Hemodynamic Changes Following Correction of Severe Aortic Stenosis Using the Cutter-Smeloff Prosthesis

By Simon J. K. Lee, M.D., M. Haraphongse, M.D., J. C. Callaghan, M.D., R. E. Rossall, M.D., and R. S. Fraser, M.D.

SUMMARY

Twelve patients with pure or dominant stenosis were studied before and after aortic valve replacement (Cutter-Smeloff). The changes in cardiac output and A-V O₂ difference were small and insignificant, but exercise stroke volume increased from 72 to 96 ml after the surgery. The aortic peak systolic gradient was 92 ± 29 mm Hg before and 4 ± 9.8 mm Hg after surgery.

The PA wedge pressure, taken as a measure of the LV diastolic pressure, decreased from 18 mm Hg to 12 mm Hg at rest and 30 mm Hg to 16 mm Hg on exercise. The PA pressures also decreased from 37/18 (mean, 26) to 28/11 (17) mm Hg at rest, and 61/31 (43) to 41/17 (27) mm Hg on exercise, but the PA and PA wedge pressures maintained their tendency to increase in a linear manner with oxygen consumption (VO₂) on exercise after surgery. As a result of decreased wedge pressure, the pulmonary vascular resistance also decreased significantly afterward. The adequacy of cardiac output at rest and on exercise expressed by cardiac output as the linear function of VO₂ improved toward the normal (values before operation, CO = 2.78 + 0.0069 VO₂, r = 0.84; after operation, CO = 3.80 + 0.0062 VO₂, r = 0.91). The exercise factor (ΔCO/ΔVO₂) or the regression coefficient, however, was similar before and after surgery. The hemodynamic result was satisfactory although some abnormalities of the left ventricular function persisted after operation.

Additional Indexing Words:

Left ventricular function  Exercise  Exercise factor

HEMODYNAMIC studies have been reported following replacement of the aortic valve using the Starr-Edwards prosthesis,¹⁻³ and various parameters of cardiac function have been found to improve although significant abnormalities may persist in some patients. To increase the effective lateral orifice of the ball-valve prosthesis, Cartwright and associates⁴ made modifications to the original prosthesis and these modifications have been widely accepted.⁵ The purpose of this communication, therefore, is to report circulatory changes at rest and exercise before and after aortic valve replacement using the Cutter-Smeloff prosthesis. Only patients with pure or dominant aortic stenosis were included in this study to assess the result of the decreasing pressure load of the left ventricle rather than the volume load as in aortic regurgitation. Of special interest was a study of the circulatory adaptation to strenuous exercise after surgery.
Clinical Summary

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Age (yr): sex</th>
<th>Symptoms</th>
<th>Preop.</th>
<th>PO</th>
<th>ECG Preop.</th>
<th>PO</th>
<th>Valve</th>
<th>Follow-up (mo)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. F.B.</td>
<td>35 M</td>
<td>Dyspnea, PND, syncope</td>
<td>No</td>
<td></td>
<td>Increased voltage</td>
<td>Same</td>
<td>Cutter, #5</td>
<td>22</td>
</tr>
<tr>
<td>2. A.D.</td>
<td>43 M</td>
<td>Dyspnea, angina</td>
<td>No</td>
<td></td>
<td>LVH, strain</td>
<td>Improved</td>
<td>Cutter, #3</td>
<td>6</td>
</tr>
<tr>
<td>3. J.W.</td>
<td>52 M</td>
<td>Syncope, angina</td>
<td>No</td>
<td></td>
<td>LVH, strain</td>
<td>Improved</td>
<td>Cutter, #4</td>
<td>23</td>
</tr>
<tr>
<td>4. G.D.</td>
<td>59 M</td>
<td>Angina, dyspnea, fatigue</td>
<td>No</td>
<td></td>
<td>LVH</td>
<td>Same</td>
<td>Cutter, #3</td>
<td>7</td>
</tr>
<tr>
<td>5. D.M.</td>
<td>52 M</td>
<td>Angina, dyspnea</td>
<td>No</td>
<td></td>
<td>LVH, strain</td>
<td>Improved</td>
<td>Cutter, #3</td>
<td>11</td>
</tr>
<tr>
<td>6. J.P.</td>
<td>53 M</td>
<td>Syncope</td>
<td>No</td>
<td></td>
<td>LVH, strain</td>
<td>Same</td>
<td>Cutter, #4</td>
<td>15</td>
</tr>
<tr>
<td>7. P.L.</td>
<td>54 F</td>
<td>Angina, syncope</td>
<td>No</td>
<td></td>
<td>LVH, strain</td>
<td>Improved</td>
<td>Cutter, #2</td>
<td>15</td>
</tr>
<tr>
<td>8. L.T.</td>
<td>40 M</td>
<td>Dyspnea, CHF</td>
<td>No</td>
<td></td>
<td>LVH, strain</td>
<td>Improved</td>
<td>Cutter, #4</td>
<td>10</td>
</tr>
<tr>
<td>9. H.C.</td>
<td>65 M</td>
<td>CHF, PND</td>
<td>Dyspnea</td>
<td></td>
<td>LVH, strain</td>
<td>Improved</td>
<td>Cutter, #6</td>
<td>7</td>
</tr>
<tr>
<td>10. G.A.</td>
<td>26 M</td>
<td>Angina, dyspnea, fatigue, dizziness</td>
<td>No</td>
<td></td>
<td>LVH, strain</td>
<td>LBBB</td>
<td>Cutter, #5</td>
<td>12</td>
</tr>
<tr>
<td>11. G.W.</td>
<td>44 M</td>
<td>Syncope, CHF</td>
<td>No</td>
<td></td>
<td>LVH, strain</td>
<td>Same</td>
<td>Cutter, #2</td>
<td>11</td>
</tr>
<tr>
<td>12. F.M.</td>
<td>40 M</td>
<td>Dyspnea, angina</td>
<td>No</td>
<td></td>
<td>LVH, strain</td>
<td>Normal</td>
<td>Cutter, #2</td>
<td>17</td>
</tr>
</tbody>
</table>

Abbreviation: PND = paroxysmal nocturnal dyspnea.

Methods

The clinical summary is listed in table 1. All 12 patients had clinical and hemodynamic findings consistent with severe valvular aortic stenosis. Patients with dominant aortic regurgitation or coexisting mitral valve disease or patients who developed significant aortic regurgitation after surgery were excluded from this series. All patients were in sinus rhythm and no patient had electrocardiographic evidence of myocardial infarction before or after operation. The postoperative studies were done between 6 and 22 mo after surgery (average 13 mo). All patients except one (case 7) were female, and their average age was 47 years (range, 26 to 65).

Technics

Cardiac catheterization was performed after a light breakfast following premedication with secobarbital (Seconal) 100 mg by mouth. For the left heart catheterization a no. 7 NIH or no. 7.5 Sones catheter was introduced into the right brachial artery via cutdown, and a no. 6 Cournand catheter was used for the venous catheterization. The aortic pressure gradient was measured by withdrawing the catheter from the left ventricle into the aorta while the pressure was continuously recorded. The midthoracic level was taken as the reference point for zero pressure. Oxygen uptake was estimated by collecting the expired gas in Douglas bags with analysis of gas for oxygen (E2, Beckman O2 analyzer) and CO2 (Capnograph-Goddart). Cardiac output was estimated using the Fick principle, and the arterial and pulmonary artery blood samples were analyzed for oxygen by the method of Van Slyke and Neill. Supine leg exercise was performed using a constant load bicycle ergometer (Fleisch), and measurements of cardiac output and pressures were made during the fourth and sixth minute of steady-state exercise. The second exercise with a higher workload was carried out after several minutes of rest. Pressure was recorded using P23Db transducers and a photographic recorder (DR-8, Electronics for Medicine). The pulmonary vascular resistance index was expressed as the ratio of the pressure difference across the pulmonary vascular bed in mm Hg (mean PA pressure minus pulmonary wedge pressure) over the cardiac output in L/min. The total pulmonary vascular resistance

Circulation, Volume XLII, October 1970
HEMODYNAMIC CHANGES OF AORTIC STENOSIS

Pulmonary artery pressures at rest and on exercise before and after surgery. Clear bar = preoperative; crossed bar = postoperative.

Results

All patients complained of one or more of the following: dyspnea, chest pain on exertion, or syncope before operation (table 1). After surgery, eight patients were entirely symptom-free while four had only mild residual symptoms including dyspnea, dizziness, or fatigue. Before operation, 11 patients had electrocardiographic evidence of left ventricular hypertrophy with increased QRS voltage and ST and T-wave changes, and one patient had only increased QRS voltage. The electrocardiograms improved definitely in seven patients; they were unchanged in four, and left bundle-branch block appeared after surgery in one.

The hemodynamic data obtained are listed in table 2, and the paired comparison of the data at rest and comparable levels of exercise is made in table 3 and figures 1 and 2.

Pressures

Resting pulmonary artery pressure (37/18; mean, 26 mm Hg) and mean PA wedge pressure (18 mm Hg) were slightly elevated before operation. In the absence of mitral valve disease, the increase in wedge pressure is due to the elevated left ventricular end-diastolic pressure (LVEDP) which was 23 ± 10 mm Hg. Following surgery, the pulmonary artery (28/11; mean, 17 mm Hg) and the wedge pressures (12 mm Hg) had...
### Hemodynamic Data

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Time</th>
<th>Exercise</th>
<th>$\dot{V}_{O_2}$ (ml/min)</th>
<th>CO (L/min)</th>
<th>HR (beats/min)</th>
<th>PA</th>
<th>PAW</th>
<th>Ao</th>
<th>LV</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Preop</td>
<td>R</td>
<td>147</td>
<td>2.82</td>
<td>72</td>
<td>22/7 (11)</td>
<td>7</td>
<td>86/53 (68)</td>
<td>150/6</td>
</tr>
<tr>
<td></td>
<td>Postop</td>
<td>R</td>
<td>145</td>
<td>2.96</td>
<td>62</td>
<td>21/7 (13)</td>
<td>7</td>
<td>105/54 (75)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>E</td>
<td>537</td>
<td>5.44</td>
<td>102</td>
<td>30/11 (19)</td>
<td>10</td>
<td>144/81 (105)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>E</td>
<td>1233</td>
<td>9.15</td>
<td>130</td>
<td>32/10 (20)</td>
<td>10</td>
<td>141/73 (105)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>E</td>
<td>118</td>
<td>40/21 (26)</td>
<td>16</td>
<td>115/80 (102)</td>
<td></td>
<td>246/23</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>E</td>
<td>986</td>
<td>11.38</td>
<td>150</td>
<td>50/31 (39)</td>
<td>24</td>
<td>115/74 (95)</td>
<td>240/41</td>
</tr>
<tr>
<td></td>
<td></td>
<td>E</td>
<td>323</td>
<td>5.66</td>
<td>90</td>
<td>33/8 (14)</td>
<td>8</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>E</td>
<td>912</td>
<td>10.18</td>
<td>104</td>
<td>35/15 (21)</td>
<td>12</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>E</td>
<td>1355</td>
<td>11.46</td>
<td>132</td>
<td>43/19 (25)</td>
<td>15</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Preop</td>
<td>R</td>
<td>227</td>
<td>5.1</td>
<td>93</td>
<td>25/11 (18)</td>
<td>11</td>
<td>110/73 (92)</td>
<td>229/20</td>
</tr>
<tr>
<td></td>
<td>Postop</td>
<td>E</td>
<td>710</td>
<td>6.67</td>
<td>108</td>
<td>41/21 (26)</td>
<td>20</td>
<td>125/80 (103)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>E</td>
<td>853</td>
<td>7.29</td>
<td>114</td>
<td>36/11 (19)</td>
<td>10</td>
<td>145/71 (99)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>E</td>
<td>1326</td>
<td>8.75</td>
<td>135</td>
<td>37/11 (23)</td>
<td>18</td>
<td>183/90 (124)</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Preop</td>
<td>R</td>
<td>287</td>
<td>5.0</td>
<td>73</td>
<td>68/35 (43)</td>
<td>31</td>
<td>114/54 (72)</td>
<td>252/36</td>
</tr>
<tr>
<td></td>
<td>Postop</td>
<td>E</td>
<td>847</td>
<td>6.6</td>
<td>98</td>
<td>95/50 (65)</td>
<td>40</td>
<td>126/68 (86)</td>
<td>268/40</td>
</tr>
<tr>
<td></td>
<td></td>
<td>E</td>
<td>182</td>
<td>4.1</td>
<td>72</td>
<td>39/17 (23)</td>
<td>14</td>
<td>132/56 (78)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>E</td>
<td>953</td>
<td>11.4</td>
<td>100</td>
<td>57/24 (36)</td>
<td>21</td>
<td>174/72 (106)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>E</td>
<td>1035</td>
<td>10.2</td>
<td>132</td>
<td>69/40 (50)</td>
<td>24</td>
<td>200/90 (102)</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>Preop</td>
<td>R</td>
<td>247</td>
<td>6.67</td>
<td>63</td>
<td>30/14 (21)</td>
<td>14</td>
<td>100/58 (76)</td>
<td>200/22</td>
</tr>
<tr>
<td></td>
<td>Postop</td>
<td>E</td>
<td>579</td>
<td>7.73</td>
<td>96</td>
<td>48/21 (30)</td>
<td>23</td>
<td>140/76 (106)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>E</td>
<td>871</td>
<td>10.50</td>
<td>106</td>
<td>40/20 (28)</td>
<td>20</td>
<td>150/82 (110)</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>Preop</td>
<td>R</td>
<td>220</td>
<td>5.64</td>
<td>60</td>
<td>26/12 (18)</td>
<td>13</td>
<td>106/64 (85)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Postop</td>
<td>E</td>
<td>807</td>
<td>9.84</td>
<td>100</td>
<td>43/20 (30)</td>
<td>20</td>
<td>136/72 (94)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>E</td>
<td>1400</td>
<td>13.9</td>
<td>118</td>
<td>40/20 (30)</td>
<td>19</td>
<td>153/77 (106)</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>Preop</td>
<td>R</td>
<td>230</td>
<td>4.44</td>
<td>69</td>
<td>29/10 (21)</td>
<td>10</td>
<td>88/48 (62)</td>
<td>206/23</td>
</tr>
<tr>
<td></td>
<td>Postop</td>
<td>E</td>
<td>251</td>
<td>4.49</td>
<td>78</td>
<td>31/12 (22)</td>
<td>12</td>
<td>111/64 (85)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>E</td>
<td>1173</td>
<td>10.58</td>
<td>114</td>
<td>50/20 (34)</td>
<td>18</td>
<td>140/72 (102)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>E</td>
<td>1525</td>
<td>11.14</td>
<td>138</td>
<td>50/24 (34)</td>
<td>20</td>
<td>157/77 (106)</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>Preop</td>
<td>R</td>
<td>175</td>
<td>3.15</td>
<td>60</td>
<td>23/10 (14)</td>
<td>10</td>
<td>96/58 (70)</td>
<td>280/28</td>
</tr>
<tr>
<td></td>
<td>Postop</td>
<td>E</td>
<td>400</td>
<td>3.66</td>
<td>96</td>
<td>47/25 (32)</td>
<td>20</td>
<td>124/74 (90)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>E</td>
<td>152</td>
<td>4.19</td>
<td>66</td>
<td>20/7 (11)</td>
<td>5</td>
<td>100/53 (73)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>E</td>
<td>468</td>
<td>6.60</td>
<td>92</td>
<td>35/11 (24)</td>
<td>12</td>
<td>132/67 (93)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>E</td>
<td>716</td>
<td>8.29</td>
<td>108</td>
<td>39/15 (26)</td>
<td>15</td>
<td>145/73 (112)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>E</td>
<td>296</td>
<td>4.53</td>
<td>78</td>
<td>30/15 (23)</td>
<td>18</td>
<td>75/56 (60)</td>
<td>128/27</td>
</tr>
<tr>
<td></td>
<td></td>
<td>E</td>
<td>1229</td>
<td>8.68</td>
<td>136</td>
<td>69/31 (49)</td>
<td>33</td>
<td>92/64 (78)</td>
<td>162/40</td>
</tr>
<tr>
<td></td>
<td></td>
<td>E</td>
<td>224</td>
<td>5.16</td>
<td>78</td>
<td>24/10 (16)</td>
<td>17</td>
<td>110/63 (82)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>E</td>
<td>1230</td>
<td>12.4</td>
<td>120</td>
<td>44/21 (30)</td>
<td>16</td>
<td>154/80 (103)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>E</td>
<td>1670</td>
<td>14.58</td>
<td>142</td>
<td>44/27 (34)</td>
<td>14</td>
<td>170/83 (110)</td>
<td></td>
</tr>
</tbody>
</table>
**Table 1.** Hemodynamic Changes of Aortic Stenosis

<table>
<thead>
<tr>
<th>9</th>
<th>Preop</th>
<th>Resting SV (L/min)</th>
<th>Cardiac Output (L/min)</th>
<th>Oxygen Consumption (ml/min)</th>
<th>Postoperative SV (L/min)</th>
<th>Cardiac Output (L/min)</th>
<th>Oxygen Consumption (ml/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>R</td>
<td>247</td>
<td>4.66</td>
<td>75</td>
<td>49/25 (35)</td>
<td>25</td>
<td>126/56 (80)</td>
</tr>
<tr>
<td></td>
<td>E1</td>
<td>162</td>
<td>7.36</td>
<td>60</td>
<td>38/18 (26)</td>
<td>24</td>
<td>164/70 (108)</td>
</tr>
<tr>
<td></td>
<td>E2</td>
<td>540</td>
<td>6.94</td>
<td>84</td>
<td>50/21 (37)</td>
<td>28</td>
<td>172/85 (114)</td>
</tr>
<tr>
<td></td>
<td>E3</td>
<td>1025</td>
<td>12.79</td>
<td>124</td>
<td>60/30 (44)</td>
<td>33</td>
<td>173/97 (129)</td>
</tr>
<tr>
<td>10</td>
<td>Preop</td>
<td>R</td>
<td>282</td>
<td>4.73</td>
<td>54</td>
<td>29/12 (16)</td>
<td>16</td>
</tr>
<tr>
<td></td>
<td>E1</td>
<td>1449</td>
<td>16.11</td>
<td>109</td>
<td>41/25 (34)</td>
<td>28</td>
<td>134/83 (102)</td>
</tr>
<tr>
<td></td>
<td>E2</td>
<td>350</td>
<td>7.78</td>
<td>63</td>
<td>31/12 (21)</td>
<td>15</td>
<td>105/55 (73)</td>
</tr>
<tr>
<td></td>
<td>E3</td>
<td>1579</td>
<td>15.64</td>
<td>99</td>
<td>34/15 (21)</td>
<td>17</td>
<td>112/63 (79)</td>
</tr>
<tr>
<td>11</td>
<td>Preop</td>
<td>R</td>
<td>309</td>
<td>4.0</td>
<td>101</td>
<td>72/41 (52)</td>
<td>30</td>
</tr>
<tr>
<td></td>
<td>E1</td>
<td>910</td>
<td>5.9</td>
<td>135</td>
<td>121/56 (81)</td>
<td>37</td>
<td>133/74 (104)</td>
</tr>
<tr>
<td></td>
<td>E2</td>
<td>769</td>
<td>8.88</td>
<td>120</td>
<td>48/20 (33)</td>
<td>20</td>
<td>147/71 (100)</td>
</tr>
<tr>
<td></td>
<td>E3</td>
<td>1035</td>
<td>11.9</td>
<td>150</td>
<td>57/34 (41)</td>
<td>37</td>
<td>172/82 (116)</td>
</tr>
<tr>
<td>12</td>
<td>Preop</td>
<td>R</td>
<td>250</td>
<td>6.94</td>
<td>76</td>
<td>27</td>
<td>101/68 (79)</td>
</tr>
<tr>
<td></td>
<td>E1</td>
<td>263</td>
<td>6.5</td>
<td>76</td>
<td>22/10 (11)</td>
<td>10</td>
<td>104/58</td>
</tr>
<tr>
<td></td>
<td>E2</td>
<td>1126</td>
<td>11.2</td>
<td>136</td>
<td>39/21 (25)</td>
<td>20</td>
<td>156/78 (116)</td>
</tr>
<tr>
<td></td>
<td>E3</td>
<td>1579</td>
<td>13.1</td>
<td>108</td>
<td>46/32 (25)</td>
<td>30</td>
<td>176/90 (128)</td>
</tr>
</tbody>
</table>

Abbreviations: R = rest; E1 and E2 = first and second exercise periods, respectively; PA = pulmonary artery; PAW = pulmonary wedge; Ao = aorta.

The chart on the right shows the relationship between exercise VO2 and CO: as heart rate decreased, VO2 and CO increased in a linear fashion. Decreased by 8 beats/min, in 96±28 ml (+30%). The increase in exercise volume (SV) was due to increased arteriovenous oxygen difference (4.73 = 5.14 L/min). The arteriovenous oxygen difference decreased from 53 to 42 vol% (18%). The peak systolic pressure (PVR) index decreased from 103.3 to 89.5 vol%. The decrease in oxygen consumption (VO2) was found to be 2.6 ml/kg after exercise in 96 ± 28 ml (+30%). The resting heart rate was slightly heavier, indicating that the intensity of cardiovascular exercise was slightly greater. Similarly, cardiac output was increased by 15%.

- **Resting oxygen consumption (VO2)**: decreased by 106 (fig. 2) from 245 to 218 ml/min after the first exercise (E1) was 72/41 (52) mm Hg. The average VO2 was 72/41 (52) mm Hg. The resting heart rate was 15%.
- **Postoperative SV**: decreased by 3.9 (±10) mm Hg. The average VO2 was also decreased by 15%.
- **Cardiac output**: decreased by 21% while VO2 increased by 15%.
Figure 2

Cardiac output, arteriovenous oxygen difference, stroke volume, and the ratio of stroke volume to wedge pressure (SV/WP) before and after surgery. Clear bar = preoperative; crossed bar = postoperative.

Figure 3

Cardiac output in relation to oxygen consumption at rest and on exercise.

(CO = 2.782 + 0.0069 VO₂, r = 0.84, P < 0.001) and after the surgery (CO = 3.7995 + 0.0062 VO₂, r = 0.907, P < 0.001). Thus, the regression of cardiac output on VO₂ improved toward normal, and it was almost identical to that found in patients with normal cardiovascular systems of similar age (30 to 64 years) studied by us: CO = 3.83 + 0.0061 VO₂, r = 0.90, n = 52 (11 males and 12 females). The pulmonary artery systolic (PAS) and the wedge pressure (PAW) increased progressively with exercise before surgery: PAS = 31.27 + 0.031 VO₂, r = 0.463, P < 0.05; PAW = 13.47 + 0.0149 VO₂, r = 0.618, P < 0.001. After surgery, however, the pressure increase due to exercise was significantly less: PAS = 28.2 + 0.0135 VO₂, r = 0.55,
HEMODYNAMIC CHANGES OF AORTIC STENOSIS

Table 3

<table>
<thead>
<tr>
<th>Paired Comparison of Preoperative and Postoperative Data</th>
<th>S</th>
<th>D</th>
<th>M</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time (h)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Preop</td>
<td>24 ± 14</td>
<td>4.731 ± 1.25</td>
<td>77 ± 18</td>
</tr>
<tr>
<td>PO</td>
<td>218 ± 16</td>
<td>5.133 ± 1.56</td>
<td>71 ± 9</td>
</tr>
<tr>
<td>P</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>VO2 (m/min)</td>
<td>897 ± 115</td>
<td>10.348 ± 2.88</td>
<td>107 ± 11</td>
</tr>
<tr>
<td>PAW (mm Hg)</td>
<td>28 ± 6</td>
<td>7.05 ± 2.6</td>
<td>NS</td>
</tr>
<tr>
<td>SV/PAW</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>77 ± 18</td>
<td>4.45 ± 2.10</td>
<td>71 ± 9</td>
</tr>
<tr>
<td>Rest</td>
<td>18 ± 11</td>
<td>37 ± 17</td>
<td>NS</td>
</tr>
<tr>
<td>Exercise</td>
<td>11 ± 4</td>
<td>7 ± 5</td>
<td>NS</td>
</tr>
</tbody>
</table>

Abbreviations: SV = stroke volume; PAW = pulmonary artery wedge pressure.

$P < 0.05$, PAW $= 12.2 + 0.0064 \cdot V_{O2}$, $r = 0.44$, $P < 0.05$ (fig. 4).

Following surgery, 11 patients were studied at two levels of exercise. For the first exercise, the average values were: $V_{O2}$, $851 \pm 262$ ml/min; CO, $9.23 \pm 2.28$ L/min; HR, $108 \pm 15$ beats/min, and SV, $84 \pm 17$ ml. For the second exercise, the $V_{O2}$, cardiac output, and heart rate increased to $1263 \pm 287$ ml/min ($P < 0.001$), $11.74 \pm 2.36$ L/min ($P < 0.001$), and 134 beats/min, respectively. In spite of the moderately severe exercise, that is, six times the resting $V_{O2}$, no patients experienced anginal pain during the study.

Discussion

In severe aortic stenosis, Lee and associates$^7$ previously showed that both the left ventricular end-diastolic and the pulmonary artery pressure may be significantly elevated at rest and that exercise commonly causes a further increase in these pressures. The preoperative data of the present study support the previous findings. One of the significant changes following surgery was in the wedge pressure, which decreased both at rest (33%, $P < 0.05$) and on exercise (45%, $P < 0.001$), suggesting that the left ventricular end-diastolic pressure...
also decreased significantly. We suggest that this is due to decreased systolic pressure and also to the changes in the myocardial compliance, possibly because of diminished left ventricular wall thickness.

Before operation, the wedge pressure had a tendency to increase in a linear fashion with the degree of exercise, that is, with exercise \( \text{VO}_2 \) \( (r = 0.618) \). A similar correlation was found in the patients with severe aortic stenosis studied previously\(^7\) (LA or PAW pressure was \( 10.8 + 0.0199 \times \text{VO}_2, \ r = 0.594, P < 0.01 \)). The reason for the increasing end-diastolic pressure and the apparent decrease in the myocardial compliance during exercise has not been established, but it is likely due to the increasing systolic pressure and the relative myocardial ischemia which may develop from the increased myocardial oxygen demand. Following surgery, the wedge pressure decreased both in the intercept and in the slope of the regression (from 0.0149 to 0.0064) but still maintained its tendency to increase with exercise (fig. 4), indicating the residual abnormalities of the left ventricular function.

The pulmonary artery systolic pressure, as a linear function of the exercise \( \text{VO}_2 \), also decreased in intercept and in the regression coefficient (from 0.030 to 0.013), which must be due to the reduction in the wedge pressure and the pulmonary vascular resistance after surgery. The paired comparison of pulmonary vascular resistances decreased by 25% at rest and on exercise after surgery, as a result of decreased wedge pressure, and the total pulmonary resistance (PA mean pressure, mm Hg/CO, L/min) also decreased by 38% at rest and 32% during exercise (5.39 to 3.36 and 4.13 to 2.81). The degree of hyperventilation (ventilation/\( \text{VO}_2 \)) during exercise is an objective measurement of dyspnea, and among the hemodynamic parameters studied it was found to be most closely related to the total pulmonary vascular resistance\(^8\) \( (r = 0.816) \). Hence, the disappearance of dyspnea on exertion, noted by most of these patients, appears to be related to this decrease in pulmonary vascular resistance.

The average peak systolic gradient measured in eight patients after operation was 3.9 (± 10) mm Hg; no gradient was found in six patients, and it was 28 mm Hg and 3 mm Hg in the remaining two patients. Therefore, the residual abnormalities of the left ventricular function do not appear to be due to the inadequate valve orifice. Our result is significantly different from that of McHenry and associates\(^9\) who found an average gradient of 19 (± 8) mm Hg in seven patients with the same prosthesis. In 60 patients with the Starr-Edwards prosthesis reported on by others,\(^1, 2, 10-12\) the average gradient was 13.9 (± 11.1) mm Hg.

As found by others, the changes in cardiac output following surgery are rather small (table 2, fig. 2). Hultgren and associates\(^3\) found an increase in resting cardiac index of 16% and a decrease in arteriovenous oxygen difference of 19%, while Bristow\(^4\) and Ross\(^2\) and their co-workers have reported an increase in resting cardiac index of only 4% and 3%, respectively. In the present study, the arteriovenous oxygen difference as a measure of the adequacy of cardiac output decreased by 18% at rest and by 7% during exercise. The rather small magnitude of these changes is probably due to the fact that only a few patients were in true heart failure with low cardiac output before surgery. Although the changes in cardiac output were small, the increase in stroke volume was greater at rest (18%) and especially during exercise (30%). This suggests increased myocardial contraction or fiber shortening, since the end-diastolic volume or the initial fiber length should not have increased afterward. This appears to be the result of decreased afterload on the left ventricle. It has been shown that exercise stroke volume is a more sensitive indicator of cardiac performance than cardiac output,\(^13-15\) as the inadequate cardiac output can be compensated by an increase in heart rate. Thus, the 30% increase in exercise stroke volume, from 74 ml to 96 ml, indicates a true improvement in the ability to increase cardiac output during exercise. The maximum stroke volume is reached with a moderate degree of
exercise and remains relatively constant with the maximum exercise,\textsuperscript{13-16} In 11 patients who were studied on two levels of exercise after operation, the stroke volumes were 84 ± 17 ml and 85 ± 16 ml on the first and second exercises, in keeping with the findings in normal individuals. Assuming the maximum heart rate is not altered due to surgery, the 33% increase in exercise stroke volume is equal to the increase in the maximum cardiac output of this magnitude.

As in normal individuals,\textsuperscript{13-15} cardiac output at rest and on submaximal exercise showed a linear increase with oxygen consumption before (r = 0.84, \( P < 0.001 \)) and after surgery (r = 0.91, \( P < 0.001 \)). Although the intercept was higher after the surgery, the regression coefficient did not change (0.0069 compared to 0.0062); this indicates that the exercise factor was unchanged. Before operation, in spite of the high systolic pressure gradient (92 ± 29 mm Hg), all patients were able to achieve an increase in cardiac output with exercise, as was found in a previous report\textsuperscript{7}; therefore, a truly fixed cardiac output in aortic stenosis must be rare.

The relationship between cardiac output and \( \dot{V}O_2 \) in the present series has been compared with that in normal subjects studied by Donald,\textsuperscript{17} Holmgren,\textsuperscript{13} and Bevegård\textsuperscript{14} and their associates, as well as the patients studied after aortic valve replacement (Starr-Edwards) by Hultgren\textsuperscript{9} and Ross\textsuperscript{2} and their coworkers. There is essentially no difference among the three groups of patients with aortic valve replacement (fig. 3). The regression in patients who have had valve replacement is significantly lower in the intercept when compared to the young normal individuals\textsuperscript{13, 14, 17} but is the same as that found in the normal individuals of similar age studied by us. It is interesting to note that the regression coefficients are similar in all these studies: Donald's group, 0.00566; Bevegård's group, 0.0061; Lee's group, 0.0061 (normals) and 0.00623 (postoperative patients); Hultgren's group, 0.00649; and Ross's group, 0.0058. These values indicate that the exercise factor, that is, the increase in cardiac output for a given increase in oxygen consumption,\textsuperscript{2} is the same in all groups. The characteristic of the low cardiac output state, therefore, is the lower intercept of this regression rather than the reduced exercise factor.

Following surgery, the ratio of the stroke volume and the pulmonary artery wedge pressure (SV/PAW), as an index of the left ventricular function, improved by 57% at rest (4.45 to 7.05) and 105% during exercise (2.96 to 6.08). In normal individuals, supine leg exercise has been found to increase stroke volume without changing PA wedge pressure,\textsuperscript{13-15} resulting in an increase in the SV/WP index. However, in the patients with aortic stenosis considered here, this index decreased with exercise by 34% (4.45 to 2.96) before operation and 14% (7.05 to 6.08) afterward, which suggests residual dysfunction of the left ventricle. It is likely that those patients with severely depressed left ventricular function before surgery may continue to have it afterward; in fact, the SV/WAP ratios during exercise before and after surgery did show a significant positive correlation (\( P < 0.001 \)): postoperative SV/PAW = 3.575 + 0.844 SV/PAW, \( r = 0.614 \). The clinical implication of this finding is that the valve surgery should be carried out before severe left ventricular failure ensues, as such patients may fail to improve significantly afterward.

From 1966 to 1969 inclusive, the Cutter-Smeloff aortic prosthesis has been used in 166 patients at the University of Alberta Hospital, and no incidence of prosthesis failure due to ball degeneration has been encountered to date. Bloodwell and associates\textsuperscript{5} also have reported no such complication in a series of 635 patients. This complication appears to be rare in the Cutter-Smeloff aortic prosthesis, as only three such cases have been reported thus far.\textsuperscript{18} It may be more common in the Starr-Edwards prosthesis.\textsuperscript{10, 20} This absence of complication, in our opinion, is due to the relatively smaller diameter of the ball in the Cutter-Smeloff prosthesis than in the other valve, which allows more space for expansion between the ball and the cage if the ball should swell.
Previously we reported three cases of failure of the mitral Cutter-Smeloff prosthesis\textsuperscript{21} due to “sticking” of the ball in the seating ring as a result of minimum swelling of the ball. The fact that the same phenomenon has not been encountered with the aortic prosthesis is likely due to the lesser force with which the aortic ball descends during diastole, while the mitral ball strikes the ring with a greater force during systole; therefore, although we have stopped using the mitral prosthesis, the aortic Cutter-Smeloff prosthesis remains our choice.

References

Hemodynamic Changes Following Correction of Severe Aortic Stenosis Using the Cutter-Smeloff Prosthesis

SIMON J. K. LEE, M. HARAPHONGSE, J. C. CALLAGHAN, R. E. ROSSALL and R. S. FRASER

Circulation. 1970;42:719-728
doi: 10.1161/01.CIR.42.4.719
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1970 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/42/4/719

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/