PULMONARY HEMODYNAMIC CHANGES IN THE FIRST 24 HOURS AFTER MITRAL VALVE REPLACEMENT IN PATIENTS WITH PULMONARY HYPERTENSION

Samir Maher, MS, MD, FRACS

From January 1992 until May 1994, 25 patients with mitral valve disease and pulmonary arterial hypertension (pulmonary artery systolic pressure > 40 mm Hg) underwent mitral valve replacement at King Khalid University Hospital. There were 15 females and 10 males with a mean age of 37 years (range, 13 to 65 years). Seven patients had mitral regurgitation (MR), 7 had mitral stenosis (MS), and 11 had combined lesions (MR + MS). Two patients were in class II, New York Heart Association (NYHA), 15 were in class III, and 8 in class IV. Hemodynamic measurements recorded before operation and in the first 24 h after surgery showed the following significant changes: mean pulmonary artery pressure (PAP), 45 ± 11 to 22 ± 5 mm Hg; pulmonary artery wedge pressure (PAWP), 27 ± 7 to 14 ± 2 mm Hg; pulmonary vascular resistance (PVR), 407 ± 195 to 166 ± 52 dyne.sec.cm⁻²; cardiac output (CO), 3.4 ± 0.8 to 4.2 ± 0.8 L/min; and cardiac index (CI), 2.1 ± 0.3 to 2.6 ± 0.4 L/min/m² (P value 0.05).

The change in percentage was 51%, 48%, 59%, 21%, and 19%, respectively. Despite the decline of PVR, the return to normal values postoperatively was not seen in all of the cases. The nature of the lesion, either MR, MS or MR + MS, had no impact on the postoperative hemodynamic changes. All patients survived the operation.

PULMONARY HYPERTENSION frequently accompanies mitral valve disease that is sufficiently severe to warrant surgical intervention. 1,2 The increase in pulmonary arterial pressure has been attributed to (1) passive transmission of high left atrial pressure across the pulmonary bed, (2) obliterative changes in the pulmonary vascular bed, and (3) pulmonary vasoconstriction in response to increased left atrial pressure (reactive pulmonary hypertension). 3 The cumulative effect of (2) and (3) leads to increased pulmonary vascular resistance.

To differentiate between the dynamic vasoconstrictive and obliterative changes responsible for pulmonary hypertension, injection of acetylcholine in the pulmonary artery has been attempted. 4 If the fall in the pulmonary artery pressure was less than 15 mm Hg, it was thought that the pulmonary vascular resistance had probably become fixed. 5

In the early days of cardiac surgery, pulmonary hypertension was considered to be a formidable factor adversely affecting the early and late results of mitral valve surgery. 5-8 With improvement in surgical and perfusion techniques and postoperative care, it became a common practice to operate on patients with pulmonary hypertension with acceptable mortality. 9-13 Many reports have demonstrated significant reduction of pulmonary hypertension and pulmonary vascular resistance when cases were reinvestigated several months postoperatively. 12-15

Few reports have shown the immediate hemodynamic changes following mitral valve surgery in patients with pulmonary hypertension. 15,16 The aim of the work in this study is to assess the...
immediate hemodynamic changes after mitral valve replacement in patients with pulmonary hypertension.

Patients and Methods

From January 1990 until May 1994, 25 patients presented with pulmonary hypertension (pulmonary artery systolic pressure above 40 mm Hg) secondary to mitral valve disease underwent mitral valve replacement in King Khalid University Hospital.

The series comprised of 15 females and 10 males with a mean age of 37 years (range, 13 to 65 years). Based on history, operative findings, and histopathologic examination, 22 patients were rheumatic in origin and 3 patients had degenerative mitral valve disease. The diagnosis of mitral valve disease was entertained by means of clinical examination, echocardiogram, and cardiac catheterization in all patients. Seven patients had isolated mitral stenosis, 7 had isolated mitral regurgitation, and 11 patients had combined lesions. Significant tricuspid incompetence that necessitated reparative procedures was found in 6 patients.

Preoperatively, 2 patients were in class II New York Heart Association (NYHA) (8%), 15 patients in class III (72%) and 8 patients in class IV (20%). Table 1 presents the clinical and hemodynamic data of the patient population.

Table 1. Preoperative clinical and hemodynamic data of 25 patients who underwent mitral valve replacement.

<table>
<thead>
<tr>
<th>Age</th>
<th>Mean 27 years (range, 13 to 65 years)</th>
<th>10</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td>males and 15 females</td>
<td></td>
</tr>
<tr>
<td>Type of lesion</td>
<td>MS (7), MR (7), MS + MR (2)</td>
<td></td>
</tr>
<tr>
<td>NYHA class</td>
<td>II, (15) class III, (8) class IV</td>
<td></td>
</tr>
<tr>
<td>Significant TR</td>
<td>6 cases</td>
<td></td>
</tr>
<tr>
<td>PAP mm Hg</td>
<td>mean, 45±11 (range, 27-75) mean, 27±11</td>
<td></td>
</tr>
<tr>
<td>PAWP mm Hg</td>
<td>7 (range: 17-46) mean, 407±195</td>
<td></td>
</tr>
<tr>
<td>PVR mm Hg</td>
<td>(range: 98-725) mean, 3.4±0.8 (range)</td>
<td></td>
</tr>
<tr>
<td>CO mm Hg</td>
<td>2.5±2.1 (range: 1.5-3)</td>
<td></td>
</tr>
<tr>
<td>CI</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

PAP mm Hg = mean pulmonary artery pressure; PA WP mm Hg = pulmonary artery wedge pressure; PVR mm Hg = pulmonary vascular resistance; CO = cardiac output (U/min); CI = cardiac index (U/min/m²); TR = tricuspid regurgitation.

All patients with isolated mitral valve disease and those with tricuspid incompetence secondary to mitral valve disease were included. Patients with associated aortic valve disease, coronary artery disease, or previous mitral valve replacement requiring second operation were excluded from the study.

Operative Technique

Under general anesthesia, arterial and central venous pressures were monitored. Swan Ganz thermodilution catheter was introduced through the right internal jugular vein.

Through median sternotomy, cardiopulmonary bypass was conducted after systemic heparinization by cannulating the ascending aorta and venous drainage by 2 caval cannulae with systemic cooling to 28°C. Myocardial protection was achieved by cold hyperkalemic crystalloid cardioplegia and topical cooling. No left ventricular vent was used. The standard approach for mitral valve replacement via left atriotomy lateral to the interatrial groove was adopted. After excision of the anterior cusp, the posterior cusp was left intact in most of the cases, the mitral valve was replaced by 2-0 Ticron interrupted everting sutures with pledgets.

Mechanical bileaflet prosthetic valve was inserted in 22 patients (13 carbomedics, 9 St. Jude), and 3 patients received bioprosthesis valve (Carpentier-Edwards). In 6 patients who had significant tricuspid incompetence, two had Carpentier-Edwards ring and four had De Vega’s tricuspid annuloplasty. After left atrial closure, deairation and rewarming, the aortic clamp was removed and the heart defibrillated. Following that, the patients were weaned from cardiopulmonary bypass and heparin was reversed. After adequate hemostasis and closure in layers with two retrosternal drains and two pacing wires, the patients were transferred to the surgical intensive care unit. Vasodilators were not used in any of these patients, and dopamine was given in renal dose of 5 ILg/kg or less that was tapered and discontinued over 24 h.

Hemodynamic Study

Preoperatively all patients underwent cardiac catheterization and the hemodynamics were analyzed. Cardiac output was estimated by the
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direct Fick principle and divided by the surface area to calculate the cardiac index (L/min/m²). Pulmonary vascular resistance was calculated as follows:

\[ \text{PVR} = \frac{\text{PAP-PAWP}}{\text{CO}} \times 80 \text{ (dynes. sec. cm}^{-5}) \]

PVR = pulmonary vascular resistance; PAP = mean pulmonary artery pressure (mm Hg); PAWP = pulmonary artery wedge pressure (mm Hg); CO = cardiac output (L/min).

Pulmonary hypertension was considered when systolic pulmonary artery pressure was > 40 mm Hg and raised pulmonary vascular resistance if it was > 125 dynes.sec.cm⁻⁵.

The immediate postoperative hemodynamics were measured by Swan Ganz catheter positioned in the pulmonary artery. Cardiac output was measured by the thermodilution technique. Postoperative measurements, including mean pulmonary artery pressure, pulmonary artery wedge pressure, cardiac output, pulmonary vascular resistance, and cardiac index, were recorded immediately after arrival at the surgical intensive care unit and thereafter 6 hourly for 24 h. This was followed by removal of the Swan Ganz catheter.

Statistics

Pair-wise t-test was used to compare the hemodynamics preoperatively with those measured postoperatively over 24 h, expressed as a mean of the 5 postoperative readings.

One-way analysis of variance (ANOVA) was used to evaluate the hemodynamic measurements postoperatively over 24 h. The same test was used to compare the preoperative readings with each of the 5 postoperative measurements. One-way analysis of variance was used to evaluate the impact of the type of the lesion (mitral incompetence, mitral stenosis, or combined) on hemodynamic measurements.

P value of less than 0.05 was considered statistically significant.

Results

This retrospective study was comprised of 10 male and 15 female patients with a mean age of 37 years (range, 13 to 65 years). Two patients were in NYHA class II, 15 in class III, and 8 in class IV. Seven patients had mitral stenosis and 11 patients had mixed lesions. There were 6 patients with significant tricuspid incompetence, four had De Vega tricuspid annuloplasty, and two received tricuspid Carpenter-Edwards rings. All patients survived the operation.

Table 2 as well as Figures 1 to 5 show the preoperative and postoperative (immediately at 6, 12, 18, and 24 h) measurements of mean pulmonary artery pressure (PAP), pulmonary artery wedge pressure (PAWP), pulmonary vascular resistance (PVR), cardiac output (CO), and cardiac index (CI). There was a significant decrease of PAP from a mean of 45±11 mm Hg (range, 27 to 75 mm Hg) preoperatively to a mean of 22±5 mm Hg (range, 14 to 36 mm Hg) over 24 h postoperatively (P value < 0.05). The percentage of change was 51%.

PAWP showed a significant drop from a mean

Table 2. Preoperative and postoperative (in the first 24 h) hemodynamic measurements.

<table>
<thead>
<tr>
<th>POST-OP</th>
<th>PRE-OP</th>
<th>Immed</th>
<th>6h</th>
<th>12h</th>
<th>24h</th>
<th>Mean</th>
<th>% of change</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>PAP</td>
<td>45±11</td>
<td>22±5</td>
<td>23±6</td>
<td>22±5</td>
<td>23±5</td>
<td>22±5</td>
<td>22±5</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>PAWP</td>
<td>27±7</td>
<td>14±2</td>
<td>15±3</td>
<td>14±2</td>
<td>14±2</td>
<td>14±2</td>
<td>14±2</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>PVR</td>
<td>407±195</td>
<td>162±92</td>
<td>166±106</td>
<td>164±78</td>
<td>174±92</td>
<td>167±94</td>
<td>166±52</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>CO</td>
<td>3.4±0.5</td>
<td>4.3±0.9</td>
<td>4.3±0.5</td>
<td>4.3±0.8</td>
<td>4.3±0.5</td>
<td>4.2±0.9</td>
<td>4.2±0.5</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>CI</td>
<td>2.1±0.35</td>
<td>2.6±0.41</td>
<td>2.6±0.41</td>
<td>2.6±0.47</td>
<td>2.6±0.41</td>
<td>2.6±0.44</td>
<td>2.6±0.4</td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>

PAP mm Hg = mean pulmonary artery pressure; PAWP (mm Hg) = pulmonary artery wedge pressure; PVR (dyne. sec. cm⁻⁵) = pulmonary vascular resistance; CO (L/min) = cardiac output CI (L/min/m²) = cardiac index.

of 27 ± 7 mm Hg (range, 17 to 46 mm Hg) before operation to a mean of 14 ± 2 mm Hg (range, 10 to 20 mm Hg) after operation over 24 h. The percentage of change was 48% (P value < 0.05).

PVR decreased from a mean of 407 ± 195 dyne.sec.cm⁻⁵ (range, 98 to 725 dyne.sec.cm⁻⁵) preoperatively to a mean of 166 ± 52 dyne.sec.cm⁻⁵ (range, 58 to 253 dyne.sec.cm⁻⁵) postoperatively over 24 h. The percentage of change was 59% (P value < 0.05).

CO showed an increase from a mean of 3.4 ± 0.8 L/min (range, 1.6 to 4.8 L/min) preoperatively to a mean of 4.2 ± 0.8 L/min (range, 2 to 5.7 L/min) postoperatively over 24 h. The percentage of change was 21% (P value < 0.05).

There was an increase of CI from a mean of 2.1 ± 0.3 L/min/m² (range, 1.5 to 3 L/min/m²) preoperatively to a mean of 2.6 ± 0.4 L/min/m² (range, 1.8 to 3.6 L/min/m²) postoperatively over 24 h. The percentage of change was 19% (P value < 0.05).

One-way analysis of variance test (ANOVA) did not show any statistically significant difference (P value > 0.05) when the postoperative measurements at different intervals were analyzed for each parameter (PAP, PAWP, PVR, CO, CI).

When the preoperative readings of PAP, PAWP, PVR, CO, CI were compared to the postoperative measurement at each interval, i.e., immediately and after 6, 12, 18 and 24 h, the ANOVA test showed significant statistical difference (P value < 0.05). The impact of the type of lesion (mitral incompetence, mitral...
Table 3. Preoperative and postoperative (in the first 24 h) measurements of patients with mitral regurgitation, mitral stenosis, and combined lesions.

<table>
<thead>
<tr>
<th>Lesion</th>
<th>No.</th>
<th>PAP (mm Hg)</th>
<th>PAWP (mm Hg)</th>
<th>PVR (dynes sec cm⁻²)</th>
<th>CO (L/min)</th>
<th>CI (L/min m²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>MR</td>
<td>7</td>
<td>39±5</td>
<td>21±5</td>
<td>34±3</td>
<td>14±3</td>
<td>234±133</td>
</tr>
<tr>
<td></td>
<td></td>
<td>49±15</td>
<td>24±17</td>
<td>49±3</td>
<td>14±3</td>
<td>373±133</td>
</tr>
<tr>
<td>MS</td>
<td>7</td>
<td>49±15</td>
<td>24±17</td>
<td>49±3</td>
<td>14±3</td>
<td>373±133</td>
</tr>
<tr>
<td>MR + MS</td>
<td>11</td>
<td>46±10</td>
<td>23±3</td>
<td>46±10</td>
<td>14±3</td>
<td>46±10</td>
</tr>
</tbody>
</table>

MR = mitral regurgitation; MS = mitral stenosis; PAP (mm Hg) = mean pulmonary artery pressure; PAWP (mm Hg) = pulmonary artery wedge pressure; PVR (dynes sec cm⁻²) = pulmonary vascular resistance; CO (L/min) = cardiac output; CI (L/min m²) = cardiac index.

Postoperative readings are the mean of all measurements over 24 h.

The late effects of mitral valve replacement on pulmonary vascular dynamics have been reported by several investigators. The hemodynamic changes were similar in the three groups (mitral regurgitation, mitral stenosis, and combined lesions).

One-way analysis of variance of postoperative measurements did not show statistically significant difference (P value > 0.05) among the patients with MR, MS, or MR + MS.

Discussion

The late effects of mitral valve replacement on pulmonary vascular dynamics have been reported by several investigators. In this study 25 patients with pulmonary arterial hypertension due to mitral valve disease underwent mitral valve replacement. Hemodynamic measurements in the first 24 h following operation showed significant drops of PAP and PAWP. In spite of the fact that there was a significant decrease of PVR, the decline to normal values was not confined to all patients. Both CO and CI showed significant increases. These changes were unique to the whole series, irrespective of the nature of the lesion, i.e., mitral stenosis, mitral regurgitation, or combined lesions. Pharmacologic manipulation of the hemodynamic changes was ruled out where vasodilators and inotropes were not used (renal dose of dopamine < 5 JL/kg).

Pulmonary arterial hypertension secondary to mitral valve disease is attributed to (1) passive transmission of high left atrial pressure across the pulmonary bed which is eliminated by mitral valve replacement, (2) obliterator morphologic changes in the pulmonary vascular bed, and (3) pulmonary vasoconstriction due to increased left atrial pressure (dynamic or reactive component of pulmonary hypertension).

Both obliterative and reactive components of pulmonary hypertension lead to raised pulmonary vascular resistance. In this study PVR showed significant decline in the first 24 h after operation, however, the drop of PVR to normal values was not seen in all patients. This means that mitral valve replacement by virtue of lowering left atrial pressure, immediately after operation, causes the drop of PVR by eliminating the reactive component of pulmonary hypertension. The return of PVR to normal value after mitral valve replacement needs the obliterative changes of the pulmonary vasculature to be abolished which may take place over an extended period of time.

In an attempt to evaluate the immediate hemodynamic changes after mitral valve replacement in patients with pulmonary hypertension, McIlduff et al. observed the hemodynamics 3 h after mitral valve replacement. Probably, this period is too short to elaborate the hemodynamic changes.

Austen et al. reported the immediate hemodynamics for 48 h following mitral valve replacement with Starr-Edwards prostheses and closed mitral valvotomy.

In this series, there was a significant drop of left atrial pressure and pulmonary artery pressure but pulmonary vascular resistance remained high. There were a few patients who demonstrated a decrease in pulmonary vascular resistance.

Foltz et al. studied the early course of
pulmonary arterial hypertension after mitral valve replacement for 36 h with special emphasis on the immediate changes (first 4 h after operation). Patients with pulmonary hypertension and elevated preoperative pulmonary vascular resistance index showed marked reduction in pulmonary artery pressure and pulmonary vascular resistance 0 to 4 h after operation.

Several investigators studied the immediate effects of percutaneous mitral valvuloplasty in patients with mitral stenosis and pulmonary hypertension. The hemodynamic changes following PMV were similar to the abovementioned reports.

**Conclusion**

In an attempt to evaluate the early hemodynamic changes after mitral valve replacement in patients with pulmonary hypertension (pulmonary artery systolic pressure > 40 mm Hg), 25 patients were studied in the first 24 h following surgery.

Postoperative hemodynamic measurements showed significant reductions of mean pulmonary artery pressure and pulmonary artery wedge pressure compared with preoperative readings. There was a significant drop of pulmonary vascular resistance, however, not all patients returned to normal values. There were significant improvements of both cardiac output and cardiac index compared with preoperative measurements. Mitral valve replacement in patients with pulmonary hypertension has an immediate hemodynamic effect leading to decreases in pulmonary artery pressure and pulmonary artery wedge pressure. Persistence of elevated pulmonary vascular resistance in some patients may be due toobliterative morphologic changes of the pulmonary vasculature.

Mitral valve replacement has gratifying early and late results in patients with pulmonary hypertension.

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**References**


