Evaluation of the Left Ventricle in Patients with Mitral Stenosis

By Harvey Feigenbaum, M.D., Richard W. Campbell, M.D., Charles M. Wunsch, M.D., and Edward F. Steinmetz, M.D.

MANY THEORIES have been advanced concerning the mechanisms responsible for the reduced cardiac output in patients with mitral stenosis.1 Although the mechanical block at the mitral orifice is traditionally the most important factor,2 it is obviously not the entire answer since cardiac output frequently does not increase following mitral commissurotomy.3-5 Some investigators consider that this failure to improve after surgery is due in part to left ventricular dysfunction.6 This ventricular abnormality has been referred to as the “myocardial factor”7 and has been diagnosed primarily by exclusion. Although there is some anatomic evidence to suggest left ventricular alterations in patients with mitral stenosis,8 objective hemodynamic evidence to that effect is still lacking. Thus, there remains serious doubt that altered left ventricular function is in any way an important hemodynamic factor in these patients.9

This study was undertaken (1) to see if a modified form of supine leg exercise during simultaneous left and right-heart catheterization would help uncover any left ventricular abnormality in patients with mitral stenosis, (2) to attempt to evaluate the left ventricle by scrutinizing changes in pressure and volume during diastole, and (3) if any left ventricular abnormality should be demonstrated, to see what relationship it might have with cardiac output.

Methods

Thirty-two patients proven to have pure mitral stenosis as determined by left-heart catheterization and selective cineangiography10 and nine “control” patients with either innocent functional heart murmurs or hemodynamically insignificant stenotic valvular lesions were studied. Simultaneous right and left-heart catheterizations were performed in the fasting state without any premedication. The right-heart catheter was positioned in the pulmonary artery (PA) while a retrograde aortic catheter was maneuvered across the aortic valve into the left ventricle (LV). In addition, all patients with mitral stenosis and some control subjects had a transseptal catheter introduced into the left atrium (LA). Utilizing these three catheters plus an arterial needle in the brachial artery (BA), simultaneous measurements were made of PA, LA, LV, and BA pressures together with cardiac output, as determined by the direct Fick method. The pressures were recorded on a multichannel recorder utilizing Statham P-23-D pressure transducers.

Following the resting determinations, each patient exercised the lower extremity in which no catheter was inserted. The patient slid the heel of that foot along the top of the x-ray table 60 times per minute, as paced by a metronome. The exercise increased the mean oxygen consumption of all 41 patients from 130 ± 23 ml/min/m² to 251 ± 51 ml/min/m².2 Since the extremities in which catheters were introduced were not moved, this form of exercise minimized artifactual interference in the pressure tracings. After exercising for at least 2 minutes, simultaneous pressures and cardiac output were again obtained. Following exercise, selective cineangiocardiology was performed with injections into the left atrium, left ventricle, and central aorta to exclude any other valvular lesions, to evaluate further the degree of mitral stenosis,11 and to estimate the size of the left ventricular chamber.
LEFT VENTRICLE IN MITRAL STENOSIS

The mean diastolic mitral valve pressure gradient (MVG) was determined by dividing the planimetrically determined area between the left atrial and left ventricular diastolic pressure tracings by the diastolic filling period (DFP). Mitral valve flow (MVF) was calculated by using the following formula:

$$\text{MVF} = \frac{\text{Cardiac output}}{\text{Heart rate} \times \text{DFP}},$$

and mitral valve area (MVA) was derived from the formula:

$$\text{MVA} = \frac{\text{MVF}}{31 \sqrt{\text{MVG}}}^{12, 13}$$

The pulmonary vascular resistance (PVR) was obtained by dividing the difference between the mean PA and LA pressures by the cardiac output and converting the answer to dynes sec cm⁻⁵.

In an attempt to evaluate the status of the left ventricle, the average rate or slope of the rise in left ventricular pressure during diastole (Ap/At) was measured from the pressure tracings. In order to relate change in diastolic pressure to change in diastolic volume, Δp/Δt was then divided into the average rate of diastolic volume change (Δv/Δt). Provided that there is no valvular insufficiency, the rate of left ventricular diastolic volume change is the same as mitral valve flow (MVF). The resultant ratio $\frac{\text{MVF}}{\Delta p/\Delta t}$ or $\Delta v/\Delta p$ was calculated during rest and exercise on all 41 patients. This ratio, Δv/Δp in milliliters per mm Hg, is an estimate of the mean increase in left ventricular diastolic volume per mm Hg increase in left ventricular diastolic pressure during ventricular filling. Since there is frequently some beat-to-beat variation in left ventricular pressure tracings, at least six individual cardiac cycles were used to determine Δp/Δt. More cardiac cycles were utilized when atrial fibrillation was present.

Results

The results of studies on all nine patients without mitral stenosis are given in Table 1. Except for insignificant pressure gradients in four patients, the hemodynamic data, including both resting and exercise cardiac output (CO) and left ventricular end-diastolic pressures (LVEDP), were within normal limits. The resting $\Delta v/\Delta p$ ratio ranged from 8.0 to 10.9 ml/mm Hg with a mean of 9.6 ± 0.77 ml/mm Hg. Except for one subject who increased his ratio by 2.5 ml/mm Hg, there was no appreciable change with exercise. Figure 1 illustrates the pressure tracings obtained from one of these patients. All of the pressures were within normal limits. With exercise there was an increase in $\Delta v/\Delta t$; however, there was a comparable increase in $\Delta p/\Delta t$ resulting in no essential change in the ratio $\Delta v/\Delta p$.

Table 2 presents the results of observations on the patients with mitral stenosis. There was no uniformity in severity of the disease. The mitral valve area ranged from 0.5 cm² to 2.3 cm². The mean cardiac output was reduced both at rest (2.6 ± 0.7 L/min/m²) and with exercise (3.3 ± 0.8 L/min/m²) compared with the control values of 3.6 ± 0.7 L/min/m² and 4.9 ± 0.5 L/min/m², resting and with exercise, respectively. Twenty-five of the 32 patients with mitral stenosis had resting cardiac outputs below the lowest value for the controls (3.0 L/min/m²). Two of the remaining seven patients failed to increase their output with exercise and only one patient increased the output by 1 L/min/m² with exercise.

There was a wide range in $\Delta v/\Delta p$ ratios. Twenty-two of the patients had either a resting or exercise ratio or both which were lower than noted among the control subjects. One outstanding difference was that whereas the control subjects had relatively little change or a slight increase in $\Delta v/\Delta p$ during exercise, 19 of the patients with mitral stenosis decreased this ratio with exercise. All of the patients with atrial fibrillation, high pulmonary vascular resistance (more than 400 dynes sec cm⁻⁵), or markedly elevated LVEDP (more than 14 mm Hg) were among those who decreased their $\Delta v/\Delta p$ with exercise. Figure 2 shows the pressure tracings from one such patient with atrial fibrillation, and figure 3 illustrates the pressures in a patient with marked pulmonary hypertension. The prominent feature was that with exercise the increase in the slope of the diastolic ventricular pressure (Δp/Δt) was out of proportion to the increase in Δv/Δt.

Although there was no quantitative measurement of left ventricular volume, a comparative estimate was made by using selective cineangiocardiology. These studies showed no sig-
Table 1

Control Subjects

<table>
<thead>
<tr>
<th>Patient</th>
<th>Diagnosis</th>
<th>Age (yr) &amp; sex</th>
<th>SA (m²)</th>
<th>( \dot{V}O_2 ) (ml/min/m²)</th>
<th>LVEDP (mm Hg)</th>
<th>HR (per min)</th>
<th>CO (L/min/m²)</th>
<th>( \Delta V/\Delta t ) (ml/sec)</th>
<th>( \Delta p/\Delta t ) (mm Hg/sec)</th>
<th>( \Delta V/\Delta p ) (ml/mm Hg)</th>
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</thead>
<tbody>
<tr>
<td>M.H.</td>
<td>Functional murmur</td>
<td>31 F</td>
<td>1.64</td>
<td>133 193</td>
<td>8 9</td>
<td>85 95</td>
<td>3.5 4.1</td>
<td>296 376</td>
<td>35.0 47.6</td>
<td>8.5 8.0</td>
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<tr>
<td>D.F.</td>
<td>Aortic stenosis*</td>
<td>18 M</td>
<td>1.85</td>
<td>188 281</td>
<td>10 3</td>
<td>75 92</td>
<td>4.2 5.7</td>
<td>302 498</td>
<td>32.1 47.2</td>
<td>9.8 10.6</td>
</tr>
<tr>
<td>M.R.</td>
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<td>34 F</td>
<td>1.67</td>
<td>121 234</td>
<td>8 3</td>
<td>70 87</td>
<td>3.2 4.0</td>
<td>272 286</td>
<td>28.4 27.7</td>
<td>10.9 10.1</td>
</tr>
<tr>
<td>R.W.</td>
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<td>1.79</td>
<td>94 251</td>
<td>8 5</td>
<td>79 115</td>
<td>3.0 3.9</td>
<td>222 684</td>
<td>21.7 56.6</td>
<td>10.5 13.0</td>
</tr>
<tr>
<td>W.M.</td>
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<td>32 F</td>
<td>1.92</td>
<td>121 222</td>
<td>6 5</td>
<td>75 100</td>
<td>3.1 5.6</td>
<td>182 339</td>
<td>22.0 39.9</td>
<td>8.4 8.7</td>
</tr>
<tr>
<td>T.H.</td>
<td>Pulmonic stenosis‡</td>
<td>24 M</td>
<td>1.98</td>
<td>127 270</td>
<td>8 12</td>
<td>72 89</td>
<td>3.3 4.7</td>
<td>216 336</td>
<td>25.3 42.0</td>
<td>8.6 8.8</td>
</tr>
<tr>
<td>J.S.</td>
<td>Pulmonic stenosis§</td>
<td>24 M</td>
<td>1.81</td>
<td>136 312</td>
<td>10 12</td>
<td>74 83</td>
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<td>9.3 9.5</td>
</tr>
<tr>
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<td>164 267</td>
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<td>66 97</td>
<td>4.9 5.3</td>
<td>257 330</td>
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</tr>
<tr>
<td>P.S.</td>
<td>Functional murmur</td>
<td>23 F</td>
<td>1.46</td>
<td>129 232</td>
<td>4 3</td>
<td>95 121</td>
<td>3.9 4.8</td>
<td>245 365</td>
<td>30.8 42.1</td>
<td>8.0 8.7</td>
</tr>
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*17 mm gradient
‡3.3 mm gradient
§29 mm gradient
\$26 mm gradient

Abbreviations: SA = body area; \( \dot{V}O_2 \) = oxygen consumption per square meter of body area; LVEDP = left ventricular end-diastolic pressure; CO = cardiac output; \( \Delta V/\Delta t \) = change in volume/change in time; \( \Delta p/\Delta t \) = change in pressure/change in time.
Intracardiac pressures in a patient with a functional heart murmur. With exercise the increase in volume change (Δv/Δt) and the increase in diastolic pressure change (Δp/Δt) are proportional so that there is no change in the ratio Δv/Δp.

significant difference in left ventricular volumes in any of the 41 subjects.

Twenty-four of the 32 patients with mitral stenosis had gone to surgery at the time of this writing. Whether or not there was histological evidence of carditis in the atrial appendage is noted in table 2. Only four patients showed no Aschoff nodules, mononuclear cellular infiltration, or fibrosis. Three of these patients (numbers 3, 4, and 6 of table 2) had normal Δv/Δp ratios. The fourth patient had a slightly subnormal resting ratio.

Assuming that the Δv/Δp measurements might reflect some aspect of left ventricular function, an attempt was made to determine whether the abnormal Δv/Δp ratios noted in some of the patients with mitral stenosis were related to the reduced cardiac output also present in most of these same patients. A digital computer was used to perform a stepwise multiple regression with cardiac output as the dependent variable and all of the other hemodynamic parameters listed in table 2 as the independent variables. The results of this statistical endeavor to identify those factors responsible for determining cardiac output are shown in table 3. The resting Δv/Δp ratio proved to be the hemodynamic parameter which correlated best with resting cardiac output. Resting pulmonary vascular resistance and rhythm, whether sinus or atrial fibrillation, were significant additional factors; however, mitral valve area was slightly above the usually accepted 5% level of significance. The exercise pulmonary vascular resistance was by
Patients with Mitral Stenosis

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr) &amp; sex</th>
<th>SA (m²)</th>
<th>Rhythm</th>
<th>MVA (cm²)</th>
<th>Carditis</th>
<th>( \dot{V}O_2 ) (ml/min/m²)</th>
<th>( P_A ) (mm Hg)</th>
<th>LA (mm Hg)</th>
<th>LVEDP (mm Hg)</th>
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<td>1. J.N.</td>
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<td>1.52</td>
<td>NSR</td>
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<td>R</td>
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<td>28 27</td>
<td>5 3</td>
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<tr>
<td>2. R.E.</td>
<td>36 F</td>
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<td>NSR</td>
<td>1.5</td>
<td>R</td>
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<td>30 47</td>
<td>19 28</td>
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<tr>
<td>3. N.L.</td>
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<td>46 75</td>
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<td>14 13</td>
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<tr>
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<td>29 F</td>
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<td>NSR</td>
<td>0.9</td>
<td>-</td>
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<td>27 40</td>
<td>5 4</td>
</tr>
<tr>
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<td>-</td>
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<td>23 30</td>
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<td>53 F</td>
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<td>-</td>
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<td>32 39</td>
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<tr>
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<td>NSR</td>
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<td>37 76</td>
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<tr>
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<td>12 13</td>
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<td>36 47</td>
<td>22 32</td>
<td>9 10</td>
</tr>
<tr>
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<td>NSR</td>
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<td>+</td>
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<td>22 37</td>
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<td>11 6</td>
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<tr>
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<td>+</td>
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<tr>
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<td>-</td>
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<tr>
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<td></td>
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<td>±15 ±22</td>
<td>±8 ±9</td>
<td>±3 ±6</td>
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</table>

Abbreviations: SA = surface area; MVA = mitral valve area; \( \dot{V}O_2 \) = oxygen consumption per square meter body surface; \( P_A \) = pulmonary artery mean; LA = left atrial mean; LVEDP = left ventricular end-diastolic pressure; PVR = pulmonary vascular resistance; MVG = mitral valve gradient; HR = heart rate; CO = cardiac output; \( \Delta v/\Delta t \) = change in volume/change in time; \( \Delta p/\Delta t \) = change in pressure/change in time.

Discussion

The ratio \( \Delta v/\Delta p \) is considered to be an estimate of the mean increase in ventricular volume per mm Hg rise in pressure during diastole. There are, however, distinct limitations to the conclusions that can be drawn from the use of this ratio as an index of ventricular diastolic function. First of all, left ventricular pressures, as measured by the retrograde aortic catheter technique, frequent-
ly possess artifacts especially during exercise. Limiting the exercise to only one lower extremity minimized but did not entirely eliminate this problem. This difficulty, however, was common to both the control subjects and the patients with mitral stenosis, and artifacts could not easily explain the observed differences between the two groups. In addition, it must be remembered that the ratio $\Delta v/\Delta p$ is only a crude attempt to relate the left ventricular diastolic pressure and volume changes. The significance that can be attached to the values of this ratio is limited by the fact that total left ventricular volumes were not measured and mitral valve flow is not constant.

Despite these limitations and the unsophisticated methodology, the following observations were considered impressive and therefore worthy of reporting. First of all, eight of our patients with mitral stenosis had LVEDP greater than 14 mm Hg either at rest or with exercise. Despite the technical difficulties of obtaining LV pressures during exercise, none of our control subjects had an LVEDP greater than 12 mm Hg. By modifying the exercise, the artifact was actually minimal, thus it is felt that the LVEDPs are valid measurements.

\[ \text{LVEDP} = \text{LV end-diastolic pressure} \]

\[ \text{CO} = \text{cardiac output} \]

\[ \Delta v/\Delta p = \text{ratio of volume change to pressure change} \]

\[ \text{HR} = \text{heart rate} \]

\[ \text{PVR} = \text{pulmonary vascular resistance} \]

\[ \text{MVG} = \text{mitral valve gradient} \]

\[ \text{R} = \text{resistance} \]

\[ \text{sec} = \text{seconds} \]

\[ \text{mm Hg} = \text{millimeters of mercury} \]

\[ \text{L/min} = \text{liters per minute} \]

\[ \text{mL/sec} = \text{milliliters per second} \]

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\[ \text{mL/sec} = \text{milliliters per second} \]

\[ \text{mL/mm Hg} = \text{milliliters per millimeter of mercury} \]
Secondly, in 19 of the 32 patients with mitral stenosis, exercise produced a rise in left ventricular diastolic pressure (Δp/Δt) which was proportionally greater than the increase in left ventricular diastolic inflow (Δv/Δt). Thus the ratio Δv/Δp decreased with exercise. Again this finding was not present among the control subjects in whom this ratio was fairly constant.

The abnormal left ventricular response to exercise occurred often enough among the patients with mitral stenosis that some explanation was deemed necessary. The primary explanation might be that there was valvular insufficiency which worsened with exercise and Δv/Δt was actually higher than calculated. This possibility was excluded by using selective cineangiography to eliminate those patients with any mitral or aortic regurgitation. If it is assumed that the Δv/Δp measurements do reflect some aspect of the left ventricular pressure-volume relationship, the different values might only indicate that the patients with mitral stenosis and the con-
Intracardiac pressure tracings in a patient with mitral stenosis and markedly elevated pulmonary vascular resistance. Both resting and exercise pulmonary artery (PA) pressures are at systemic levels. The ratio $\Delta v/\Delta p$ is reduced at rest and falls even further with exercise.

Control subjects have chambers which are functioning along different portions of the same pressure-volume curve. Such an explanation would be valid if the mitral stenosis patients had increased left ventricular volumes. Without actually quantitatively measuring left ventricular volumes, this explanation can not be refuted. However, there was no obvious left ventricular enlargement on the cineangio-grams, and this is not in general characteristic of pure mitral stenosis.

It is well appreciated that other factors may have an etiological role in the observations made in this study; however, the one explanation which is most attractive is that those patients whose $\Delta v/\Delta p$ ratio declined and whose LVEDP rose with exercise possessed left ventricles which were abnormal. One might go further to suggest that the abnormality was in

*Figure 3*
some way related to abnormal left ventricular compliance. Decreased compliance would be
an attractive explanation for the abnormal increase in diastolic ventricular pressure.\textsuperscript{14} In
addition, the close statistical relationship between cardiac output and $\Delta v/\Delta p$ suggests that
the left ventricle may indeed be a factor in limiting cardiac output in patients with
mitral stenosis.

The etiology of the proposed ventricular abnormality is not obvious. As noted, of our 24
patients who had surgery, only four failed to exhibit evidence of active or chronic carditis in
their atrial appendages. Interestingly enough, all four had normal or nearly normal LVEDPs and $\Delta v/\Delta p$ values, thus suggesting an
association between the pathological changes and the hemodynamic changes. Such an
association would be consistent with the supposition that the myocardial abnormality was
a consequence of rheumatic carditis.\textsuperscript{8} It must be pointed out, however, that the histological
appearance of the atrial appendage does not necessarily reflect the status of the left ventricle. In fact, only 10 of 101 autopsied cases of patients dying with mitral stenosis showed
evidence of either active or chronic myocarditis involving the left ventricle.\textsuperscript{15}

It is quite possible that abnormal ventricular compliance could be a reflection of the anatonic abnormalities noted by Grant.\textsuperscript{8} He
found that the posterior wall of the left ventricle in patients with mitral stenosis was
shortened and atrophic. He likened these atrophic changes to those occurring in skeletal
muscle immobilized by a cast. These ventricular alterations were attributed to partial im-
mobilization of the ventricle by the fibrotic posterior mitral leaflet and also to the chronic
ventricular inactivity resulting from long-standing reduced cardiac output. It is conceivable
that once cardiac output falls because of mechanical obstruction, elevated pulmonary
vascular resistance, atrial fibrillation, or ventricular dysfunction, a vicious cycle may en-
sue with a resultant further deterioration of ventricular function and further reduction in
blood flow.

This theory may help explain the observation that all of the patients with atrial fibrilla-
tion and markedly elevated pulmonary vascular resistance were among those who had a
decrease in $\Delta v/\Delta p$ with exercise. Both atrial fibrillation and elevated pulmonary vascular resistance may contribute to left ventricular
inactivity by helping to reduce blood flow.

Table 3
Stepwise Building of Regression Equation for Predicting Cardiac Output

<table>
<thead>
<tr>
<th>Step</th>
<th>Variable entering</th>
<th>Multiple R</th>
<th>Significance test on increment in mult. R</th>
</tr>
</thead>
<tbody>
<tr>
<td>Resting cardiac output</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>$\Delta v/\Delta p$ (R)</td>
<td>0.538</td>
<td>$P &lt; 0.005$</td>
</tr>
<tr>
<td>2</td>
<td>PVR (R)</td>
<td>0.628</td>
<td>$P &lt; 0.05$</td>
</tr>
<tr>
<td>3</td>
<td>Rhythm</td>
<td>0.710</td>
<td>$P &lt; 0.025$</td>
</tr>
<tr>
<td>4</td>
<td>MVA</td>
<td>0.744</td>
<td>$P &lt; 0.10$</td>
</tr>
<tr>
<td>5</td>
<td>All other parameters grossly insignificant</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exercise cardiac output</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>PVR (E)</td>
<td>0.624</td>
<td>$P &lt; 0.0005$</td>
</tr>
<tr>
<td>2</td>
<td>$\Delta v/\Delta p$ (E)</td>
<td>0.764</td>
<td>$P &lt; 0.001$</td>
</tr>
<tr>
<td>3</td>
<td>MVA</td>
<td>0.781</td>
<td>$P &lt; 0.10$</td>
</tr>
<tr>
<td>4</td>
<td>Rhythm</td>
<td>0.801</td>
<td>$P &lt; 0.10$</td>
</tr>
<tr>
<td>5</td>
<td>All other parameters grossly insignificant</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Difference in cardiac output (E-R)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>$\Delta v/\Delta p$ (dif)</td>
<td>0.460</td>
<td>$P &lt; 0.01$</td>
</tr>
<tr>
<td>2</td>
<td>Rhythm</td>
<td>0.553</td>
<td>$P &lt; 0.10$</td>
</tr>
<tr>
<td>3</td>
<td>All other parameters grossly insignificant</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Circulation, Volume XXXIV, September 1966
Another explanation for the association of atrial fibrillation, elevated pulmonary vascular resistance, and possible abnormal left ventricular compliance may be that long-standing mitral obstruction may be a common etiological factor for all three hemodynamic abnormalities.

This concept of ventricular atrophy would also help explain the observation that, when the cardiac output does improve after mitral commissurotomy, this improvement may take several months to occur. It is well known that it takes time and training for an immobilized skeletal muscle to regain full strength. It is quite conceivable that the myocardium likewise requires time to recover from its prolonged immobilization and inactivity.

**Summary**

In an attempt to objectively evaluate the status of the left ventricle in 32 patients with mitral stenosis, a modified form of supine leg exercise was done during simultaneous left and right heart catheterization. Besides measuring the usual hemodynamic parameters, a ratio of mitral valve flow (MVF) or diastolic left ventricular volume change (Δv/Δt) and the slope of the rise of left ventricular diastolic pressure (Δp/Δt) was calculated at rest and during exercise (Δv/Δp). The results of these measurements were compared with those obtained on nine patients with either innocent functional heart murmurs or hemodynamically insignificant stenotic valvular lesions.

None of the nine control patients had left ventricular end-diastolic pressures (LVEDP) greater than 12 mm Hg either at rest or with exercise. Exercise also did not appreciably alter the Δv/Δp ratio. Among the patients with mitral stenosis eight patients had distinctly elevated (greater than 14 mm Hg) LVEDP with exercise and 19 patients, including the eight with abnormal LVEDP, decreased the Δv/Δp ratio with exercise.

The difficulties and limitations associated with this type of study are discussed. However, there is a distinct possibility that these results are indicative of an abnormal left ventricle in some patients with mitral stenosis. It is further suggested that this abnormality may be related in some way to decreased left ventricular compliance. In addition, a close statistical correlation between Δv/Δp and cardiac output implicates this left ventricular abnormality as a possible etiological factor in the reduced cardiac output seen in many patients with mitral stenosis.

**Acknowledgment**

The authors wish to express their sincere gratitude to Dr. John B. Hickam for his invaluable advice and criticism. They also wish to thank Mrs. Sonia Chang and Mrs. Garnette Miles for their technical assistance and Dr. James A. Norton, Jr., for his aid in the statistical analysis of the data.

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*Circulation, Volume XXXIV, September 1966*

Subacute Bacterial Endocarditis—Ruptured Chordae Tendineae,
Dr. Peacock 1860

He ascribed his illness to cold taken four months before, and stated that though he had served for ten years in the army, he had not had rheumatism or any serious illness or accident before his present attack. ... A systolic murmur was audible in all parts of the chest, but it was decidedly most distinct and harsh at the apex, and thence towards the left axilla. At the level of the third cartilage it was very indistinct, and it was inaudible at the upper part of the sternum. It was feebly heard at the lower angle of the left scapula. There was also a distinct musical murmur heard at the base of the heart, with the diastole, but this was inaudible at the apex. ... The heart hypertrophied and dilated and weighed twenty-one ounces avoird. The free edge of the posterior semilunar valve was retroverted, and hung loosely into the cavity of the left ventricle ... , so as to have allowed regurgitation from the aorta. The under surface of the retroverted portion had a few fibrinous fringes adhering to it. The free fold of the mitral valve was perfectly loose, owing to the destruction of several of the chordae tendineae, and must have allowed of free regurgitation from the ventricle into the left auricle. The fragments of the destroyed chordae tendineae were covered by vegetations, as was also the under surface of the valve itself.—Tr Path Soc Lond 12: 59, 1861.
Evaluation of the Left Ventricle in Patients with Mitral Stenosis
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and EDWARD F. STEINMETZ

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