Cardiac Exercise Hemodynamics Late After Partial Left Ventriculectomy

Karen B. James, MD; Garrie Haas, MD; Suzanne R. Lutton, MD; David Prior, MD; Kathy Wolski, MPH; Tiffany Buda, RN; Patrick M. McCarthy, MD

**Background**—Although some patients report favorable activity levels late after partial left ventriculectomy (PLV), their exercise physiology has not been well described.

**Methods and Results**—We performed upright bicycle hemodynamics in 10 patients (9 men) aged 56±12 years at 1.7 years after PLV. Ejection fraction was 25±4%. Patients biked 10±7 minutes. With exercise, the mean pulmonary arterial pressure rose from 36±12 to 52±10 mm Hg ($P=0.0003$). The mean pulmonary capillary wedge pressure rose from 25±14 to 36±9 mm Hg ($P=0.0566$), and the cardiac index rose from 2.2±0.5 to 3.8±1.6 L · min$^{-1}$ · m$^{-2}$ ($P=0.0077$). The mixed venous oxygenation with exercise declined from 44±9% to 24±17% ($P=0.0220$), and the pulmonary vascular resistance increased from 2.0±0.9 to 2.3±1.1 Wood units ($P=0.5566$).

**Conclusions**—In late follow-up after PLV with exercise, the cardiac index is significantly augmented. However, there are further rises in pulmonary artery and pulmonary capillary wedge pressures, suggesting abnormal compliance, with marked decline in mixed venous oxygenation. Elucidating late physiology after PLV may help pave the way for future innovative heart failure surgeries. (*Circulation. 2000;102[suppl III]:III-200-III-203.*)

**Key Words:** exercise ▪ hemodynamics ▪ surgery ▪ ventricles

Partial left ventriculectomy (PLV), also known as the “Batista” operation, drew attention as a possible alternative surgery to cardiac transplantation in the setting of end-stage heart failure.¹ The operation involves resection of a portion of left ventricular muscle, decreasing the left ventricular diameter, and altering the cardiac geometry.

Follow-up over time has revealed varied hemodynamic and clinical outcomes in patients after PLV.² In some settings in which ejection fraction and hemodynamics remain subnormal after the surgery, patients nonetheless note clinical improvement, with 30% to 40% reporting better quality of life. In an effort to better understand the physiology of hearts late after PLV, we performed pulmonary arterial catheterization at rest and during bicycle exercise in a series of patients returning for 1- to 2-year follow-up evaluations.

**Methods**

Patients included in the present study were individuals who had undergone PLV 1 or 2 years earlier and who were returning for annual follow-up evaluations. The technique used for PLV at our institution has previously been described.² Patients were excluded if they displayed decompensated heart failure or did not give consent. All participants gave informed consent in accordance with the Institutional Review Board at our institution.

During the same follow-up visit, history and physical examinations were performed, and New York Heart Association (NYHA) functional class was ascertained. Echocardiograms were performed to assess left ventricular function. Mitral inflows to measure velocity in early diastole and velocity of atrial filling in late diastole as well as deceleration times were included.³ Treadmill cardiopulmonary stress testing to measure maximal oxygen consumption was also performed during the visit. Upright treadmill exercise was performed by using a modified Naughton protocol with increments every 2 minutes. Oxygen consumption was measured by use of a ventilatory analyzer (Sensor-medics System 4400). Anaerobic threshold was derived during the metabolic stress test by computer-assisted analysis of ventilatory end-tidal gases. At the same time, the respiratory exchange ratio was calculated as volume of CO₂ produced divided by volume of O₂ consumed, with a ratio of $>1.09$ indicating good effort.

**Pulmonary Arterial Catheterization**

The patients were asked not to eat, drink, or take their medications for 12 hours before testing. They did not receive antecedent training on the exercise bicycle. Under sterile conditions, a local anesthetic was administered to the right (or left) neck area, followed by percutaneous insertion of a venous sheath, by use of the Seldinger technique. An 8F Oximetric 3 thermocatheter was then advanced from the right atrium to the pulmonary artery, recording intracardiac pressures as the catheter traversed each chamber of the right heart. Serial cardiac outputs in triplicate were then obtained with the catheter in the pulmonary artery by use of a standard thermodilution technique with 10 mL of dextrose solution with each injection. When $>10\%$ variability was found, an additional value was obtained, and the outlier measurement was excluded. Continuous pulmonary arterial mixed venous oxygenation was measured during rest and exercise via the catheter. Continuous peripheral systemic oxygen saturation was also monitored with a digital oximeter.

**Exercise Hemodynamics**

After the indwelling thermodilution catheter was taped securely in place with the tip remaining in the pulmonary artery, the patient was
Hemodynamics in PLV Patients

<table>
<thead>
<tr>
<th>Variable</th>
<th>Before Exercise</th>
<th>With Exercise</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic PAP, mm Hg</td>
<td>52±20</td>
<td>79±17</td>
<td>0.0001</td>
</tr>
<tr>
<td>Diastolic PAP, mm Hg</td>
<td>24±9</td>
<td>35±9</td>
<td>0.004</td>
</tr>
<tr>
<td>Mean PAP, mm Hg</td>
<td>36±12</td>
<td>52±10</td>
<td>0.0003</td>
</tr>
<tr>
<td>PCWP, mm Hg</td>
<td>25±14</td>
<td>36±9</td>
<td>0.0566</td>
</tr>
<tr>
<td>Systolic BP, mm Hg</td>
<td>108±13</td>
<td>128±30</td>
<td>0.0853</td>
</tr>
<tr>
<td>Diastolic BP, mm Hg</td>
<td>64±8</td>
<td>78±27</td>
<td>0.1599</td>
</tr>
<tr>
<td>Cardiac index, L · min⁻¹ · m⁻²</td>
<td>2.2±0.5</td>
<td>3.8±1.6</td>
<td>0.0077</td>
</tr>
<tr>
<td>Heart rate, bpm</td>
<td>76±14</td>
<td>108±23</td>
<td>0.0023</td>
</tr>
<tr>
<td>MV̇O₂, %</td>
<td>44±9</td>
<td>24±17</td>
<td>0.0220</td>
</tr>
<tr>
<td>O₂ saturation, %</td>
<td>98±3</td>
<td>96±4</td>
<td>0.2644</td>
</tr>
<tr>
<td>Stroke volume, mL/m²</td>
<td>63±15</td>
<td>77±38</td>
<td>0.2270</td>
</tr>
</tbody>
</table>

Values are mean±SD. PAP indicates pulmonary arterial pressure; PCWP, pulmonary capillary wedge pressure; BP, blood pressure; MV̇O₂, mixed venous oxygenation; and PVR, pulmonary vascular resistance.

carefully assisted onto an upright bicycle exercise ergometer positioned adjacent to the fluoroscopy table. The patient exercised to limiting fatigue beginning at a workload of 20 W, which was increased by 10 W every 3 minutes. During exercise, ECG heart rate and rhythm and arterial and pulmonary arterial pressures were monitored continuously; pulmonary capillary wedge and arterial pressures were measured every minute. Cardiac output was measured in triplicate by the thermodilution technique in the final 2 minutes of the highest completed exercise stage. Systemic and pulmonary vascular resistances as well as stroke volume were calculated as previously described with computer assistance.

Analysis

Right heart resting pressures and cardiac output were compared with the same parameters measured during bicycle exercise. Paired t testing was used to perform comparisons. A value of P<0.05 was considered significant.

Results

Patient Characteristics

Ten of the patients who had undergone PLV also underwent bicycle hemodynamics between October 1997 and January 1999. The group consisted of 9 men and 1 woman with a mean age of 56±12 years. The preoperative cardiac index was 2.2±1.2 L · min⁻¹ · m⁻²; mean pulmonary arterial pressure, 40±5 mm Hg; mean pulmonary capillary wedge pressure, 24±11 mm Hg; mixed venous oxygenation, 48±16%; and pulmonary vascular resistance, 3.7±2.9 Wood units. The preexercise hemodynamics are shown in Table.

By clinical history, 1 patient was designated NYHA functional class I, 4 were class II, and 5 were class III. In terms of medications, all were on diuretics, 9 were on angiotensin-converting enzyme inhibitors, 3 were on nitrates, 1 was on other vasodilators, 7 were on amiodarone, and 9 were on a digitalis preparation.

Echocardiography

The mean left ventricular ejection fraction for the group was 25±4%, with a range of 10% to 30%. The mean end-diastolic dimension was 6.6±0.7 cm. Mean left ventricular end-diastolic volume was 276.8±88.6 mL, and end-systolic volume was 229.9±78.5 mL. The right ventricular systolic pressure was 42±8 mm Hg.

Mitrval insufficiency was graded 0 to 4+ as previously described. There was grade 0 to trivial mitral insufficiency in 5 patients and grade 1+ mitral insufficiency in the other 5 patients.

Mitral inflow evaluation of diastolic function assessing maximal velocities in early diastole and of atrial filling in late diastole and deceleration times was performed by a technique previously described. The inflows revealed pseudonormal diastolic filling in 4 patients, restrictive filling in 2, and abnormal relaxation in 1. Inflow data were not available for 3 patients. Deceleration times lengthened, with a mean preoperative deceleration time of 120±20 ms and postoperative time of 188±56 ms (P=0.0115).

Cardiopulmonary Stress Testing

Treadmill cardiopulmonary stress tests were performed in all of the patients. Mean metabolic equivalent expenditure was 5±2. The mean maximal oxygen consumption for the group was 16.6±4.1 mL · kg⁻¹ · min⁻¹, with a range of 10.7 to 22.4 mL · kg⁻¹ · min⁻¹. Mean anaerobic threshold was 10.7±22.4 mL · kg⁻¹ · min⁻¹. The mean respiratory exchange ratio for the group was 1.2±0.1.

Bicycle Hemodynamics

The duration to peak exercise for the group was 10±7 minutes. The Table and Figures 1 through 4 depict the hemodynamic results. Of note, the resting systolic pulmonary arterial pressure was moderately elevated at 52±20 mm Hg and rose markedly to 79±17 mm Hg at peak exercise (P=0.0001). The mean pulmonary capillary wedge pressure was also elevated at rest (-25±14 mm Hg) and rose to 36±9 mm Hg at peak exertion (P=0.0566). The cardiac index was augmented 1.7-fold with exercise, increasing from 2.2±0.5 to 3.8±1.6 L · min⁻¹ · m⁻², with a decline in pulmonary arterial mixed venous oxygenation from 44±9% at rest to 24±17% with exercise.

Discussion

PLV is a procedure performed on cardiomyopathic hearts, in which a section of left ventricular muscle is excised from end-stage dilated hearts. In removing the muscle, the ven-
tricular diameter is decreased, thereby lowering the wall tension according to the law of Laplace. There is functional improvement, with 60% of patients improving from NYHA functional class III or IV to class I or II. Preliminary data have revealed no significant changes in resting hemodynamics in patients undergoing PLV, with no difference in cardiac index at baseline compared with 3 months after surgery (2.2 ± 0.5 versus 2.1 ± 0.6 L · min⁻¹ · m⁻²).

Resting hemodynamics alone, however, do not provide a complete picture of the status of a heart failure patient. Exercise tolerance is also an important functional aspect in heart failure. Unfortunately, resting hemodynamics do not correlate well with exercise capacity as measured by treadmill maximal oxygen consumption. Exercise hemodynamics, on the other hand, provide additional physiological and prognostic information.

Griffin et al⁸ reported that exercise hemodynamic evaluation in patients with chronic congestive heart failure provides important prognostic information. They studied 49 heart failure patients, with a 1-year follow-up. By multiple logistic regression analysis, they found that pulmonary arterial wedge pressures at rest and peak exercise stroke work index were independent predictors of mortality. Receiver-operating characteristic curve analysis revealed that the peak-exercise stroke work index provided significant incremental prognostic information compared with the information provided by resting hemodynamic variables.

Roul et al⁹ described the exercise hemodynamic results in patients with congestive heart failure. In a group of patients with favorable outcome (alive without major events), mean pulmonary arterial pressure rose from 29 ± 1.2 to 46 ± 1.3 mm Hg with exercise; mean pulmonary capillary wedge pressure, from 11 ± 0.5 to 25 ± 0.7 mm Hg; mean cardiac index, from 2.45 ± 0.05 to 5.9 ± 0.2 L · min⁻¹ · m⁻²; and mean pulmonary vascular resistance, from 528 ± 29 to 356 ± 21 dynes · cm⁻². In heart failure patients with poor outcome (death or major events), mean pulmonary arterial pressure rose from 37 ± 1 to 54 ± 1.5 mm Hg with exercise; mean pulmonary capillary wedge pressure, from 15 ± 0.7 to 31 ± 1 mm Hg; mean cardiac index, from 2.3 ± 0.11 to 3.0 ± 0.2 L · min⁻¹ · m⁻²; and mean pulmonary vascular resistance, from 706 ± 37 to 813 ± 57 dynes · cm⁻². Roul et al concluded that exercise hemodynamics in heart failure are useful in assessing prognosis at the 21-month follow-up.

Bicycle exercise hemodynamics have also been performed in healthy patients to define the normal hemodynamic response to exertion. Thadani and Parker¹⁰ reported that in 10 healthy patients, at supine rest mean pulmonary arterial pressure was 13 ± 1 mm Hg, with a pulmonary capillary wedge pressure of 6 ± 1 mm Hg. These pressures rose to a mean pulmonary arterial pressure of 22 ± 1 mm Hg and pulmonary capillary wedge pressure of 6 ± 1 mm Hg with bicycle exercise. The cardiac index rose from 3.5 ± 0.3 to 7.3 ± 0.5 L · min⁻¹ · m⁻². Higginbotham et al¹¹ also described the normal hemodynamic response to upright bicycle exercise in humans. In his series, the pulmonary arterial systolic blood pressure upright at rest was 15 ± 4 mm Hg and rose to 34 ± 6 mm Hg at peak exertion. The cardiac index increased 3.2-fold, from 3.0 to 9.7 L · min⁻¹ · m⁻².

Our exercise hemodynamic studies in patients with cardiomyopathy who had undergone PLV 1 to 2 years earlier yield multiple observations. The resting pulmonary arterial pressures remain moderately elevated and rise markedly with exercise. The mean pulmonary capillary wedge pressure is elevated at rest, indicating some hypervolemia, but rises markedly with exertion, suggesting abnormal compliance as well.
The mean pulmonary arterial mixed venous oxygenation at rest is subnormal and falls markedly with exercise, indicating increased oxygen extraction due to the cardiac output inadequately meeting the peripheral circulatory demands. Although the mean cardiac index rose with exertion in our patients, it nonetheless remained subnormal at a 1.7-fold increase.

Contrasted with the hemodynamic findings, at last half of the patients studied reported clinical improvement in functional class and well-being since the PLV surgery, in keeping with earlier published data. Objectively, the cardiopulmonary stress tests revealed maximal peak oxygen consumption of $16.6 \pm 4.1 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ in our patients.

There are several limitations to the present study. Simultaneous measurements of peak oxygen consumption during bicycle exercise in the catheterization laboratory would have been ideal. The cardiopulmonary stress tests were performed separately from the bicycle catheterization but within temporal proximity (within 24 hours).

Small sample size is a limitation, although all surviving nontransplanted PLV patients who returned for late follow-up were offered participation. There were no refusals, with exclusion only of patients too ill to exercise.

Also, we do not have preoperative exercise hemodynamic data. However, we do have data by Roul et al and others to serve as historical controls for exercise hemodynamics in unoperated heart failure patients.

Last, diastolic function remains an important area of assessment in heart failure and after PLV. Resting echocardiographic assessment of diastolic function revealed a shift from restrictive filling in 8 of 9 patients before surgery to pseudonormalization in 4 of 7 after PLV. This was accompanied by a lengthening of deceleration time. Echocardiographically, diastolic function appeared to improve somewhat after PLV, although it remained abnormal. We do not have invasive assessment of diastolic function before or after surgery beyond right ventricular waveforms, which excluded overt constrictive/restrictive filling in all 10 patients. The present study was designed to assess exercise hemodynamics rather than specific diastolic data. However, the postoperative exercise data do suggest that abnormal compliance superimposed on elevated resting filling pressures may be present in these patients.

It is helpful to look at the overall clinical and survival data on PLV as a background to the present study, as summarized by Starling et al at our institution recently. At the 26-month follow-up, 62 patients had undergone PLV. Of this group, 11 of the 62 patients were bridged to transplant with a left ventricular assist device. At 24 months, the left ventricular ejection fraction increased from $13 \pm 6\%$ to $21 \pm 7\%$, peak oxygen consumption rose from $11 \pm 4$ to $16 \pm 4 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$, and the left ventricular diastolic diameter decreased from $8 \pm 1$ to $6.5 \pm 0.87 \text{ cm}$. Starling et al reported the actuarial survival at 12, 24, and 30 months as $80\%$, $71\%$, and $68\%$, respectively, with freedom from death, need for transplantation or assist device, or NYHA functional class IV as $49\%$, $36\%$, and $29\%$ at 12, 24, and 30 months, respectively.

Regarding our hemodynamics in the above context, our exercise data likely represent the healthier survivors of PLV. Our data do not help in the preoperative selection of PLV candidates because this is a postoperative study. As noted by Starling et al, although $29\%$ of PLV patients realized some clinical benefit, the failure rate is high, and PLV does not provide benefit to equivalent to transplantation.

Conclusions
In late follow-up after PLV, cardiac output is augmented with exertion during exercise. However, there is a marked rise in pulmonary arterial and ventricular filling pressures with exercise, indicating that underlying derangements of cardiac performance persist despite surgical partial restoration of ventricular geometry. These data also indicate a need for more aggressive diuretic and vasodilator use in PLV patients. Irrespective of the future role of PLV, it is important that we look back and try to better understand the physiological reasons for both long-term benefits and persisting abnormalities in the recipients of this novel procedure to pave the way for future innovations in heart failure surgery.

References
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