Mechanical and Myocardial Factors in Rheumatic Heart Disease with Mitral Stenosis

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Sixteen patients with rheumatic heart disease and pure mitral stenosis, studied by cardiac catheterization, are presented to illustrate the relative importance of mitral block and myocardial insufficiency in this disease. Analysis of hemodynamic data permitted a separation of those patients with predominantly mechanical mitral block from those in whom myocardial insufficiency appeared to be the predominant lesion. The importance of recognizing the existence of the latter group is emphasized, since commissurotomy will not be of benefit in such cases.

Circulatory dysfunction in rheumatic heart disease has long been recognized as springing from at least two main sources, the mechanical difficulties imposed by valvular lesions and the insufficiency of the myocardium itself. This insufficiency may result from longstanding strain inflicted on the cardiac muscle by altered valvular function or, independently of mechanical cause, may occur consequent to intrinsic myocardial damage from the rheumatic process. Our understanding of cardiac function in rheumatic patients would be increased if one could separate the mechanical from the myocardial components in order to investigate further the disability these subjects experience. The surgical approach to rheumatic mitral stenosis, attacking as it does only the mechanical features of valvular lesions, affords an opportunity to study this problem. Furthermore, if it can be shown that myocardial insufficiency exists as a separate dysfunction, it behooves the physician and surgeon to be certain that the prospective candidate for mitral commissurotomy is suffering from a predominantly mechanical lesion, namely block at the mitral valve, and not chiefly from myocardial insufficiency. A number of reports have been published discussing the element of mechanical valvular block and its hemodynamic characteristics. In some instances block has been clearly demonstrated by postcommissurotomy hemodynamic studies. On the other hand, the predominance of myocardial insufficiency in patients with mitral stenosis and particularly the fact that it can occur without the existence of any significant mechanical block at the mitral valve, has not been stressed from a hemodynamic point of view.

As a result of studies, using the cardiac catheterization technique, made in a series of patients with mitral stenosis who were being considered for mitral surgery, two groups of individuals have emerged whose clinical and physiologic findings have led us to the conclusion that in one the predominant difficulty was due to mechanical block, while in the other it resulted from myocardial insufficiency. The substance of this report is concerned with a presentation and differentiation of the findings in these two groups. It should be emphasized that these individuals were specifically selected to illustrate the predominance of one or the other dysfunction, although it was recognized that in any one rheumatic subject these dysfunctions may coexist equally or in varying proportions. Our complete experience with mitral surgery will be reported more fully in a following communication.

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<table>
<thead>
<tr>
<th>Case, Sex, Age</th>
<th>Diagnosis</th>
<th>Time</th>
<th>Cardiac Output (l/min.)</th>
<th>Cardiac Index (L/min. m² BSA)</th>
<th>Heart Rate (beats/min.)</th>
<th>Oxygen Consumption (cc/min. m² BSA)</th>
<th>R.Q.</th>
<th>AV Diff (vol. %)</th>
<th>cont. (vol. %)</th>
<th>sat (%)</th>
<th>Systemic arterial s/0, m</th>
<th>Pulmonary arterial s/0, m</th>
<th>Right ventricle (%)</th>
<th>PV (cc/M² BSA)</th>
<th>PV (cc/M² BSA)</th>
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<td>Normal</td>
<td>EH.MS. (T.I.) AF. IIC.</td>
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<td>60</td>
<td>124</td>
<td>0.83</td>
<td>6.4</td>
<td>16.6</td>
<td>93</td>
<td>120/70, 90</td>
<td>5/4/30, 37</td>
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<td>Post-op. 1 month</td>
<td>4.00</td>
<td>2.35</td>
<td>52</td>
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<td>0.87</td>
<td>5.3</td>
<td>15.9</td>
<td>96</td>
<td>84/56, 66</td>
<td>61/27, 39</td>
<td>3</td>
<td>2838</td>
<td>2010</td>
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<td>#595. AB., M. 28 yrs.</td>
<td>EH.MS. AI. NSR. IIC.</td>
<td>Post-op. 1 month</td>
<td>4.98</td>
<td>2.98</td>
<td>74</td>
<td>134</td>
<td>0.89</td>
<td>4.5</td>
<td>17.4</td>
<td>97</td>
<td>120/90, 83</td>
<td>45/18, 32</td>
<td>10</td>
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<td>#613. HI., F. 21 yrs.</td>
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<td>Pre-op. 1 month</td>
<td>4.21</td>
<td>2.61</td>
<td>62</td>
<td>138</td>
<td>0.88</td>
<td>5.3</td>
<td>15.8</td>
<td>95</td>
<td>120/80, 71</td>
<td>58/32, 46</td>
<td>8</td>
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<td>#663. LS., F. 37 yrs.</td>
<td>EH.MS. NSR. IIC.</td>
<td>Post-op. 1 month</td>
<td>3.90</td>
<td>2.80</td>
<td>85</td>
<td>137</td>
<td>0.86</td>
<td>4.8</td>
<td>15.2</td>
<td>95</td>
<td>120/71, 90</td>
<td>55/33, 34</td>
<td>6</td>
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<td>#703. MA., F. 83 yrs.</td>
<td>EH.MS. (G-S) NSR. IIC.</td>
<td>Post-op. 6 months</td>
<td>3.80</td>
<td>2.38</td>
<td>66</td>
<td>111</td>
<td>0.87</td>
<td>4.9</td>
<td>16.5</td>
<td>98</td>
<td>151/76, 104</td>
<td>87/57, 58</td>
<td>4</td>
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<td>1585</td>
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<td>#707. CC., M. 30 yrs.</td>
<td>EH.MS. (G-S) SA. IIC.</td>
<td>Post-op. 1st Study (R)</td>
<td>4.18</td>
<td>2.71</td>
<td>78</td>
<td>135</td>
<td>0.93</td>
<td>5.0</td>
<td>15.4</td>
<td>91</td>
<td>128/71, 90</td>
<td>77/32, 45</td>
<td>5</td>
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<td>EH.MS. SA. IIC.</td>
<td>Pre-op. (E)</td>
<td>4.11</td>
<td>2.21</td>
<td>84</td>
<td>124</td>
<td>0.79</td>
<td>5.6</td>
<td>17.6</td>
<td>98</td>
<td>120/71, 90</td>
<td>50/18, 30</td>
<td>6</td>
<td>2570</td>
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<td>#713. EW., F. 28 yrs.</td>
<td>EH.MS. (G-S) NSR. IIC.</td>
<td>Pre-op. (R)</td>
<td>4.08</td>
<td>2.28</td>
<td>82</td>
<td>130</td>
<td>0.80</td>
<td>5.7</td>
<td>17.2</td>
<td>93</td>
<td>113/71, 88</td>
<td>38/18, 23</td>
<td>5</td>
<td>2742</td>
<td>1723</td>
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<td>#715. PD., M. 42 yrs.</td>
<td>EH.MS. (G-S) NSR. IIC.</td>
<td>Post-op. 1½ months</td>
<td>5.40</td>
<td>3.14</td>
<td>79</td>
<td>138</td>
<td>0.83</td>
<td>4.4</td>
<td>15.1</td>
<td>96</td>
<td>132/71, 90</td>
<td>38/18, 23</td>
<td>5</td>
<td>2742</td>
<td>1722</td>
</tr>
</tbody>
</table>

* Read on right atrial curve 
† Not in basal state 
MBBSA = per square meter of body surface area 
AV. diff. = arteriovenous oxygen difference 
RQ = respiratory quotient 
TBV = total blood volume 
PV = plasma volume 
Hcrit = hematocrit 
(MR) = mitral stenosis 
(MR) = mitral regurgitation 
(E) = during exercise 
(S) = systolic 
(T.I.) = tricuspid insufficiency 
SA = sinus arrhythmia 
EH = enlarged heart 
AF = atrial fibrillation 
STENOSIS (R) = pulmonary (E) = aortic 

**Table 1.—Physiologic Data in 8 Patients with Rheumatic Mitral Stenosis and Predominantly Mitral Block**

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**Note:** The table provides detailed physiologic data for patients with rheumatic mitral stenosis, including measurements of cardiac output, cardiac index, heart rate, oxygen consumption, and various hemodynamic parameters such as arterial blood oxygen, pressures in mm Hg, etc. The data are presented in a tabular format with columns for different parameters and time points, allowing for a comprehensive analysis of the patients' conditions.
MATERIAL AND METHODS

Sixteen patients with mitral stenosis have been selected for presentation in this report. Their diagnoses can be found in tables 1 and 2 and conform to accepted criteria. Three of the subjects, (cases 703, 707, 713, table 1) with a basal diastolic murmur were thought to have pulmonic incompetence with the murmur of Graham-Steele (indicated by bracketed letters G–S) because of the severity of pulmonary hypertension and the absence of left ventricular enlargement or confirmatory evidence of aortic regurgitation. In another three patients (cases 595, table 1; 591, 699, table 2) with a basal diastolic murmur, aortic insufficiency was diag-

TABLE 2—Physiologic Data in 8 Patients with Rheumatic Mitral Stenosis andPredominantly Myocardial Insufficiency

<table>
<thead>
<tr>
<th>Case, Sex, Age</th>
<th>Diagnosis</th>
<th>Time</th>
<th>Cardiac Output (L/min)</th>
<th>Heart Rate</th>
<th>Stroke Volume</th>
<th>Arterial Blood Oxygen</th>
<th>Right AV Diff. (vol %)</th>
<th>R.Q.</th>
<th>Saturation (%)</th>
<th>Pressures in mm Hg</th>
<th>TRV (cc/m² BSA)</th>
<th>PV (cc/m² BSA)</th>
<th>Hg Hct. (%)</th>
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<td>Normal</td>
<td>EH. MS. Al. AF. IIC.</td>
<td>Pre-op. (R)</td>
<td>5.08</td>
<td>2.90</td>
<td>67</td>
<td>130</td>
<td>0.77</td>
<td>4.8</td>
<td>15.3</td>
<td>96</td>
<td>120/70, 90</td>
<td>&lt;30/10, 15</td>
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<tr>
<td></td>
<td></td>
<td>1st study (E)</td>
<td>—</td>
<td>—</td>
<td>80</td>
<td>190</td>
<td>0.77</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>137/76, 96</td>
<td>39/22, 28</td>
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<td></td>
<td></td>
<td>2 weeks later (E)</td>
<td>5.33</td>
<td>3.04</td>
<td>69</td>
<td>140</td>
<td>0.85</td>
<td>4.6</td>
<td>16.0</td>
<td>95</td>
<td>118/71, 87</td>
<td>28/13, 18</td>
<td>2</td>
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<tr>
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<td></td>
<td>Post-op. 6 months (E)</td>
<td>5.67</td>
<td>3.24</td>
<td>96</td>
<td>194</td>
<td>0.87</td>
<td>6.0</td>
<td>16.0</td>
<td>94</td>
<td>125/77, 93</td>
<td>31/16, 23</td>
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<td>#099. PB., M. 27 yrs.</td>
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<td>1st study in CHF (R)</td>
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<td>1.68</td>
<td>76</td>
<td>117</td>
<td>0.81</td>
<td>7.0</td>
<td>16.1</td>
<td>91</td>
<td>128/90, 106</td>
<td>32/22, 26</td>
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<td>6 weeks later (R)</td>
<td>3.44</td>
<td>2.08</td>
<td>67</td>
<td>110</td>
<td>0.80</td>
<td>5.3</td>
<td>17.4</td>
<td>99</td>
<td>136/67, 93</td>
<td>27/12, 18</td>
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<td>18 months later (E)</td>
<td>3.42</td>
<td>1.98</td>
<td>64</td>
<td>105</td>
<td>0.79</td>
<td>5.3</td>
<td>15.9</td>
<td>97</td>
<td>129/63, 86</td>
<td>27/16, 20</td>
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<td>Post-op. 1 month (E)</td>
<td>3.52</td>
<td>1.85</td>
<td>54</td>
<td>122</td>
<td>0.81</td>
<td>6.6</td>
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<td>97</td>
<td>106/56, 75</td>
<td>28/16, 20</td>
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<td>1st study (R)</td>
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<td>1.68</td>
<td>76</td>
<td>117</td>
<td>0.81</td>
<td>7.0</td>
<td>16.1</td>
<td>91</td>
<td>128/90, 106</td>
<td>32/22, 26</td>
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<td>6 weeks later (R)</td>
<td>3.44</td>
<td>2.08</td>
<td>67</td>
<td>110</td>
<td>0.80</td>
<td>5.3</td>
<td>17.4</td>
<td>99</td>
<td>136/67, 93</td>
<td>27/12, 18</td>
<td>2</td>
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<td>18 months later (E)</td>
<td>3.42</td>
<td>1.98</td>
<td>64</td>
<td>105</td>
<td>0.79</td>
<td>5.3</td>
<td>15.9</td>
<td>97</td>
<td>129/63, 86</td>
<td>27/16, 20</td>
<td>4</td>
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<td>Post-op. 1 month (E)</td>
<td>3.52</td>
<td>1.85</td>
<td>54</td>
<td>122</td>
<td>0.81</td>
<td>6.6</td>
<td>18.3</td>
<td>97</td>
<td>106/56, 75</td>
<td>28/16, 20</td>
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<td>3.46</td>
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<td>149</td>
<td>0.84</td>
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<td>15.3</td>
<td>97</td>
<td>131/70, 100</td>
<td>28/12, 21</td>
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<td>21 months later (E)</td>
<td>4.98</td>
<td>2.96</td>
<td>58</td>
<td>136</td>
<td>0.92</td>
<td>4.6</td>
<td>17.6</td>
<td>94</td>
<td>102/64, 81</td>
<td>26/11, 16</td>
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<td>159</td>
<td>0.90</td>
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<td>20/13, 16</td>
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<td>21 months later (E)</td>
<td>4.98</td>
<td>2.96</td>
<td>58</td>
<td>136</td>
<td>0.92</td>
<td>4.6</td>
<td>17.6</td>
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<td>102/64, 81</td>
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<td>3.54</td>
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<td>159</td>
<td>0.90</td>
<td>4.5</td>
<td>16.4</td>
<td>98</td>
<td>113/69, 89</td>
<td>20/13, 16</td>
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<td></td>
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<td>2 weeks later (R)</td>
<td>4.98</td>
<td>2.96</td>
<td>58</td>
<td>136</td>
<td>0.92</td>
<td>4.6</td>
<td>17.6</td>
<td>94</td>
<td>102/64, 81</td>
<td>26/11, 16</td>
<td>4</td>
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<tr>
<td>#555. EB., F. 29 yrs.</td>
<td>EH. MS. (T.L.) AF. Healed SBE. IIC.</td>
<td>1st study in CHF (R)</td>
<td>2.64</td>
<td>1.42</td>
<td>91</td>
<td>122</td>
<td>0.98</td>
<td>8.6</td>
<td>16.2</td>
<td>96</td>
<td>104/70, 82</td>
<td>56/37, 44</td>
<td>11</td>
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<td>2 weeks later (E)</td>
<td>3.94</td>
<td>2.15</td>
<td>86</td>
<td>108</td>
<td>0.93</td>
<td>5.0</td>
<td>14.9</td>
<td>92</td>
<td>100/59, 81</td>
<td>40/16, 25</td>
<td>5</td>
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</tbody>
</table>

For abbreviations see end of table 1.
Healed SBE = healed subacute bacterial endocarditis.
nosed in the absence of left ventricular enlargement and a widened pulse pressure because pulmonary hypertension was either absent or only moderate at rest. The diagnosis of tricuspid insufficiency was made from right atrial pressure tracings in two patients (case 618, table 1; case 555, table 2) although the clinical evidence of the lesion was not present. This lesion is indicated in tables 1 and 2 by the bracketed letters T.I. None of the patients had any evidence whatsoever of an apical systolic murmur, nor did any have left ventricular enlargement by fluoroscopy or roentgenogram.

Prior to their evaluation by cardiac catheterization, all of these patients were closely observed in order to rule out, as far as possible, not only such complications as active rheumatic carditis, subacute bacterial endocarditis, and intractable heart failure, but also, to establish the best possible status obtainable by medical means. This period of observation in the hospital lasted two to three weeks in most instances.

After careful clinical appraisal of these individuals over a considerable period of time, physiologic studies were undertaken, using the method of cardiac catheterization as described in previous reports.8-10 Observations were made at rest, during exercise, during acute digitalization and, in some subjects, before and after commissurotomy. The preoperative measurements were obtained shortly before surgery and in no instance was there thought to be any clinical change in the patient between the time of study and commissurotomy.

All of the eight subjects considered to have mitral block (table 1) were subjected to mitral commissurotomy. Two of the subjects with myocardial insufficiency also had mitral valvular surgery (table 2).

Interpretation of results. In order to determine the significance of any hemodynamic change produced in these subjects following acute digitalization or exercise, the criteria previously presented in detail have been employed.8-10 To summarize these, any change in cardiac output greater than 9 per cent of the control value is considered significant, provided that in the studies made at rest, the oxygen consumption and the respiratory gas exchange ratio (respiratory quotient) do not vary by more than 18 cc. per square meter of body surface area, or 0.11, respectively. During exercise the measurement of cardiac output was considered valid only if respiratory gas exchange ratio was equal to or greater than the resting value, thus indicating that a steady state of exercise had been reached when the measurements involved in the Fick equation were made. As indicated in a previous communication,1 a rough estimate of the normal response to the mild type of exercise employed in these subjects is at least a 650 cc. rise in blood flow per 100 cc. increase in oxygen consumption. Changes in lesser circulation blood pressures were considered significant only if they exceeded 5 mm. Hg, provided obviously that no change greater than this was noted during the control period in any individual patient. Since variations in heart rate are known to influence these pressures, especially in patients with mitral stenosis, particular care was taken to secure at least four control readings in order to obtain a good sampling. Although only one representative value is given in tables 1 and 2, the range of values can be seen in the illustrations where all pressure values are depicted.

A further word should be added here with regard to the interpretation of pressure tracings in patients with atrial fibrillation. With the marked variation in cycle length which may be encountered in this arrhythmia, there may be a greater variation in the amplitude of pulse waves than is found with a regular ventricular rate. For this reason in order to obtain representative pressure values, it is often necessary to analyse and average as many as 20 consecutive beats which may include three or four complete respiratory cycles instead of the usual two. This is true not only in single studies but is crucial when comparison of several separate studies is made.

The criteria which have just been reviewed are applicable to changes which may be encountered over a few hours' time. The magnitude of variations in pressure and blood flow which may occur in a normal individual in studies repeated at intervals of several months has not been determined and hence specific criteria for significant change are lacking. Recognizing the fact that long term variations are unknown, recourse was made to the only criteria available, namely, those previously described. One would appear justified in employing these since serial studies were also made each time in a steady, postabsorptive basal state as judged by oxygen consumption and respiratory gas exchange ratios. Although there may be changes in the patient's body weight between the several examinations, percentile changes in systemic blood flow have been calculated, using the cardiac indices.

Results

The 16 patients comprising this report have been divided into one group of eight (group I) in whom mechanical block at the mitral valve was considered the primary difficulty and a second group of eight (group II) in whom the clinical and physiologic findings suggest myocardial insufficiency as the predominant dysfunction. All 16 subjects had cardiovascular symptoms of varying severity. Indeed 12 of the 16 had had documented evidence of pulmonary or peripheral congestion at some time in their course. One particular historical feature differ-
entiated one group from the other. Group I had almost constant and often progressive disability, group II, while completely incapacitated by their symptoms occasionally, nonetheless had relatively asymptomatic periods when they could resume their occupations. Comparison of other clinical features was less rewarding. The heart size, as shown in figures 1 and 2, was not always distinctive; although the largest hearts were found in group II, one can find hearts with slight to moderate enlargement in both groups. There were more patients with atrial fibrillation in group II, and none of these individuals showed the electrocardiographic pattern of right ventricular hypertrophy, although two (cases 699, 591, table 2) had a small double peak of R in V1. On the other hand, four patients in group I (cases 567, 703, 714, 713, table 1) showed an electrocardiographic pattern of right ventricular hypertrophy, as indicated by a large R wave and a late intrinsicoid deflection in the right precordial leads. Brief clinical summaries will be given together with the hemodynamic data of each of the subjects.

Fig. 1. Roentgenograms (posteroanterior 6-foot films) of patients in group I are in the same order as patients listed in table 1. J.C. = case 567, A.B. = case 595, H.I. = case 618, L.S. = case 663, M.A. = case 703, C.C. = case 707, R.D. = case 714, E.W. = case 713.
**Group I.—Mitral Block**

In this group the hemodynamic findings were fairly uniform and support the concept that the major difficulty in circulatory function resulted from a mechanical block at the mitral valve which could be attacked surgically.

The first patient (case 567, table 1) had experienced exertional dyspnea for 12 years. For the year and a half prior to his first study, despite a more sedentary existence, dyspnea was present on the mildest exertion and orthopnea appeared. Small hemoptyses had occurred in the past three years. Digitalization and mercurial diuretics did not alter his symptoms although they rid him of mild edema. He was free of pulmonary rales and signs of peripheral congestion when first catheterized. His x-ray films, as well as those of each patient in group I appears in figure 1. Preoperatively a low cardiac output, severe pulmonary hypertension and a normal right ventricular diastolic pressure were present. The blood volume was slightly increased. As can be seen in figure 3, exercise produced an interesting combination of changes: a marked further rise in pulmonary hypertension with an insignificant rise in cardiac output (370 cc. increase in blood flow per 100 cc. increase in oxygen consumption). The right ventricular diastolic pressure reached abnormal levels during exertion. Commis-
Surotomy effected a widening of the orifice as judged by the exploring finger and the postoperative course was not remarkable. One month later his clinical improvement was dramatic. He was comfortable on ward activity, orthopnea was absent and he was able to climb two flights of stairs without dyspnea. Maintenance Digoxin was continued in this interval, no changes in physical findings could be detected and the x-ray film of the heart had not altered appreciably. Postoperative studies at rest at this time (table 1 and fig. 3) demonstrated a fall of 15 mm. Hg in the pulmonary artery pressures. There was an increase in cardiac output (+21 per cent) and a fall in heart rate. The response to exercise differed from the preoperative study in that the entire level of pulmonary hypertension was lower and the right ventricular diastolic pressure did not rise above normal although the pulmonary blood flow was slightly larger than occurred on effort during the first study. The increase in blood flow of 434 cc. per 100 cc. of oxygen consumption, however, was not significantly different than preoperatively. After a period of convalescence, the patient returned to full time employment and remained asymptomatic. Maintenance Digoxin was continued. Physical examination, electrocardiograms and chest x-ray films were unchanged over the next 10 months. At the time of the third study, 11 months after operation, the most significant finding at rest was a further fall in pulmonary artery pulse pressure. On exertion the pulmonary artery pressures did not exceed those of the previous studies although the cardiac output, and hence the pulmonary blood flow, was considerably greater than had been encountered on previous studies, with a value of 635 cc. per 100 cc. increase in oxygen consumption. Right ventricular diastolic pressure rose on exertion. The brachial artery pressures were higher at rest and on exercise than preoperatively. There was little variation in blood volume throughout the whole period of observation. This patient is now over four and one-fourth years post commissurotomy and is fully employed.

The second patient in table 1, (case 505) had had two years of exertional dyspnea, cough and easy fatigue. During the month prior to admission he had several large hemoptyses associated with pulmonary edema and was by then dyspneic on slightest activity despite digitalis and mercurial diuretics. In the preoperative study, as seen in table 1 and figure 4, he had moderate pulmonary

![Fig. 3. Graphic representation of the hemodynamic findings in patient 567 (J. C.). For discussion see text. In this, and all subsequent figures, triangles = stroke volume; squares = cardiac index; target dots = heart rate; closed circles = pulmonary artery systolic and diastolic, and right ventricular end diastolic pressures; open circles = brachial artery systolic and diastolic pressures; cross marks = mean pressure. The normal values are plotted in the first vertical column.](http://circ.ahajournals.org/lookup/doi/10.1161/01.CIR.53.3.537)
hypertension and a low cardiac output at rest. Exertion called forth severe pulmonary hypertension and a high ventricular diastolic pressure with what was probably a normal increase in blood flow (658 cc. per 100 cc. increase in oxygen consumption) although the level reached was probably not normal. At operation the anterolateral commissure of the mitral valve was split by the finger. He was asymptomatic on ward activity but his physical signs, chest x-ray film and electrocardiogram were not altered at the time of the second cardiac catheterization, which was done one month postoperatively. Despite a larger systemic and pulmonary blood flow, the pulmonary artery pressures both at rest and during exercise were lower than preoperatively. The right ventricular diastolic still rose to abnormal levels on exercise. This patient was able to resume full activity, return to work and was asymptomatic at the time of the third study. This was performed 12 months after operation and the level of cardiac output at rest and during exercise remained essentially the same as was found preoperatively with a 500 cc. increase in blood flow per 100 cc. increase in oxygen consumption. However, he now had normal pulmonary artery pressures both at rest and during exercise. The ventricular diastolic pressure no longer increased abnormally during effort. Blood volume did not vary appreciably throughout the observation period. It is now four years since surgery and the patient no longer receives Digoxin.

The third patient (case 618, table 1) had 12 years of dyspnea and orthopnea for which she had been digitalized. In contrast to the previous patients, she had also had a large pulmonary infarction and cerebral emboli, three and two years ago. At the time of the first study exertional dyspnea and hepatomegaly were the chief clinical findings and physiologic measurements demonstrated a slightly reduced cardiac output, severe pulmonary hypertension and an elevated right ventricular diastolic pressure and blood volume. Pressure tracings in the right atrium were characteristic of tricuspid insufficiency. One month after commissurotomy, the patient had no dyspnea on ward activity. Hepatomegaly persisted and the other physical findings, the cardiac silhouette by roentgenogram and the electrocardiogram were the same as preoperatively. The significant hemodynamic changes at this time consisted of some reduction in pulmonary hypertension and blood volume, while the cardiac output and the end diastolic pressure in the right ventricle as read from the right atrial tracing, were not appreciably altered. Evidence of tricuspid insufficiency persisted in the right atrial tracings. Three and a
third years have elapsed since surgery and she is free of cardiac symptoms. She is unemployed for psychiatric reasons.

The fourth patient (case 663) of this group, a nurse and a diabetic, had known mitral stenosis for 10 years but was asymptomatic until she developed pulmonary edema two years before admission. Symptoms of pulmonary congestion grew progressively worse, forcing her to cease work. Digitalis, mercurial diuretics and rest did not bring appreciable symptomatic relief. She was, however, free of rales, hepatomegaly and edema when first studied. The preoperative measurements (table 1) revealed a normal cardiac output, blood volume and right ventricular diastolic pressure at rest, and moderate pulmonary artery hypertension. She had a bout of atrial flutter postoperatively which yielded to drug therapy. The physiologic measurements made one month after commissurotomy are not comparable to the preoperative values as the patient had considerable anxiety as can be seen by the elevated resting oxygen consumption (table 1). In view of this fact it is not clear whether in addition pressure values are comparable or not. Following operation she continued taking digitalis, but did not require diuretics. She returned to work in a busy premature infant unit after six months, and had no symptoms at all on any ordinary activities, but on one occasion did become severely dyspneic and noted blood streaked sputum while pulling oxygen tanks along the ward. The study made one year postoperatively showed a fall in pulmonary artery pressure at rest and no change in cardiac output or blood volume. On exercise these pulmonary artery pressures rose and the blood flow did not increase. It is now two and three-quarter years since operation and she has been free of cardiac symptoms, although her diabetes has been difficult to control.

The fifth subject (case 703), a concert pianist, sought relief of her symptoms by surgical means because, after seven years of increasing dyspnea which did not yield to digitalis and diuretics, she found herself unable even to play the piano without discomfort. Ankle edema was occasionally present. Free of physical signs of congestion when catheterized, she was found to have very severe pulmonary hypertension, increased blood volume and arterial blood oxygen saturation, the latter suggesting some fluid or other barrier to gas exchange (table 1). For technical reasons adequate data for calculation of cardiac output were not obtained. Her postoperative course was complicated by two episodes of paroxysmal atrial fibrillation which reverted to a normal sinus rhythm with quinidine. Six months postoperatively she was living a normal existence without any symptoms and required no digitalis. Cardiac catheterization at this time showed a fall of 13 mm. Hg in pulmonary artery mean pressure, a reduction in blood volume and return to a normal arterial oxygen saturation. The cardiac output was less than normal. In the two years following operation her marked clinical improvement has been maintained, permitting full-time work.

The sixth patient (case 707), a salesman, had been forced to a desk job by fatigue and exertional dyspnea increasing over a four-year period. Two years prior to admission he was in bed for five months with acute rheumatic fever. Four months before the catheterization studies he had a series of multiple systemic emboli—to brain, abdominal organs and arteries of the lower extremities. He had not been digitalized when first studied (table 1, fig. 5) and had no rales or evidence of peripheral congestion. Acute digitalization produced no alteration in the reduced cardiac output or the normal right ventricular diastolic pressure (fig. 5). There was no significant change in the level of the markedly elevated pulmonary artery diastolic and mean pressures but the systolic pressure did fall from 77 to 69 mm. Hg during the 40 minutes of study together with a slowing in heart rate. Although it was felt that this decrease in systolic pressure was due to reduction in heart rate, a second preoperative study was made after two weeks of maintenance Digoxin, in order to learn if there was any further diminution in pulmonary artery pressures after full digitalization. The clinical status remained the same and the study demonstrated that there was no further significant change in cardiac output and right heart pressures. No element of left heart failure then was apparent and hence the medication was withdrawn. The abnormal response to exercise during this second study was similar to that seen in the first patient, namely, an almost fixed cardiac output (with a blood flow increase of 206 cc. per 100 cc. increase in oxygen consumption) and the further aggravation of pulmonary hypertension with evidence of strain in right ventricular performance as manifested by a marked elevation of end diastolic pressure. Operation was carried out in this patient and at one month postoperatively he was strikingly improved despite a lack of change in physical signs, heart size or electrocardiogram. Physiologic studies at this time (fig. 5) revealed a marked fall in pulmonary artery pressures at rest with only a small rise during exercise. The cardiac output which at rest was the same as preoperatively now increased satisfactorily during exercise (968 cc. per 100 cc. increase in oxygen consumption). There was really no change in plasma volume in this patient, although the red cell mass and hemoglobin were higher postoperatively. It is now 20 months since operation and the patient no longer has any disability and has returned to full activity, including some sports.
Fifth patient (case 714), a bank accountant, had had acute rheumatic fever at the age of 10 years. On somewhat curtailed activity he had remained asymptomatic until one year before admission when progressive dyspnea appeared. He had one attack of paroxysmal nocturnal dyspnea three months before entry, and when first seen he was almost too short of breath to speak. Cough and blood-streaked sputum occasionally accompanied severe dyspnea. He was not on any cardiac medication when first evaluated. As can be seen in figure 6, acute digitalization produced a reduction in heart rate accompanied by a slight decrease in the markedly elevated pulmonary artery pressures, but the low cardiac output fell slightly (-11 per cent) indicating the absence of heart failure, at least at rest, since other studies have indicated that in the presence of failure Digoxin always calls forth an increase in cardiac output. The response to exercise was observed during the same catheterization study, and the leg motion was begun one hour after digitalization. The cardiac output rose subnormally (410 cc. rise in blood flow per 100 cc. of increase in oxygen consumption). A marked increase in heart rate and in all lesser circulation pressures were other features noted in the exertion period. No further digitalis was given. The patient was operated upon and six weeks later had noted considerable improvement. The cardiac output at rest was now 42 per cent higher than preoperatively and there had been a sharp fall in systolic pulmonary artery pressure with little change in the diastolic or mean. The response to exercise demonstrated even more clearly the improvement in function as the pulmonary artery pressures were not nearly so high despite the fact that the pulmonary blood flow was a liter greater than it had been during exercise in the preoperative study. However the blood flow increase per 100 cc. increase in oxygen consumption was still subnormal (513 cc.). The postoperative plasma volume was slightly increased over the normal preoperative value. At 19 months after surgery, his improvement continues despite increasing activity.

The eighth patient (case 713) was a 28 year old housewife whose cardiac history began five years before admission. At that time she had an hemopty- sis in the sixth month of a second pregnancy. She was asymptomatic for a year after this until she had a pulmonary infarction immediately after delivery of her third child. Subsequently exertional dyspnea appeared and grew worse during the next
three years. For the year prior to admission, because of this symptom, she was bed ridden either at home or in the hospital. A third pulmonary infarct precipitated mild right heart failure two months before admission. Digitalis had been tried on several occasions but since it never afforded her any relief of dyspnea, had been discontinued. Right heart failure had disappeared at the time of study. The values obtained at rest revealed severe pulmonary hypertension and a somewhat reduced cardiac output (table 1). Commissurotomy was performed and following a period of convalescence her functional capacity was strikingly improved. It is now 16 months since surgery and the patient is living a normal existence, having been able to resume care of her three small children as well as carry a full time job as a sales girl. She is asymptomatic without medication. This improvement was fully substantiated by the physiologic studies made at one year after commissurotomy. At this time pulmonary arterial and right ventricular pressures as well as the cardiac output were well within normal limits (table 1), the latter 35 per cent higher than before operation. On exercise the blood flow increased 790 cc. per 100 cc. increase in oxygen consumption which is a normal response and mild pulmonary hypertension appeared during the exertion. The plasma volume was normal and was less than preoperatively. The electrocardiographic changes were also quite striking as she no longer has evidence of a right hypertrophy pattern or of right axis deviation. There also has been a definite decrease in heart size.

In summary, all of these eight patients with mitral stenosis had a history of progressive cardiac disability and a disordered hemodynamic function characterized by moderate to severe pulmonary hypertension and either a low or normal cardiac output with a diminished blood flow response during leg exercise. At surgery the mitral orifice of each one was found to be narrowed, as judged by the fact that it would not admit the tip of the index finger. Following commissurotomy and widening of the orifice to a two-finger width, the pulmonary hypertension decreased to a greater or lesser degree both at rest and during exercise in every patient. This reduction was greatest in the systolic phase, hence producing
a fall in pulse pressure toward normal, despite the fact that in no instance was there a decrease in cardiac output or any significant difference in heart rate. In fact in three patients (cases 567, 714, 713) the blood flow was higher than preoperatively at rest and during exercise, while in a fourth (case 707), although the resting value after surgery was unchanged, it reached much higher values during exercise. This decrease in pulmonary artery pulse pressure suggests that blood has been mobilized out of this area postoperatively thus reducing the distention of the pulmonary vascular bed. Since left ventricular failure was excluded as far as possible by prolonged intensive medical therapy and specifically by acute digitalization studies in two patients, and since there was no striking or definitive reduction in total blood or plasma volumes in any subject after surgery, the reduction in pulmonary hypertension shown by this group of patients with mitral stenosis may be safely attributed to commissurotomy and was taken as evidence that mitral valvular block had been present and was at least partially relieved by surgery.

One point of interest should be stressed regarding the postoperative level of the pulmonary artery pressures. In only one of the eight subjects was this pressure restored to normal at rest and during exercise. One must assume that one of two causes, or both, is implicated in this residual hypertension, namely, the presence of some persistence of mitral block due to insufficient widening of the orifice by the surgeon, or due to pulmonary vascular atherosclerotic lesions. The behavior of these latter lesions, and indeed the contribution which they made in absolute terms to the level of pulmonary hypertension in any one patient, is not yet known. That the increased resistance due to these sclerotic lesions may be slowly reversible is suggested by the gradual decrease in pulmonary pressures over one year's time in one patient (case 595, table 1). However it could also be attributed to further mobilization of pulmonary blood volume. Thus it is conceivable that further improvement may occur in such individuals as time goes on, so long as there is no further mitral valve obstruction due to reactivation of the rheumatic valvulitis.

Group II.—Myocardial Insufficiency

In this group the hemodynamic findings at rest were uniform; during exercise, however, two patterns of response were found in the pulmonary circulation. After analysis these data support the concept that the major difficulty in these patients was not the result of mitral block, especially since valvulotomy proved ineffective in two instances, and could best be ascribed to imperfect myocardial function.

The first patient (case 699, table 2) in group II was a young handyman with a known heart murmur for nine years. His initial x-ray film, as well as those of each of the other patients in group II, appears in figure 2. He had his first cardiac symptoms, hemoptysis and cough, two years before the initial catheterization study. These symptoms cleared completely after digitalization and he returned to work, became relatively asymptomatic and hence eventually stopped taking the drug. Three months before study he again noted the onset of these first symptoms along with progressive dyspnea, and had severe right heart failure and atrial fibrillation. He was admitted to another hospital where digitalis again relieved the congestive failure and quinidine failed to revert the atrial fibrillation to sinus rhythm. Digitalis was continued after he was ambulatory and there were no signs of pulmonary or peripheral congestion when he was first evaluated. On this first catheterization the cardiac output was normal at rest, the pulmonary artery pressures were slightly increased with a greater rise in diastolic than systolic level, and the plasma volume was definitely elevated. On exercise the pulmonary hypertension increased somewhat. The mixed venous blood unfortunately clotted thus preventing the measurement of cardiac output during this exertion period. After two weeks of bedrest in the hospital a second catheterization was done. During this interval the only clinical change was a distinct decrease in heart size (fig. 7). On this second evaluation the cardiac output at rest was the same as on the first measurement; but, with the same ventricular rate, there had been a small but definite fall in the pulmonary artery pressures, a fall involving predominantly the diastolic and mean values, and the plasma volume had also decreased. Comparison of the resting hemodynamics in these two preoperative studies (table 2, fig. 8) reveals that the change on medical management is best explained as due to a decrease in left ventricular myocardial failure. This assumption is strengthened by the response to the exercise performance he made on this second study.
Although the patient reached the same level of oxygen consumption during effort on both occasions, the second time, despite a ventricular rate which was even higher than in the first exercise period, there was no significant rise in pulmonary artery pressures as there had been when the mild left ventricular failure still existed. The cardiac output did not increase significantly on exercise (370 cc. per 100 cc. increase in oxygen consumption). It was recognized that this patient's resting hemodynamics and his response to exertion, namely, a fixed cardiac output without rise in pulmonary artery pressures, were quite different from those of patients in group I. The absence of significant pulmonary hypertension particularly was at variance with the usual experience in severe mitral valvular block. Since one could not, in view of our lack of knowledge, be certain if mitral stenosis in its mechanical aspects was responsible for the abortive response in cardiac output during exercise and in view of his history of...
RHEUMATIC HEART DISEASE WITH MITRAL STENOSIS

The second subject (case 695, table 2), a 44 year old woman, had been asymptomatic, save for an attack of rheumatic fever at the age of 16, until four weeks prior to study, when, following ingestion of a large amount of alcohol, she felt the sudden onset of a rapid heart beat. Subsequently, edema and severe right upper quadrant pain were noted. At the time of the first study she had signs of pulmonary congestion, a very large and tender liver, slight ankle edema and atrial flutter with 2:1 A-V response. Physiologically, the cardiac output at rest was quite low (fig. 9), there was moderate elevation of the pulmonary artery diastolic and mean pressures with only slight elevation in systolic and hence a small pulse pressure. The arterial blood showed oxygen unsaturation and the plasma volume was at the upper limits of normal. Acute digitalization with Digoxin produced a rise of 24 per cent in cardiac output and a slight decrease in pulmonary artery diastolic pressure. Following continued digitalization, mercurial diuretics and conversion to sinus rhythm by quinidine, she was recatheterized. At this time, six weeks after the first study, she had no clinical evidence of pulmonary or peripheral congestion, was in normal sinus rhythm and had received no quinidine for 18 days. The physiologic findings (table 2, fig. 9) indicated a resting cardiac output that was 41 per cent higher than the pre-Digoxin cardiac output and 13 per cent higher than the post-Digoxin figure. The pulmonary artery pressures were lower, particularly the diastolic level, and in fact were almost normal. The arterial blood oxygen was fully saturated and the plasma volume had fallen by nearly 300 cc. per square meter of body surface area. Comparison of these two evaluations suggests that congestive heart failure accounted for the findings of the first study, since the decrement in pulmonary artery pressures on the second study, despite a larger pulmonary blood flow, could only be explained as due to relief of left heart failure. On Digoxin she remained free of symptoms for 17 months, save for slight dyspnea on moderate to severe exertion. At this time a bout of flutter-fibrillation precipitated congestive heart failure and dyspnea at rest although she had continued to take Digoxin. Once again conversion to sinus mechanism was accomplished by quinidine and all signs of cardiac failure disappeared. At this time, 18 months after the first study (table 2, fig. 9), she was re-evaluated. Clinically and physiologically she was in the same state as at the time of the second study, with resting hemodynamics which were identical to those obtained at that time. When leg exercise was performed there was little or no rise in cardiac output (350 cc. per 100 cc. increase in oxygen consumption) and after a very slight initial increase at four minutes, there was no real change in pulmonary artery or right ventricular pressures. These hemodynamic observations are totally different from those of group I and hence it must be concluded that this

Fig. 9. Graphic representation of the hemodynamic findings in patient 695 (S.B.). For discussion see text.

repeated disability, this patient was permitted to undergo mitral commissurotomy. The orifice was somewhat narrowed and the edges split easily. For five months after operation, on limited activity he complained of little if any symptoms. Indeed he was in much the same state as preoperatively when he was out of congestive failure with a well-controlled ventricular rate. However, five months after surgery, following an upper respiratory infection, he once more went into congestive failure with increase in heart size, dyspnea, edema and hepatomegaly. After bed rest and diuretics the heart size again decreased but at the time of his third catheterization, when all signs of congestion had disappeared, it was still larger than preoperatively (fig. 7). At this time, six months postoperatively, (table 2, fig. 8) the cardiac output both at rest (−18 per cent) and during exercise was 1 liter lower than preoperatively, the blood flow was still fixed during exercise (196 cc. increase per 100 cc. rise in oxygen consumption), and the plasma volume was increased to the same level as before surgery. The pulmonary artery and right ventricular pressures were the same as on the second study, and were almost normal. They increased only very slightly if at all during exercise, as was the case during the second study. The operative intervention then had produced no improvement in hemodynamic function. Moreover, the level of blood flow was now below normal, probably as a result of the direct and continuing effects of the rheumatic process on the myocardium. It is concluded, therefore, that mitral block was not the primary cause of dysfunction in this man. It is now two years since commissurotomy, and the patient, remaining on digitalis, has not shown any clinical improvement. In fact, he has had to obtain lighter work than he was able to do preoperatively.
woman did not have an important degree of block at the mitral valve. In view of this, it is interesting to note that she had evidence of calcification of this valve by fluoroscopy.

The similarity of the hemodynamics at rest of the pulmonary circulation in these first two members of group II, both when they were in and then out of congestive failure, as well as during exercise, is striking. In view of the lack of improvement after commissurotomy shown by the first (case 699), the second patient was not offered surgery.

Just as these first two subjects in this group could be paired hemodynamically, so the next five patients were alike not only in their resting values but also in response to exercise which was characterized by an increase in pulmonary arterial pressure. They will be discussed individually and are well represented by the following case.

The third patient in group II (case 635, table 2), a 39 year old salesman, knew of cardiac enlargement and a heart murmur for 21 years, but had been asymptomatic until eight years before admission. Since then, easy fatigability was a prominent complaint along with dyspnea, intermittent ankle edema, orthopnea and one episode of hemoptysis despite digitalization. He was free of pulmonary rales, hepatomegaly and edema at his first catheterization. This latter (fig. 10) revealed a very low cardiac output at rest, minimal pulmonary hypertension with a normal right ventricular diastolic pressure and a plasma volume which was increased. On exercise, despite a subnormal rise in cardiac output (425 cc. per 100 cc. of oxygen consumption increase), pulmonary artery pressures were quickly increased to a moderately hypertensive level, in sharp contrast to the first two subjects in this group. This effort was accomplished without right ventricular strain as shown by the diastolic pressure remaining within normal limits. Although the resting pressure in the pulmonary artery was only slightly elevated, the exercise hypertension was disturbing, as it could be ascribed either to some degree of mitral block, which only became important when blood flow increased, or to left ventricular failure which appeared on exertion. Since his symptoms and exercise hypertension could be due to mitral block, commissurotomy was done and the surgeon widened a narrowed valve orifice which would not admit the tip of the index finger. Immediately postoperatively he was difficult to mobilize because of apprehension, hence any symptomatic change, if present, was impossible to appreciate. His physical findings, electrocardiogram and heart size did not alter. The postoperative catheterization at one month (table 2, fig. 10) showed no change over the preoperative performance at rest or during the same degree of exercise, except for some decrease in plasma volume. Three years and three months have passed since surgery and there is no evidence of clinical improvement.

The fourth patient (case 675, table 2), a 41 year old male, had no symptoms until the sudden onset of dyspnea associated with severe weakness, 10 days prior to hospitalization. He denied hemoptysis, chest pain and all other cardiac symptoms. He was in shock, had atrial fibrillation and was in acute respiratory distress on admission, with a liver which was considerably enlarged. The x-ray shadow indicated a large pulmonary infarct. Congestive failure yielded to digitalization and the wedge-shaped shadow in the x-ray film of the lungs disappeared in two weeks. He had been in the hospital nine weeks and free of signs or symptoms for seven weeks, when studied. The cardiac output and blood volume were normal and there was only a slight elevation of the diastolic and mean pulmonary artery pressures at rest with a systolic level within normal range. On exertion the blood flow increased subnormally (428 cc. per 100 cc. increase in oxygen consumption) and moderate pulmonary hypertension appeared without right ventricular strain. It was felt that the episode of failure in this man was associated with pulmonary infarction and the rapid ventricular rate and probably had little to do with significant block at the mitral valve since there was little or no resting pulmonary hypertension. It is possible that the hypertension during exercise resulted from persistent changes in the pulmonary vasculature consequent to the embolization and not from mitral disease.

Fig. 10. Graphic representation of the hemodynamic findings in patient 635 (G.B.). For discussion see text.
The fifth patient (case 591, table 2) of this second group, whose first study was given in a previous report (9), had been a difficult problem to unravel. A 28 year old Puerto Rican laborer, he had had some mild exertional dyspnea for six months before a severe fist fight precipitated pulmonary edema and hemoptysis. After three weeks of bedrest he had no abnormal clinical signs save his murmur. His first catheterization (table 2, fig. 11) indicated a normal cardiac output, an increased plasma volume, and pulmonary artery pressures which were nearly normal at rest and which rose somewhat during exercise and then declined although exertion continued. This effort called forth an increase in blood flow of only 290 cc. per 100 cc. increase in oxygen consumption. This performance did not suggest the same hemodynamic state as was seen in the patients of group I, particularly since a fall in pulmonary pressures as exercise continued has never been seen in our experience in patients with mitral block if exercise remains steady.

The patient was lost to follow-up until 21 months after the first study, during which interval he was asymptomatic. Mild dyspnea had recently recurred and the question of mitral surgery was raised. A second evaluation (table 2, fig. 11) showed no change in resting hemodynamics. In view of these findings it was felt that surgery was not warranted. After another lapse of time, during which he worked as a dishwasher and peddler, mild dyspnea again brought the patient to the clinic. At this time a basal diastolic murmur could be heard in the fourth left intercostal space close to the sternum. A third catheterization, done 33 months after the first (table 2, fig. 11), again revealed no change in dynamics at rest. During exercise, which at this time was carried out at a higher level of oxygen consumption, the cardiac output rose 586 cc. per 100 cc. increase in oxygen consumption, a value which is lower than a normal individual would achieve. Pulmonary pressures again rose and this time remained elevated at a higher plateau during the whole of exercise. Complaints of mild dyspnea and easy fatigability continued for the ensuing 15 months. The liver became slightly enlarged, but was never tender. There were no other objective evidences of systemic or pulmonary congestion. Mercurial diuretics were reported to alleviate dyspnea but did not affect the size of the liver. The enlargement of the latter may well have been related to the patient's excessive alcoholic intake. At the time of the fourth study the heart was slightly larger than noted previously, but in other respect the clinical findings were the same as at the third study. Similarly the hemodynamic findings were unchanged. The appearance of the basal diastolic murmur and the enlarging heart

Fig. 11. Graphic representation of the hemodynamic findings in patient 591 (N. G.). For discussion see text.
suggested that active rheumatic endocarditis and myocarditis were present. For this reason and because resting pulmonary artery pressures were unchanged for four years and because of the surgical experience of the third subject in the group (case 635), no operation was offered this man.

The sixth subject (case 761, table 2) had as her prime complaint palpitations when excited. She had chorea at age 14, and had led a very active life as a swimming instructor and clerk. Six years ago she had had some dizzy spells, was digitalized and told to curtail her activities. As a result of emphasis on her previously unknown heart lesion, the patient became increasingly apprehensive, conscious of palpitations but had no dyspnea. Her physician referred her to the hospital for evaluation in reference to surgery. There were no abnormal signs save the cardiac murmur and abnormal rhythm when she was studied. It was evident during the physiologic evaluation that she had a labile ventricular rate. When she had been at rest for sometime, however, her pulmonary artery pressures were only minimally elevated, blood volume was normal but the cardiac output was low. A subnormal rise in blood flow (257 cc. per 100 cc. increase in oxygen consumption) occasioned a brisk rise in pulmonary artery pressures and a slight fall in systemic artery pressures at a time when the ventricular rate reached 158. Surgery was not felt to be indicated in view of her resting pulmonary artery pressures.

The seventh patient (case 552, table 2), a 52 year old elevator operator, had had several attacks of rheumatic fever until the age of 30, but after digitalization for his first bout of failure at age 36, was able to work steadily until one month before admission, when, on losing his job, he stopped taking digitalis for financial reasons. Within a short while dyspnea, orthopnea and edema returned, and he was hospitalized. After bed rest, all signs of failure receded and the hemodynamics at rest were characterized by minimal pulmonary hypertension, a low cardiac output and an elevated plasma volume. The patient was digitalized and showed no rise in cardiac output in this acute study as described in a previous report. On examination two weeks later there were no circulatory changes from the first catheterization, and, at this time, when the man exercised he developed pulmonary artery and right ventricular diastolic hypertension with a rise in cardiac output which, in the light of our recent experience, must be classed as subnormal (500 cc. per 100 cc. increase in oxygen consumption).

In summary, all of the subjects in group II had moderate to severe cardiac symptoms which were episodic in their occurrence. The last five patients presented (cases 635, 675, 591, 761, 552) all had minimal if any pulmonary hypertension at rest and a rise in these pressures during exercise, in contrast to the first pair of subjects in group II (cases 699, 695) whose resting pulmonary artery pressures were also almost normal but in whom these pressures did not increase significantly on effort. In each of these subgroupings one representative patient was subjected to mitral surgery as a pilot investigation. In neither one was there clinical or physiologic evidence of any improvement in hemodynamic function after commissurotomy. If one can accept the experience encountered in group I as characteristic of mitral block, a state which was expressed hemodynamically by pulmonary hypertension at rest which was aggravated by exertion, and which yielded to valve fracture as attested to not only by clinical improvement, but also by objective measurements of a decrease in lesser circuit pressures postoperatively, then the patients in this second group do not have appreciable mitral valve block. The subnormal response in cardiac output during exercise, be it normal or low at rest, was common to all of these patients and hence does not help in differentiating them.

If one does not accept the deranged dynamics in these individuals in group II as chiefly the result of mitral block, one must attempt a further explanation of their difficulties. All are victims of rheumatic heart disease and hence it is likely that myocardial lesions exist in them as well as valvular cicatrices. Although mitral valvular damage has occurred as indicated by auscultation, there appears to be little hemodynamic evidence of obstruction to blood flow at rest or at the mitral valve in these patients with almost normal lesser circulation pressures. Furthermore in the two patients whose pressures were not increased with exercise, there seemed to be further proof of no impedance to blood leaving the left atrium. In none of these individuals, however, does the cardiac output respond normally to the demands of exertion. This suggests an insufficiency in myocardial performance which is not related to mechanical obstruction within the circulatory channels. Indeed, this insufficiency is probably the major circulatory defect of the patients in group II. In those five subjects in whom pulmonary
hypertension appeared on exertion this insufficiency had progressed to a further stage than in the first two subjects who remained normotensive, and one might even say they had reached the stage of frank left heart congestive failure on effort. In all instances save one (case 552) the effort, however, was not severe enough to produce physiologic evidence of right heart failure, namely, a rise in right ventricular diastolic pressure.

An example of a still more advanced phase of this type of predominantly myocardial insufficiency in a patient with rheumatic heart disease and mitral stenosis is presented in the eighth patient (case 555, table 2) of group II. Her first study was done while she was in right and left sided congestive failure and physiologically demonstrated severe pulmonary hypertension with a high right ventricular diastolic pressure, a low cardiac output and a very large blood volume. Two weeks later, after digitalization, mercurials and bedrest, a very striking decrease in right heart pressures had occurred in association with a rise in cardiac output. This case serves to demonstrate the point that pulmonary hypertension due predominantly to left heart failure exists in some patients with mitral stenosis, and that this hypertension declines in response to medical management. It may well be that after further treatment this patient would have presented the same hemodynamic picture at rest as demonstrated by the first seven patients in group II.

**DISCUSSION**

It has long been an accepted clinical teaching that a block at the mitral valve would produce pulmonary hypertension either with or without anatomic changes in the pulmonary vascular bed. The physiologic resultant of this mechanical interference with flow from the left atrium were eventually established in absolute terms and in addition to confirming the presence of pulmonary hypertension, a low cardiac output was often seen. The latter was also ascribed, at least in part, to impedance to blood flow at the valve. Since mitral stenosis can progress to a stage of blocking egress of atrial blood and hence producing pulmonary hypertension, it is important to learn more about the various stages of mitral block. It has been shown that the mere ausculatory finding of the typical murmur, particularly in the absence of symptoms, does not imply elevation of pulmonary artery pressures. Indeed, there are patients with this murmur and perfectly normal cardiodynamics. It is likely that there are various degrees of obstruction at the valve and that minor ones do not interfere with circulatory performance, at least at the levels of activity which have been measured so far. Perhaps severe taxing exertion may uncover such small obstructions. Once pulmonary hypertension exists at rest in a patient with mitral stenosis and rises with exercise, mitral block can be assumed to have become important. It is recognized, of course, that left ventricular failure can and often does produce pulmonary hypertension independently of any valvular lesion, and this cause for pulmonary hypertension should be ruled out, whenever possible, in the course of evaluating the dynamics of mitral stenosis.

If then, failure of the left heart can be excluded in a patient with mitral stenosis and pulmonary hypertension, it would appear that mitral block exists. The subjects in group I were characterized by this hypertensive dynamic state and were partially relieved of it by surgery as evidenced by the disappearance or decrease in elevated pulmonary artery pressures. Since both symptoms and pulmonary hypertension were ameliorated by surgical dilatation of the valve orifice, it would seem that significant mitral block produces pulmonary hypertension at rest, and, without this hypertension at rest, significant mitral obstruction does not exist. The poor operative result in the second group of patients lends strength to this conclusion.

Furthermore, the totally different dynamics in this second group, namely, absence of pulmonary hypertension but a restricted cardiac output, have pointed out a physiologic state which is certainly abnormal, but which probably springs from intrinsic myocardial insufficiency and not from mechanical cause. It is suggested that the pulmonary hypertension which some of these subjects demonstrate
during exercise is a reflection of this poor myocardial function in that it implies left heart failure during stress, a response which the authors have found in patients with non-valvular cardiac disease. This point is illustrated in figure 12 which depicts the findings at rest and during exercise in a 42 year old woman with hypertensive cardiovascular disease and cardiac enlargement without cardiac symptoms. These observations were made before and three and one-half weeks after digitalization. At rest she had mild pulmonary hypertension with a normal cardiac output and right ventricular diastolic pressure. During exertion there was an inadequate rise in cardiac output and she developed severe pulmonary hypertension. The second study, made when she was fully digitalized, demonstrated no change at rest or during exercise when compared with the findings of the first study. These data indicate that even in the absence of the clinical expressions of left ventricular failure, the hemodynamic state of this patient demonstrated a type of insufficiency of the left ventricular myocardium which persisted in spite of digitalization.

The question could be raised of course, are not all these patients in group II merely examples of a mild degree of stenosis at the valve? In the first two subjects of this group it is impossible to imagine any real valve obstruction in the absence of pulmonary hypertension either at rest or on exertion. Insofar as the five patients who developed pulmonary hypertension only on exercise are concerned, it is difficult to accept such an explanation since it would imply less disability on the part of these subjects than those in group I, and yet, from a clinical point of view, their hearts are as large or larger, there is a higher incidence of atrial fibrillation and the same or greater number of bouts of congestive failure; in summary, they show as much or more clinical disability as the patients in group I with block. Moreover in the two patients of group II in whom the valve waswidened by surgery, there was no physiologic or clinical amelioration. A review of those previous reports which give sufficient detail to permit analysis of data indicates that there is no unequivocal improvement in hemodynamics after surgery in subjects with little or no resting pulmonary hypertension, which lends support to the concept that in the absence of resting pulmonary hypertension, significant mitral block does not exist.

It is evident then that there exists in some patients with symptomatic rheumatic mitral stenosis, a hemodynamic state which, in the absence of outspoken left heart failure, is characterized by little or no pulmonary hypertension and yet is associated with an abnormally low response in cardiac output on exertion. More than half this group of eight had low outputs at rest and one wonders, in view of this fact, whether the level of blood flow in patients with mitral stenosis, at rest and during exercise, is not chiefly an index of myocardial integrity and is much less influenced by the mechanical valvular block than was formerly postulated. In support of this suggestion one notes that only three of the eight patients with successful commissurotomies (group I) had any change in the resting level of cardiac output after surgery.

The importance of differentiating the group with mitral block from that with predomi-
nantly myocardial insufficiency—and the catheterization data appear to make this a feasible differentiation—is obvious when one considers offering surgery to any patient with mitral stenosis. It would seem only logical to insist on a demonstration of pulmonary hypertension in each prospective candidate in order to avoid selecting one with predominantly myocardial insufficiency. It should be recalled that such patients are not rare since this group of eight was culled out of a total of 45 patients with mitral stenosis who were studied physiologically as possible candidates for mitral surgery.

The abnormal dynamic state which is considered to have as its basic mechanism poor myocardial function, is not to be confused with the state of certain other patients with mitral stenosis who also do not have pulmonary artery hypertension. These latter subjects are either asymptomatic entirely or have symptoms which are noncardiac and often are iatrogenic. Studies in these individuals have revealed a normal level of cardiac output and a normal response of this function on exertion.6, 9, 12

The 16 patients discussed in this presentation were selectively chosen and separated into two groups because of the predominance of one or the other basic dysfunction, namely, mechanical obstruction or myocardial insufficiency. It is recognized that in any one individual with rheumatic heart disease there may well be an element of both present, making analysis difficult. Unfortunately there is no way of defining the exact etiology of the limitation of myocardial function in the second group. Rheumatic carditis is certainly likely; but whether this is in the active stage or not cannot be stated. The data demonstrate that this hypodynamic state exists in patients with mitral stenosis who have no evidence of mechanical valve obstruction.

From the data presented in this paper, one can conclude that without proven pulmonary hypertension of moderate to severe degree at rest there is probably little or no important degree of block at the mitral valve. Furthermore, patients with mitral stenosis and little or no hypertension must be carefully and extensively evaluated. In the light of our current knowledge they should not, at least for the present, be subjected to commissurotomy since they may be suffering either from predominantly myocardial insufficiency, a stage herein described, or have no circulatory dysfunction at all.

**SUMMARY AND CONCLUSIONS**

1. Sixteen patients with rheumatic heart disease and pure mitral stenosis who were studied by the cardiac catheterization technic, were selected to demonstrate the relative importance of mitral block and myocardial insufficiency in this disease.

2. Analysis of the dynamics at rest and during exercise has permitted a division of these patients with mitral stenosis into two groups, one with mitral block characterized by pulmonary hypertension of varying degrees and a fixed or subnormal response in cardiac output on exercise, and the other in whom little or no pulmonary hypertension exists but in whom cardiac output does not increase normally on exercise. In the latter group myocardial insufficiency was felt to be the predominant lesion uncomplicated by any important element of mechanical block.

3. The importance of recognizing the existence of a group of rheumatic patients with mitral stenosis and primarily myocardial insufficiency is emphasized since commissurotomy will not produce any improvement in function in such cases.

**SUMMARIO E CONCLUSIONES IN INTERLINGUA**

1. 16 patientes con cardiopathia rheumatica e pura stenosi mitral, qui esseva studiate per medio del technica de catheterisation cardiac, esseva seligite pro demonstrar le importantia comparative de bloco mitral e insufficientia myocardica in iste morbo.

2. Super le base del dynamica a reposo e a exercitio il esseva possibile gruppar iste patientes con stenosis mitral in duo categorias: le prime con bloco mitral characterisate per hypertension pulmonar de varie grados e un fixe o subnormal responsa de rendimento cardiac in observationes a exercitio, e le secunde in qui pauc o nulle hypertension pulmonar existe sed in qui le rendimento cardiac
non mostra le normal augmento post esercizio. In seconde gruppo sos supponeva que insufficientia myocardiac esseva le lesion predominante, non complicate per ulle importante elemento de blocco mechanic.

3. Le importantia de riconoscere le existentia de un gruppo de patientes rheumatic con stenosis mitral e un insufficientia primarmente myocardiac es sublineate proque in tal consensus nulle melioration functional poter esser attingite per medio de commissurotomia.

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Mechanical and Myocardial Factors in Rheumatic Heart Disease with Mitral Stenosis
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