurring in five LV segments, perpendicular to the long heart axis, can be measured throughout the cardiac cycle. By comparing changes in LV segmental volumes with total LV volume changes, the LV synchrony, representing mechanical efficiency, can be calculated throughout the cardiac cycle. To evaluate the acute hemodynamic effects of PLV in patients with dilated cardiomyopathy, we measured LV pressure-volume (P-V) loops and LV synchrony before, during, and up to five days after the operation.

METHODS

Patients. Eight consecutive patients, 45 to 60 years old, scheduled to undergo PLV were studied preoperatively, during the procedure, and up to five days postoperatively. All patients had dilated cardiomyopathy, either idiopathic or related to postischemic damage, or Chagas disease (Table 1). At the time of the PLV procedure, seven patients were in NYHA functional class IV, and one in class III (patient 1). All patients were receiving chronic treatment with diuretics, digoxin, and angiotensin-converting enzyme (ACE) inhibitors at the time of PLV. The patients studied were operated on in the Hospital Angelina Caron, Brazil.

Before surgery as well as up to five days after the operation, the patients were catheterized. All operations were performed by R.J.V.B. The study was approved by the medical ethics committee of the hospital. Informed patient consent was obtained for insertion of the catheters and the measurements.

Surgical procedure. The surgical technique has been described previously by Batista et al. (1). After inducing anesthesia, a midline sternotomy was performed. Bypass was routinely instituted, using double venous cannnulas. All procedures with exception of the autotransplantation (Table 1) were performed on a beating heart. In seven patients a tricuspid valvuloplasty was done by a modified De Vega plasty. Subsequently, in all cases the posterolateral wall was excised, starting near the apex of the heart. The incision was performed down to the crux of the heart, excising the obtuse marginal coronary artery, and in between the papillary muscles to preserve the mitral valve. The mitral valve was reconstructed using the Alfieri repair technique, approximating the mid-portion of the free edge of the anterior and posterior leaflets with a single suture (12). The ventriculotomy was closed with absorbable sutures.

Anesthesia. All patients received 0.05 mg/kg of lorazepam as oral premedication 2 h before surgery. Anesthesia was induced with hypnomidate (0.25 mg/kg) and maintained by etranre (0.2% to 0.5%) and fentanyl (3µg/kg/h). For muscle relaxation, 0.1 mg/kg of pancuronium bromide was given. The patients were ventilated with an oxygen/air mixture (FiO2 0.5) at a ventilatory rate of 12/min and ventilatory volume was adjusted to maintain arterial CO2 tension between 32 and 42 mm Hg. Sodium-nitroprusside (SNP), isoprenaline and adrenaline were used during surgery before and after bypass as needed to maintain systolic blood pressure between 80 to 100 mm Hg and heart rate between 90 to 120 beats/min.

Instrumentation. Patients were sedated and heparinized before catheterization. A Swan-Ganz thermodilution catheter was placed via a subclavian vein into the pulmonary artery. A dual-micromanometer transducer conductance

<table>
<thead>
<tr>
<th>Pts</th>
<th>Age; Gender</th>
<th>Origin</th>
<th>CI l/min/m²</th>
<th>Associated Surgery</th>
<th>Area exc cm²</th>
<th>Mass exc g</th>
<th>Rhythm Pre–Post</th>
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<td>Idiopathic</td>
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<td>48</td>
<td>SR-SR</td>
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<tr>
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<tr>
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</tr>
<tr>
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<td>86</td>
<td>AF-AF</td>
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<tr>
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<td>60; M</td>
<td>Ischemic</td>
<td>1.54</td>
<td>TR, MR, CABG 2X</td>
<td>33</td>
<td>66</td>
<td>AF-SR</td>
</tr>
<tr>
<td>6</td>
<td>45; M</td>
<td>Chagas</td>
<td>1.54</td>
<td>TR, MR, RAR, RVR</td>
<td>43</td>
<td>70</td>
<td>AF-SR</td>
</tr>
<tr>
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<td>74</td>
<td>83</td>
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<tr>
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<td>2.44</td>
<td>TR</td>
<td>26</td>
<td>46</td>
<td>SR-SR</td>
</tr>
</tbody>
</table>

AF = atrial fibrillation; AT = autotransplant; AVR = aortic valve reconstruction; CABG = coronary artery bypass grafting; CI = cardiac index; LAR = left atrial reconstruction; MR = mitral valve repair; RAR = right atrial reconstruction; RVR = right ventricular reduction; SR = sinus rhythm; Th = thrombectomy; TR = tricuspid valve repair.
catheter (F7, Sentron, CD Leycom, Zoetermeer, The Netherlands) was inserted via the femoral artery into the LV for measurement of aortic and LV pressures and LV volume. The feasibility of the conductance catheter method during cardiac surgery has been shown in a previous study (13). The correct position of the conductance catheter was verified by fluoroscopy and by inspection of the segmental conductance signals. The conductance catheter was connected to a Leycom Sigma-5DF signal conditioner-processor (CD Leycom, Zoetermeer, The Netherlands) to measure instantaneous LV and segmental volumes (7,11,13,14). The dual-field excitation mode, which has been shown to improve the accuracy of the method, was used in all patients (15).

The conductance catheter measures the instantaneous volumes of five ventricular segments delineated by selected catheter electrodes. It has been shown previously that the time-varying segmental conductance reflects time-varying segmental LV volume as obtained by cine computed tomography (CT) in canine hearts (16). Total volume is calculated as the sum of the segmental volumes. Occasionally, the segment near the base of the heart was in the aorta and therefore not included in the calculations.

The conductance catheter not only continuously measures the relative amount of blood in the LV but also the conductance of the myocardium and other surrounding tissues, the parallel conductance. This parallel conductance offset term (Vc) for the LV was estimated by injection of 7.5 ml hypertonic saline (6%) into the pulmonary artery (11). The Vc estimation was performed by the dedicated software package CONDUCT-PC (CD Leycom, Zoetermeer, The Netherlands). The implemented algorithm finds the best Vc values, thus avoiding an operator-dependent bias. At each stage of the procedure the parallel conductance measurements were performed in duplicate. To get absolute volumes with the conductance catheter technique, the effective conductance SV must be matched with a gold standard SV measurement technique. Therefore, cardiac output (CO) was determined at each stage of the procedure by performing five thermodilution measurements, using a cardiac output computer (COM-2, Baxter), and using injections of 10 ml ice-cold glucose 5%. Consequently, absolute LV volumes were calculated by matching effective conductance SV with simultaneously measured thermodilution SV, and by subtracting parallel conductance correction volume from total conductance volume. Both methods are indicator-dilution methods and therefore largely independent of anticipated geometric changes, as will be caused by the PLV.

**Ventricular synchrony.** Regional contraction patterns were studied by recording the segmental conductance signals individually, making assessment of regional synchrony of contraction and relaxation possible. The volume segments are located perpendicular to the long heart axis. A segmental volume signal was defined as synchronous whenever the change in this segmental signal was in the same direction as the simultaneous change of the total volume signal. The LV segmental synchrony along the long heart axis was quantified by calculating the percentage of time over the cardiac cycle that a segmental signal was synchronous with the total volume signal. Thus, a segmental synchrony of 80% means that the segmental conductance signal was in phase (i.e., showing changes in the same direction) with the total volume signal during 80% of the full cardiac cycle. Consequently, during the remaining 20% of time this segmental signal would show volume changes in the opposite direction of the total volume signal. An overall LV synchrony index (Sync) was obtained by determining the synchrony for all individual segments, and calculating their mean value.

**Time-varying wall stress.** Obtaining ventricular pressure P(t) and volume V(t) simultaneously enabled us to calculate time-varying wall stress, WS(t). Arts et al. (17) have shown that WS is fairly uniform throughout the ventricular wall and is relatively independent of the geometry of the ventricle and can be calculated as

\[
WS(t) = P(t) + (1 + 3\cdot V(t)/V_{wall}).
\]

Preoperative (pre-op) wall volume (Vwall) was calculated as the volume of a spherical shell with a thickness equal to the thickness of the excised segment and an internal diameter as estimated from pre-op end-diastolic volume (EDV). Postoperative (post-op) Vwall was calculated by subtracting the volume of the excised segment from the pre-op wall volume. Peak WS is the maximum of the WS(t) curve.

**Data acquisition and analysis.** Electrocardiogram ECG (extremity leads), aortic pressure (PaO), LV pressure and LV volume signals were digitized at a sampling rate of 200 Hz and stored on hard disc for subsequent analysis. The dedicated data-acquisition and data-analysis software package CONDUCT-PC was applied for conductance-catheter related data analysis. In addition to the volumetric variables, pressures, and the peak first derivatives, the following variables were calculated: tau, the time constant of LV pressure relaxation, which was defined as the time required from the LV pressure at peak negative dP/dt to be reduced by half (18); peak ejection rate and peak filling rate, calculated as maximal −dV/dt and maximal dV/dt, respectively. Effective LV ejection fraction (EF) was calculated from the thermodilution derived stroke volume (SV) and the LVEDV measured by the conductance catheter.

**Measurement protocol.** Hemodynamic measurements were obtained at five stages: before anesthetic induction (pre-surgery); immediately preceding cardiopulmonary bypass (pre-bypass); 15 min after cardiopulmonary bypass (post-bypass); after sternal closure (post-surgery); and during recatheterization at two to five days after the operation (re-cath). Because the surgery and large changes in both pre- and afterload may affect catheter position and parallel conductance, we performed the following measurements to calibrate the conductance catheter. At each stage of the procedure we determined parallel conductance in duplicate,
blood resistivity and CO by thermodilution (5 injections). Steady-state P-V loops were acquired during at least 15 s, achieved by breath-holding when the patients were awake and by ventilatory stop when the patients were anesthetized. **Statistical analysis.** Statistical analysis of all hemodynamic measured variables was performed by repeated-measures analysis of variance (ANOVA), using the following multiple linear regression model:

\[ Y = a_0 + \sum a_{C_i} \cdot C_i + \sum a_{P_i} \cdot P_i \]

(19). The dependent \( Y \) represents the various hemodynamic variables (heart rate, cardiac index, etc.), the dummy variables \( C_i \) code the conditions (pre-surgery, pre-bypass, post-bypass, post-surgery and re-cath), and the dummy variables \( P_i \) code the individual patients. For the set of patient variables we used effects coding; for the conditions we used reference cell coding, with the pre-surgery condition as control group. Using this coding, the intercept \( a_0 \) yields the mean value of the dependent in the pre-surgery condition, and the coefficients \( a_{C_i} \) give the difference between the corresponding condition (pre-bypass, post-bypass, etc.) and the pre-surgery value. The \( p \) values of the coefficients \( a_{C_i} \) indicate the significance of these differences. The dummy variables \( A_i \) account for between-subject differences, allowing all patients to have a different mean value. The standard deviation (SD) of the group of patient coefficients, \( a_{P_i} \), is a measure of between-subject variability. Statistical analysis was performed using commercial software (Microsoft Excel 97, Microsoft Corp.). Statistical significance was defined as \( p < 0.05 \).

**RESULTS**

Clinical and operative data are presented in Table 1. Mean excised endocardial area was 46 ± 18 cm²; mean excised cardiac mass was 70 ± 17 g.

Missing data for Patients 1 and 3 in the pre-surgery series is due to failure to insert the conductance catheters before induction. No re-cath data are available for Patient 7, who died on the third postoperative day due to an arrhythmia. Also, in two other patients no data on re-cath are available; in Patient 1 this was due to failure to insert the conductance catheter in the LV, whereas Patient 6 was still dependent on intensive care treatment at the time of the scheduled re-cath.

Low doses (<0.5 μg/kg/min) of SNP were used pre- and post-bypass in Patients 1, 2, 3, 7, and 8, whereas Patients 4 and 6 received similar doses only at post-bypass. Low doses of isoprenaline or adrenaline were used post-bypass in Patients 2, 5, and 7; these infusions were stopped 5 min before the measurements were performed. During pre-surgery and re-cath measurements, none of these drugs were administered.

In Figure 1, the P-V loops of Patient 2 at all operative stages, and one extra re-cath at two days after PLV, are shown. In Figure 2, the left ventricular P-V loops of all eight patients before and after the PLV are given. Preoperatively, the P-V loops are heterogeneous; the P-V loops of Patients 6 and 7 show characteristics of aortic regurgitation, while signs of mitral regurgitation are present in Patient 5. Postoperatively, the P-V loops are more homogeneous. An obvious shift toward lower LV volumes after PLV is present in all patients, indicating substantial surgical reversed re-

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**Figure 1.** P-V loops of Patient 2 during all stages of the PLV procedure. The P-V loops are shifted to the left after the PLV. At the first re-cath after two days, EDP and EDV were increased compared to post-surgery, whereas five days after PLV, both EDP and EDV were lowest.
modeling. No clear signs of aortic and mitral regurgitation are present in the P-V loops after surgery.

Table 2 provides data on all eight patients measured at all stages pre-surgery, pre-bypass, post-bypass, post-surgery, and at re-cath two to five days after PLV. All P-V data were measured during 15-s sampling periods, and the values of all beats were averaged. No significant changes in heart rate (HR), SV, LV EDP, peak $+\frac{dP}{dt}$, peak $-\frac{dP}{dt}$, LV peak ejection rate, and LV peak filling rate were observed between these stages. Cardiac index (CI) increased from pre-surgery 1.5 ± 0.5 to 2.6 ± 0.6 l/min/m$^2$ (p < 0.002) at post-surgery, and was 1.8 ± 0.3 l/min/m$^2$ (NS) at re-cath. The LVEF increased after PLV in all patients from 15 ± 8% at pre-bypass to 35 ± 6% at post-surgery (p < 0.001) and remained increased at 26 ± 3% during the re-cath (p < 0.003) compared to the pre-PLV series. The LVEDV decreased from 141 ± 27 at pre-bypass to 68 ± 16 ml/m$^2$ (p < 0.001) at post-surgery and to 65 ± 6 ml/m$^2$ (p < 0.001) at re-cath. The LV end-systolic volume (ESV) decreased from 109 ± 23 at pre-surgery to 37 ± 11 ml/m$^2$ (p < 0.001) at post-surgery and to 38 ± 5 ml/m$^2$ (p < 0.001) at re-cath. Tau decreased from 54 ± 8 to 38 ± 6 ms (p < 0.03) when comparing the pre-bypass and the post-surgery values. Peak WS decreased from a pre-surgery

Figure 2. P-V loops of all eight patients before PLV (thick lines), after PLV (fine lines), and at re-cath two to five days after PLV (dotted lines).
Table 2. Hemodynamic Variables at all Stages of the PLV Procedure

<table>
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<tr>
<th>Condition</th>
<th>Patient</th>
<th>HR (bpm)</th>
<th>CI (l/min/m²)</th>
<th>SV (ml)</th>
<th>EF (%)</th>
<th>EDV (ml/m²)</th>
<th>ESV (ml/m²)</th>
<th>EDP (mm Hg)</th>
<th>+dP/dt (mm Hg/s)</th>
<th>−dP/dt (mm Hg/s)</th>
<th>TAU (ms)</th>
<th>PER (ml/s)</th>
<th>PFR (ml/s)</th>
<th>PWS (mm Hg)</th>
<th>Sync (%)</th>
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<td>92</td>
<td>14</td>
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<td>702</td>
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<td>261</td>
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</table>

Pre-bypass

| Post-bypass | 1       | 89       | 3.46          | 56     | 23     | 168         | 113         | 23          | 117            | 946            | 50       | 610        | 650        | 401        | 80      |
|            | 2       | 73       | 2.25          | 55     | 24     | 129         | 97          | 20          | 114            | 801            | 56       | 553        | 613        | 295        | 67      |
|            | 3       | 117      | 1.35          | 17     | 7      | 171         | 149         | 35          | 80             | 676            | 60       | 250        | 343        | 286        | 66      |
|            | 4       | 119      | 2.16          | 29     | 17     | 106         | 83          | 8           | 62             | 534            | 49       | 441        | 526        | 158        | 67      |
|            | 5       | 112      | 1.12          | 10     | 105    | 92          | 14          | 60          | 379            | −448           | 51       | 495        | 492        | 138        | 70      |
|            | 6       | 79       | 2.63          | 58     | 24     | 154         | 95          | 14          | 94             | 775            | 59       | 1099       | 831        | 201        | 69      |
|            | 7       | 100      | 1.34          | 21     | 8      | 167         | 135         | 21          | 79             | 536            | 63       | 574        | 912        | 272        | 58      |
|            | 8       | 134      | 1.34          | 19     | 8      | 129         | 105         | 13          | 98             | 1419           | 40       | 728        | 968        | 282        | 68      |
| Mean      |         | 103      | 1.96          | 35     | 15     | 141         | 109         | 18          | 88             | 758            | 54       | 594        | 667        | 254        | 68      |

P-value 0.1

Post-surgery

| Post-surgery | 1       | 97       | 3.11          | 46     | 33     | 98          | 57          | 18          | 59             | 630            | 34       | 436        | 616        | 219        | 76      |
|             | 2       | 93       | 2.34          | 45     | 38     | 67          | 36          | 13          | 65             | 759            | 40       | 369        | 471        | 140        | 75      |
|             | 3       | 126      | 2.63          | 30     | 41     | 51          | 27          | 27          | 72             | 964            | 40       | 341        | 431        | 137        | 74      |
|             | 4       | 105      | 1.75          | 27     | 31     | 55          | 21          | 9           | 46             | 501            | 46       | 654        | 385        | 77         | 70      |
|             | 5       | 109      | 3.58          | 76     | 43     | 76          | 37          | 25          | 81             | 915            | 40       | 636        | 820        | 153        | 90      |
|             | 6       | 123      | 2.90          | 41     | 36     | 65          | 43          | 16          | 69             | 692            | 44       | 658        | 127        | 86         |        |
|             | 7       | 111      | 2.19          | 32     | 25     | 79          | 37          | 11          | 83             | 1095           | 32       | 434        | 635        | 177        | 88      |
|             | 8       | 130      | 2.50          | 37     | 34     | 57          | 35          | 14          | 94             | 1569           | 30       | 394        | 375        | 224        | 81      |
| Mean       |         | 112      | 2.63          | 42     | 35     | 68          | 37          | 17          | 71             | 891            | 38       | 490        | 533        | 157        | 80      |

P-value 0.002, 0.1, 0.001, 0.001, 0.001, 0.004, < 0.002, 0.03, < 0.001, 0.007

(continued on next page)
The LV synchrony index (Table 2) increased from 68 ± 6% at pre-bypass to 80 ± 7% (p < 0.01) at post-surgery and to 73 ± 4% (NS) at re-cath. Five segmental volumes were included at pre-bypass in all patients, with exception of Patient 2, in which four segments were included. Four segments were included in all patients at post-bypass, except Patient 2, in which three segments were included.

Results of the calculated WS throughout one cardiac cycle of all patients are presented in Figure 3. The same cardiac cycles are presented as those used for the P-V loops in Figure 2. In all patients, systolic WS was decreased after the procedure. Immediately after PLV a decreased WS throughout the cardiac cycle was observed in all patients with the exception of Patient 1, where higher values during the diastolic filling phase were measured. In three out of five patients (Patients 2, 3, 5) a decreased WS throughout the cardiac cycle during the re-cath at two to five days after PLV was measured. In Patient 4, end-diastolic WS was increased compared to pre-surgery, and in Patient 8 the WS was increased during the diastolic filling phase.

**DISCUSSION**

This study shows the acute and short-term effects of PLV combined with various other cardiac reconstructions. The LV volumes were reduced as anticipated with a concomitant increase in LVEF, together with unchanged CI, SV, and LV EDP. Peak WS decreased in all patients after PLV. The occurrence of systolic and diastolic paradoxical and asynchronous segmental LV volume motions decreased after PLV.

Hemodynamics. The pronounced acute average decrease in LVEDV by 52% immediately post-PLV, as measured with the conductance catheter technique, concurrently with an average mass reduction of 70 g, are comparable with results as measured with transesophageal echocardiography by McCarthy et al. (3), Gorcsan et al. (4), and Popovic et al. (5). Differences in decreases in EDV for a certain amount of mass resection in different studies might be attributed to differences in wall thickness; therefore, to really compare effects of mass resection on EDV, the excised endocardial area should be known.

The increases in effective LVEF from 15% to 35% were similar to the intraoperative results of McCarthy et al. (3), from 16% to 33%. The two-week post-PLV LVEF results of Bocchi et al. (2), from 17% pre-PLV to 26% (n = 24) are also similar to the one-week post-PLV mean values of McCarthy et al. (3) of 23% and with our two to five-day post-PLV value of 26%.

The observed acute decreases in LVEDV and ESV and increases in LVEF after PLV are comparable with long-term hemodynamic effects of cardiomyoplasty in patients with dilated cardiomyopathy. In a clinical study, we ob-
served an increased LVEF from 27% to 40%, a decrease of 26% in LVEDV as well as decreases in LVEDP at six months after cardiomyoplasty. At 12 months after cardiomyoplasty, LVEDV was decreased by 40% (7).

A striking observation of the present and other PLV studies (2,3,5) is the unchanged SV after the procedure in these patients. Reducing LV dimensions in normal hearts by about 50% should also reduce SV considerably. Therefore, these dilated hearts apparently possess a property that preserves the SV when the volume has been reduced by the PLV. This property is probably related to afterload decrease, similar to the effects by which nitroprusside administration in congestive heart failure patients may increase SV (20,21).

Cardiac index (CI) did not change significantly when comparing pre-PLV to re-cath values, which accords with the study of McCarthy et al. (3). However, comparing pre-surgery data with end-of-surgery data of the present study revealed an increased CI. Moreover, in the four low-output-state patients, who were re-catheterized (Patients 2 through 5), this increase was maintained. These findings are similar to Bocchi et al. (2) and Popovic et al. (5), who also found increases in CI. Also, peak +dP/dt was increased in the four low-output patients at re-cath, which indicates an increased contractile state because LV end-systolic pressure (ESP) was similar, and the LVEDV was decreased compared to the pre-PLV state. Additionally,
these patients showed the lowest LV synchrony indices before PLV (Table 2), which were increased after PLV.

In congestive heart failure patients, the relaxation indices peak $-\frac{dP}{dt}$ and Tau are generally decreased and prolonged, respectively (8,9). In the present patient group these variables were grossly abnormal, with the exception of Patient 8. During re-cath, peak $-\frac{dP}{dt}$ was more negative in the four patients with the preoperatively low-output, and Tau was improved significantly, when comparing pre-bypass with post-surgery values, indicating a relaxation improvement of the compromised early diastolic phase.

The LVEDP did not change significantly due to PLV, although in the two Patients (Patients 2 and 3) who were recatherized on their fifth post-op day a decrease in EDP was measured (Fig. 1). From the PLV model study of Dickstein et al. (6) it can be predicted that LVEDP rises after PLV, with a positive correlation to the excised LV mass. This was confirmed by the intraoperative results of Gorcsan et al. (4), which showed increased regional end-diastolic stiffness. However, McCarthy et al. (3) observed postoperative (one week) decreases in left atrial pressures, and Popovic et al. (5) reported a significant decrease in LVEDP two weeks after surgery. This might imply that during the first days after the operation, LVEDP is in-

Figure 4. Tracings of LV volume segments of Patients 2 and 3 before and after PLV and at re-cath five days after PLV. $V_{lv}$ = total LV volume in time during four consecutive heart beats. Dotted lines = start of the ejection phase. The pre-PLV segmental synchrony values of Patient 2 were 64%, 62%, 63% and 80% for, respectively, segments 1 through 4 yielding a synchrony index (Sync) of 68%, whereas at re-cath the segmental values were 75%, 77%, 71% and 80% yielding a Sync of 76%.
creased, possibly due to edema around the suture-line, which might temporarily decrease ventricular distensibility. **Wall stress.** Ventricular wall stress is related directly to cavity volume and inversely to wall thickness. Hayashida et al. (22) demonstrated in patients with dilated cardiomyopathy a markedly elevated WS throughout the cardiac cycle. The reduction in EDV and ESV by PLV will decrease WS, which, however, will be partly offset by the reduction in wall volume. Arts et al. (17) demonstrated that the ratio of myocardial fiber stress to LV pressure depends mainly on the ratio of cavity volume to wall volume, and is quite independent of geometry. Dickstein et al. (6) predicted in their model study, using the same WS equation, a reduction in peak WS for each reduction in mass of up to 75 g. From the present clinical data we confirm the decrease in peak stress after PLV, which induces a decrease in cardiac afterload stress. Popovic et al. (5) observed significant decreases in LV circumferential end-systolic and end-diastolic stresses two weeks after PLV.

The calculated WS is an estimation of the stress averaged over the total LV wall, neglecting regional differences. In this patient group we observed before PLV a marked asynchrony between LV segmental volume changes throughout the cardiac cycle. This is an indication for marked regional differences in WS at any moment in the cardiac cycle. Therefore, ventricular efficiency and maximum work output might be compromised by asynchrony and regional stress differences. The LV nonuniformity might have led to an undetermined inaccuracy in the estimation of WS using the formula of Arts et al. (17), which assumes homogeneously distributed fiber stress and strain. In case of asynchrony the actual regional peak wall stress is likely to be higher. Therefore, the calculated reduction in WS induced by PLV probably underestimates the actual reduction.

**Wall motion asynchrony.** Figure 4 shows the segmental conductance signals of two patients before and after PLV. Prior to PLV, marked paradoxical movements throughout the cardiac cycle of the ventricular volume segments can be observed, indicating diastolic and systolic wall motion asynchrony, which were converted into more synchronous segmental volume signals after PLV. This is consistent with previous studies showing highly asynchronous regional wall motions in patients with dilated cardiomyopathy (7,22). Nonuniformity reduces the mechanical efficiency of ventricular ejection by inducing a premature onset and a decreased rate of LV pressure decline (10). Wall motion asynchrony is a major determinant of impaired LV filling in patients with healed myocardial infarction (23). Nonuniformity can be decreased by nitroprusside through afterload reduction (8).

In a previous study in patients with dilated cardiomyopathy we demonstrated that both nitroglycerin and cardiomyoplasty improved segmental synchrony, probably by reducing myocardial afterload stress (7). The synchronizing effects of cardiomyoplasty might be attributed to the active latissimus dorsi muscle stimulation synchronizing effect (24) as well as to the decrease in LVEDV (7). Analogously, the acute synchronizing effects of PLV and associated procedures might be primarily ascribed to the marked acute decrease in LVEDV, and therefore to the PLV itself.

The aspect of segmental volume asynchrony reveals the ineffective mechanical cardiac work in these severe heart failure patients. Segmental synchrony, during both contraction and relaxation, is a major determinant of efficient mechanical cardiac performance (10). Figure 5 shows a linear regression diagram between CI and the LV synchrony index from all measured values throughout the procedure in this study. This highly significant correlation clearly demonstrates the importance of a uniform contracting and relaxing ventricle for efficient cardiac performance. The occurrence of marked nonuniformity in LV contraction and relaxation in congestive heart failure patients and the synchronizing effects of sodium-nitroprusside, nitroglycerin, cardiomyoplasty and PLV indicate a reversible state in the patients studied, which is caused by decreases in afterload stress and WS.

**Study limitations.** The specific effects of PLV are difficult to identify because of associated surgical procedures: coronary artery bypass graft surgery, and mitral, tricuspid, and aortic valve reconstruction. Only in Patient 8 was an isolated PLV procedure performed. In this NYHA functional class III patient, the PLV procedure induced short-term adverse effects concerning CI, peak +dP/dt, peak −dP/dt and LVEDP when the pre-surgery and the two-day post-op data are compared. Although most patients had additional procedures, some major effects such as acute LV volume decrease, an increase in uniformity of contraction and relaxation, and a decrease in WS can be primarily attributed to PLV.

Considerable parts of the measurements were performed in patients under anesthesia. Also, during the operation inotropic and vasodilatory therapy was given. However, during the pre-surgery and the re-cath measurements,
patients did not receive drug treatment other than digoxin, ACE inhibitors, and diuretics.

Another important limitation is the absence of clinical follow-up and long-term hemodynamic measurements. Thus, our study does not address the question of the long-term value of the PLV operation.

Conclusions. Our study describes the acute and short-term effects of PLV and associated cardiac surgical procedures. The issue, therefore, remains how these early changes relate to long-term hemodynamic condition and survival.

Reduction in LV cavity volume clearly resulted in significant net reduction in peak WS and an increase in LVEF. When analyzing the entire patient group, no significant changes in CI and SV were present, but in four patients with a preoperative low output state, CI was increased at re-cath.

The synchrony in ventricular contraction and relaxation of the dilated LV probably improved because the decrease in LV volume resulted in a decrease in WS. This effect may be ascribed directly to the PLV and not to the associated procedures. This increase in synchrony led to an improved mechanical efficiency of ventricular ejection, with an increased efficiency of the transmission of WS into intraventricular pressure, compensating an expected decrease in SV. This is also demonstrated by the significantly positive correlation between CI and LV synchrony index. Therefore, the transition from a nonuniform contraction and relaxation LV pattern to a more uniform pattern might be a rationale of the working mechanism of the Batista procedure. Potentially, the PLV procedure should consequently be restricted to patients with dilated cardiomyopathy having a major asynchronous LV contraction and relaxation pattern under an optimal drug regimen.

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