The Hemodynamics of the Left Side of the Heart as Studied by Simultaneous Left Atrial, Left Ventricular, and Aortic Pressures; Particular Reference to Mitral Stenosis

By Eugene Braunwald, M.D., Howard L. Moscovitz, M.D., Salomao S. Amram, M.D., Richard P. Lasser, M.D., Samuel O. Sapin, M.D., Aaron Himmelstein, M.D., Mark M. Ravitch, M.D. and Alvin J. Gordon, M.D.

At operation the hemodynamics of the left side of the heart were studied in six patients without mitral stenosis, and in eight patients with mitral stenosis, by means of simultaneous needle puncture of the left atrium, left ventricle, and aorta. This technic permits analysis of the various phases of the cardiac cycle in normal subjects and in patients with mitral stenosis. The fundamental hemodynamic expression of mitral stenosis is the presence of an elevated left atrioventricular filling pressure gradient, which ranged from 4 to 20 mm Hg, and after valvulotomy fell in relation to the adequacy of the procedure.

Our knowledge of the human cardiac cycle has been retarded by the inability to study completely the hemodynamics of the left side of the heart. The technique of venous catheterization, which has contributed so much to an analysis of events in the right side of the heart of man does not permit the direct study of the left heart chambers except in isolated instances of congenital communications between the right and left cavities.

Previous investigation of the left side of the circulation has been carried out primarily through animal experimentation. The classic studies of Wiggers have defined the various phases of the cardiac cycle, as well as the pressure relationships between the left atrium, left ventricle, and aorta, both in the normal dog and in animals with experimentally produced valvular lesions.

Only sporadic investigations have been performed on the left side of the human heart. Limon and coworkers recorded left atrial, left ventricular and aortic pressure by aortic catheterization. Left atrial pressures have been measured by needle puncture of the left atrium at the operating table, through the bronchoscope, through the chest wall, and by cardiac catheterization in patients with interatrial septal defects. In addition, attempts have been made to estimate left atrial pressures indirectly by the so-called pulmonary capillary method.

Human left ventricular pressures have been obtained by needle puncture at operation, by puncture of the chest wall, and by catheterization of the left ventricle through a needle introduced into the left atrium through the chest wall or through the left main bronchus. Pressures in the human aorta have been measured by retrograde arterial catheterization, by puncture of the chest, and by needle puncture at the operating table, and also by catheterization through a needle introduced into the left atrium through the chest wall.

Simultaneous left atrial and left ventricular pressures in man have been measured by Wynn and associates, by Bouchard and coworkers, and by Venner and Holling. Since these...
pressures were not recorded with manometers of equal sensitivity, they could not be employed directly for calculating time and pressure relationships. Venner and Holling have, however, superimposed and rescaled simultaneous left atrial and left ventricular pressure pulses in three cases of mitral stenosis and three cases of mitral insufficiency.35

We have recently described,36 in preliminary form, a method by which simultaneous left atrial, left ventricular, and aortic pressures are obtained by needle puncture in the open chest, and are recorded with manometers of equal sensitivity with identical baselines. This report summarizes our experience with this technique in 14 patients.

**Material**

The patients studied were all on the ward and private services of The Mount Sinai Hospital.

**Group I—Cardiovascular Normals**

The three patients listed as cardiovascular normals consist of R. C., a 60-year-old white male with a tuberculoma of the left upper lobe; I. W., a 53-year-old white male who had an inoperable carcinoma of the left lower lobe, and O. W., a 16-year-old Negro girl with a mediastinal thymic cyst. None of these patients had any cardiovascular abnormality that could be elicited by a thorough history and physical examination, a 12-lead electrocardiogram, chest x-ray films and in one case (O. W.) by angiocardiography.

**Group II—Other Patients Without Mitral Stenosis**

This group consists of three patients. In two of these, studies were performed immediately after ligation of a patent ductus arteriosus. J. N. was 17 years old, while G. W. was 8. Both presented the classical clinical picture of uncomplicated patent ductus arteriosus. Both patients had normal electrocardiograms, and no cardiac enlargement by radiographic examination. Cardiac catheterization was performed on G. W., and normal pressures were found in the right side of the heart. The third patient in this group, A. B., was a 70-year-old male on whom the simultaneous pressures were obtained immediately after a left pneumonectomy for bronchogenic carcinoma had been performed. His past history, preoperative physical examination, electrocardiogram, and radiographic examination were all negative for cardiovascular disease. However, at the time of the study, the patient had a transient episode of nodal rhythm.

**Group III—Patients with Mitral Stenosis**

This group consists of eight patients with mitral stenosis who were studied several minutes before and after mitral valvulotomy. The pertinent findings related to these patients are summarized in table 1. All patients were considered on clinical grounds to have predominant mitral stenosis, and this was borne out by the findings at operation. One patient

**Table 1.—Cardiac Catheterization Data and Operative Findings in Patients with Mitral Stenosis (Group III)**

<table>
<thead>
<tr>
<th>Initials, Age and Sex</th>
<th>Functional and Therapeutic Classification</th>
<th>Cardiac Rhythm</th>
<th>Cardiac Catheterization Study (Pressures in mm. Hg)</th>
<th>Operative Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Right Ventricular systolic/ end dist.</td>
<td>Pulmonary systolic/ diast.</td>
</tr>
<tr>
<td>E. H. 28 years, female</td>
<td>III C NSR</td>
<td>Rest</td>
<td>68/8</td>
<td>70/35</td>
</tr>
<tr>
<td>I. M. 34 years, female</td>
<td>II B NSR</td>
<td>Rest</td>
<td>55/5</td>
<td>53/23</td>
</tr>
<tr>
<td>S. W. 45 years, male</td>
<td>III C NSR</td>
<td>Rest</td>
<td>41/8</td>
<td>42/17</td>
</tr>
<tr>
<td>S. P. 37 years, female</td>
<td>III D AF</td>
<td>Rest</td>
<td>60/5</td>
<td>73/28</td>
</tr>
<tr>
<td>E. S. 31 years, female</td>
<td>II C AF</td>
<td>Rest</td>
<td>30/6</td>
<td>43/19</td>
</tr>
<tr>
<td>F. S. 47 years, female</td>
<td>III C AF</td>
<td>Rest</td>
<td>100/8</td>
<td>52/25</td>
</tr>
<tr>
<td>M. G. 42 years, female</td>
<td>II B AF</td>
<td>Rest</td>
<td>31/6</td>
<td>45/20</td>
</tr>
<tr>
<td>S. K. 42 years, female</td>
<td>III C AF</td>
<td>No Catheterization Performed</td>
<td>—</td>
<td>2.9</td>
</tr>
</tbody>
</table>

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METHOD

The patients all had endotracheal intubation and were maintained under positive pressure respiration. The anesthetic agent was ether, occasionally supplemented by small amounts of cyclopropane. At the time the pressures were obtained, the left lung was partially collapsed, although it had been fully inflated for a short period immediately before.

The atrial needle was inserted through the auricular appendage, or through its stump after mitral valvulotomy. The ventricular needle was thrust perpendicularly through the lateral wall of the left ventricle, avoiding any small branches of the coronary vessels on the surface. The aorta was punctured at the level of the left subclavian artery in 11 patients, just above the aortic valve in two patients (L. M. and F. S.) and in the descending thoracic aorta in one (J. W.). The points of all three needles were estimated to be at almost identical levels.

Twenty-gage short-beveled needles were used. Specially made, thick-walled, vinyl plastic tubing with "luer-lok" adaptors was employed. This tubing is 4 feet long, with an outside diameter of 0.262 inches and a bore of 0.073 inches. Three Statham P23A pressure transducers were mounted in tandem at identical levels. The zero point was the midtrial level as determined with the chest open.

A four-channel cathode ray photographic recording system was utilized, embodying three pressure channels and an electrocardiogram.† The paper speed was either 25 or 50 mm. per second, usually the latter. The characteristics of the recorder permitted direct superimposition of the three pressure tracings so that baselines and sensitivities could be made identical. Response of the system to pressure was linear to better than 1 per cent over the entire range.

Lead II of the electrocardiogram was recorded in all instances. Pressure measurements were averaged for a minimum of 15 beats in tracings with sinus rhythm, and for a minimum of 30 beats in tracings with atrial fibrillation. Mean pressures were determined by planimetric integration.

The pressure pulses obtained by the method described here do not reproduce the cardiodynamic events as faithfully as do those which have been obtained in animal experiments, with manometers thrust directly into the cardiac chambers. On the other hand, our method probably interferes less with the action of the beating heart. Because of the obvious dangers to the patient, the problems of sterilization, and limitations of space in the operative field, it became necessary to utilize a system with a needle attached by a connecting tube to the pressure gauge. Such a system makes possible the recording of pressure pulses with less likelihood of artifacts than the technique of right heart catheterization in which catheter movement may impose considerable distortion. However, some artifact produced by motion imparted to the needle and the connecting tubing by the beating heart has been unavoidable.

Other factors which have occasionally interfered with the accurate registration of the pressure pulse are the presence of thrombus material in the left atrium, and the damping effect of the apposition of the ventricular wall to the orifice of the needle.

It is apparent that general anesthesia, positive pressure respiration, and a partially collapsed lung will have influenced to a certain extent the pressure pulses obtained. Wiggers‡ has analyzed in dogs the changes in cardiodynamics, relatively minor, produced by the open chest. The most important of these are a prolongation of the isometric relaxation period, averaging 0.02 second, and greater variations in the relative duration of the maximum and reduced ejection phases.

Analysis of our curves indicates occasional minor inconsistencies. These may be attributed to difficulties in leveling the strain gauges, and slight inequalities in the levels of the three needle orifices. The divergence of the left ventricular and aortic pressure curves during ejection and protodiastole may be explained by the fact that the aortic needle was usually unavoidably introduced at some distance from the aortic valve. This could lead to a delay in the inscription of the aortic pressure pulse, as well as a minor modification of the contour occasioned by the transformation of the pulse wave as it traveled along the aorta.

RESULTS AND ANALYSIS

Simultaneous needle puncture of the human left atrium, left ventricle and aorta appears to be both feasible and safe. We have now performed this procedure in 14 patients without any complication. In seven of these patients it was carried out both before and after mitral valvulotomy. No significant bleeding from any of the puncture sites has taken place and the only arrhythmia noted has been the regular occurrence of several ventricular premature contractions as the needle pierced the left ventricular wall. The experience of other ob-


† Manufactured by Electronics for Medicine, Inc., New York.

‡ Manufactured by U. S. Catheter and Instrument Co., Glens Falls, N. Y.
HEMODYNAMICS OF LEFT SIDE OF HEART

### Table 2.—Pressure Measurements (mm. Hg) in the Left Atrium, Left Ventricle, and Aorta of Patients Without Mitral Stenosis (Groups 1 and 2)

<table>
<thead>
<tr>
<th>Patient and Diagnosis</th>
<th>Left Atrial Mean</th>
<th>Left Atrial Mean Diastolic</th>
<th>Left Atrial 'a' Wave</th>
<th>Left Atrial 'c' Wave</th>
<th>Left Atrial 'v' Wave</th>
<th>Left Atrio-ventricular Filling Gradient</th>
<th>Left Ventricular Systolic</th>
<th>Left Ventricular Mean Diastolic</th>
<th>Left Ventricular End Diastolic</th>
<th>Aortic Systolic</th>
<th>Aortic Diastolic</th>
<th>Aortic Mean</th>
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<tbody>
<tr>
<td>R. C. cardiovascular normal</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>88</td>
<td>3</td>
<td>8</td>
<td>14</td>
<td>84</td>
<td>44</td>
<td>61</td>
</tr>
<tr>
<td>I. W. cardiovascular normal</td>
<td>8</td>
<td>9</td>
<td>12</td>
<td>12</td>
<td>14</td>
<td>0</td>
<td>121</td>
<td>8</td>
<td>14</td>
<td>128</td>
<td>83</td>
<td>91</td>
</tr>
<tr>
<td>O. W. cardiovascular normal</td>
<td>7</td>
<td>7</td>
<td>8</td>
<td>8</td>
<td>11</td>
<td>1</td>
<td>79</td>
<td>6</td>
<td>10</td>
<td>77</td>
<td>49</td>
<td>61</td>
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</tbody>
</table>

**Group I**

<table>
<thead>
<tr>
<th>Patient and Diagnosis</th>
<th>Left Atrial Mean</th>
<th>Left Atrial Mean Diastolic</th>
<th>Left Atrial 'a' Wave</th>
<th>Left Atrial 'c' Wave</th>
<th>Left Atrial 'v' Wave</th>
<th>Left Atrio-ventricular Filling Gradient</th>
<th>Left Ventricular Systolic</th>
<th>Left Ventricular Mean Diastolic</th>
<th>Left Ventricular End Diastolic</th>
<th>Aortic Systolic</th>
<th>Aortic Diastolic</th>
<th>Aortic Mean</th>
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<tbody>
<tr>
<td>J. N. after ligation patent ductus arteriosus</td>
<td>4</td>
<td>4</td>
<td>6</td>
<td>6</td>
<td>7</td>
<td>0</td>
<td>79</td>
<td>4</td>
<td>4</td>
<td>81</td>
<td>37</td>
<td>49</td>
</tr>
<tr>
<td>G. W. after ligation patent ductus arteriosus</td>
<td>10</td>
<td>10</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>0</td>
<td>83</td>
<td>10</td>
<td>12</td>
<td>83</td>
<td>61</td>
<td>70</td>
</tr>
<tr>
<td>A. B. after pneumonectomy (nodal rhythm)</td>
<td>8</td>
<td>6</td>
<td>17</td>
<td>9</td>
<td>0</td>
<td>91</td>
<td>6</td>
<td>8</td>
<td>94</td>
<td>94</td>
<td>57</td>
<td>73</td>
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</table>

**Group II**

### Table 3.—Pressure Measurements (mm. Hg) in the Left Atrium, Left Ventricle and Aorta of Patients with Mitral Stenosis (Group 3) Before and After Valvulotomy

<table>
<thead>
<tr>
<th>Patient</th>
<th>Left Atrial Mean</th>
<th>Left Atrial Mean Diastolic</th>
<th>Left Atrial 'a' Wave</th>
<th>Left Atrial 'c' Wave</th>
<th>Left Atrial 'v' Wave</th>
<th>Left Atrio-ventricular Filling Gradient</th>
<th>Left Ventricular Systolic</th>
<th>Left Ventricular Mean Diastolic</th>
<th>Left Ventricular End Diastolic</th>
<th>Aortic Systolic</th>
<th>Aortic Diastolic</th>
<th>Aortic Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>E. H.</td>
<td>29</td>
<td>28</td>
<td>28</td>
<td>34</td>
<td>36</td>
<td>19</td>
<td>76</td>
<td>9</td>
<td>8</td>
<td>76</td>
<td>53</td>
<td>63</td>
</tr>
<tr>
<td>Before valvulotomy</td>
<td>9</td>
<td>7</td>
<td>10</td>
<td>11</td>
<td>11</td>
<td>3</td>
<td>84</td>
<td>4</td>
<td>7</td>
<td>85</td>
<td>53</td>
<td>68</td>
</tr>
<tr>
<td>After valvulotomy</td>
<td>26</td>
<td>24</td>
<td>23</td>
<td>25</td>
<td>29</td>
<td>14</td>
<td>94</td>
<td>12</td>
<td>15</td>
<td>95</td>
<td>58</td>
<td>81</td>
</tr>
<tr>
<td>I. M.</td>
<td>21</td>
<td>19</td>
<td>19</td>
<td>23</td>
<td>29</td>
<td>8</td>
<td>95</td>
<td>12</td>
<td>12</td>
<td>99</td>
<td>65</td>
<td>89</td>
</tr>
<tr>
<td>Before valvulotomy</td>
<td>24</td>
<td>23</td>
<td>23</td>
<td>28</td>
<td>32</td>
<td>20</td>
<td>84</td>
<td>3</td>
<td>2</td>
<td>85</td>
<td>51</td>
<td>65</td>
</tr>
<tr>
<td>After valvulotomy</td>
<td>26</td>
<td>19</td>
<td>19</td>
<td>26</td>
<td>37</td>
<td>14</td>
<td>92</td>
<td>5</td>
<td>9</td>
<td>96</td>
<td>61</td>
<td>77</td>
</tr>
<tr>
<td>E. S.</td>
<td>24</td>
<td>23</td>
<td>atrial fibrillation</td>
<td>27</td>
<td>27</td>
<td>11</td>
<td>85</td>
<td>12</td>
<td>10</td>
<td>83</td>
<td>58</td>
<td>70</td>
</tr>
<tr>
<td>Before valvulotomy</td>
<td>20</td>
<td>16</td>
<td>atrial fibrillation</td>
<td>23</td>
<td>23</td>
<td>0</td>
<td>108</td>
<td>16</td>
<td>15</td>
<td>108</td>
<td>71</td>
<td>87</td>
</tr>
<tr>
<td>After valvulotomy</td>
<td>11</td>
<td>8</td>
<td>atrial fibrillation</td>
<td>12</td>
<td>12</td>
<td>4</td>
<td>65</td>
<td>4</td>
<td>4</td>
<td>65</td>
<td>49</td>
<td>54</td>
</tr>
<tr>
<td>S. K.</td>
<td>11</td>
<td>7</td>
<td>atrial fibrillation</td>
<td>13</td>
<td>12</td>
<td>1</td>
<td>86</td>
<td>6</td>
<td>4</td>
<td>86</td>
<td>52</td>
<td>65</td>
</tr>
<tr>
<td>Before valvulotomy</td>
<td>13</td>
<td>12</td>
<td>atrial fibrillation</td>
<td>17</td>
<td>17</td>
<td>5</td>
<td>91</td>
<td>7</td>
<td>9</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>After valvulotomy</td>
<td>7</td>
<td>6</td>
<td>atrial fibrillation</td>
<td>—</td>
<td>—</td>
<td>0</td>
<td>92</td>
<td>6</td>
<td>8</td>
<td>92</td>
<td>52</td>
<td>68</td>
</tr>
<tr>
<td>M. G.</td>
<td>28</td>
<td>27</td>
<td>atrial fibrillation</td>
<td>34</td>
<td>35</td>
<td>17</td>
<td>91</td>
<td>10</td>
<td>9</td>
<td>87</td>
<td>60</td>
<td>72</td>
</tr>
<tr>
<td>Before valvulotomy</td>
<td>18</td>
<td>15</td>
<td>atrial fibrillation</td>
<td>23</td>
<td>25</td>
<td>8</td>
<td>95</td>
<td>7</td>
<td>8</td>
<td>95</td>
<td>60</td>
<td>75</td>
</tr>
<tr>
<td>F. S.</td>
<td>29</td>
<td>28</td>
<td>atrial fibrillation</td>
<td>30</td>
<td>31</td>
<td>18</td>
<td>109</td>
<td>10</td>
<td>10</td>
<td>113</td>
<td>68</td>
<td>100</td>
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</tbody>
</table>
servers who have performed direct puncture of the heart chambers at the time of cardiac surgery confirms that this procedure is without danger. 8-10, 21-23, 32, 35, 36

Detailed pressure measurements are presented in tables 2 and 3. Representative tracings are shown in figures 1 through 5.

Particular attention should be directed to the following pressure values: In the patients without mitral stenosis (groups I and II), the left atrial mean pressure ranged between 4 and 10 mm Hg, the mean left ventricular diastolic pressure between 3 and 10 mm Hg, and the mean left atrioventricular filling pressure gradient between 0 and 1 mm Hg. In the patients with mitral stenosis (group III), the left atrial mean pressure measured between 11 and 29 mm Hg, the mean left ventricular diastolic pressure between 3 and 12 mm Hg, while the mean left atrioventricular filling gradient ranged between 4 and 20 mm Hg before valvulotomy. Following mitral valvulotomy this pressure gradient fell significantly in every instance to values between 0 and 14 mm Hg.

The Normal Cardiac Cycle

The cardiac cycle of the left side of the normal heart is depicted in figure 1. The definitions and symbols given to the various phases of the cardiac cycle by Wiggers 4 will be adhered to in this discussion.

Ventricular contraction begins at 1 and virtually simultaneously mitral valve closure occurs, as indicated by the crossover of the atrial and ventricular curves. The c wave in the left atrium begins at this time. This point marks the onset of the left ventricular isometric contraction period, which consists of a slow phase followed by a rapid phase of pressure rise. The isometric contraction period ends at 2, the moment of aortic valve opening, as indicated by the onset of the aortic pressure rise. The period of maximum ejection (2 to 3) lasts until the peak of the aortic pressure pulse (3) and is followed by the period of reduced ejection (3 to 4) which is completed at the beginning of the incisura (4), a point sometimes difficult to identify on our tracings. This marks the end of ejection and of systole. During protodiastole (4 to 5) the pressures in the ventricle and aorta continue to decline. This phase terminates at the moment of aortic valve closure (5), i.e., at the bottom of the incisura of the aortic pressure curve. Isometric relaxation (5 to 6) then follows and ventricular filling begins at the time the mitral valve reopens, which is indicated by the crossing of the atrial and ventricular pressure curves (6).

The period of rapid early filling, i.e., the diastolic inflow period, is marked by a continuous decline in the atrial pressure curve. Diastasis, or the period of slowed ventricular filling, begins when the atrial pressure begins to rise during diastole (7). The onset of the wave produced by atrial contraction marks the end of diastasis (8). The dynamic interval of atrial systole lasts until the peak of the atrial contraction wave (9), while the inflow phase which follows ends at the onset of ventricular isometric contraction (10) and completes the cardiac cycle.

The Cardiac Cycle in Mitral Stenosis

In patients with mitral stenosis the sequence of events differs in several important respects (fig. 2), and will of course vary somewhat when atrial fibrillation occurs. It has already been noted that in the presence of a normal mitral valve the crossing of the left atrial and left ventricular curves, the onset of the atrial c wave, and presumably the moment of mitral valve closure, all occur immediately after the onset of ventricular contraction. In contrast, in patients with mitral stenosis, since the left atrial pressure at the end of diastole is often significantly higher than the left ventricular pressure, the crossing of the left atrial and left ventricular curves and the onset of the atrial c wave are delayed until the left ventricular pressure rises to that of the left atrial. It is apparent, therefore, that the left ventricular isometric contraction period in mitral stenosis consists of two distinct phases. The initial phase, which actually may not be truly isometric, begins with ventricular contraction and lasts until the crossover of the left atrial
and left ventricular pressure curves (X). It has not yet been established that this moment, (X), of crossing of the left atrial and ventricular curves in mitral stenosis represents the moment of closure of the stenotic mitral valve since closure could be delayed by the rigidity of the cusps.

Because of the presence of a positive pressure gradient between the left atrium and left ventricle at the beginning of ventricular contraction it is possible that the paradox exists in which the ventricular chamber fills while its wall is contracting. If this hypothesis is correct, left ventricular filling in mitral stenosis is not strictly limited to diastole, and the term "diastolic filling period" should not be employed. The final phase of isometric contraction begins with the crossing of the left atrial and left ventricular curves, and ends at the moment of aortic valve opening (2).

At the completion of isometric relaxation, that is, at the moment the mitral valve opens,
there emerge other points of difference between the normal heart and that with mitral stenosis. Thereafter, in the normal, the left atrial and left ventricular pressure curves virtually coincide. Despite the fact that no measurable gradient appears to be present from the examination of some of our curves, a very small gradient must exist during diastole in order for blood to flow across the normal mitral valvular orifice. In contrast, in mitral stenosis after the mitral valve opens, the left atrial and left ventricular pressures diverge; the left ventricular pressure continues to fall while the left atrial pressure remains elevated, producing a distinct and easily measurable pressure gradient. This pressure gradient between the left atrium and left ventricle is the fundamental hemodynamic expression of mitral stenosis. It results in a slight shortening of the isometric relaxation phase and permits an earlier onset of mitral valvular opening and, therefore, of ventricular filling. It is thus ap-
Fig. 3. Pressure recordings in a patient before and after mitral valvulotomy. Note the drop in the left atrioventricular filling pressure gradient following valvulotomy. The third beat in the upper tracing represents a "frustrate" ventricular contraction. The second beat in the lower tracing demonstrates that with prolongation of diastole the filling pressure gradient falls progressively.
parent that the time available for blood to flow into the left ventricle is increased by small increments at either end of the ventricular filling period.

A further difference between the normals and the patients with mitral stenosis whom we have studied is the absence of diastasis in the latter. The period of diastasis, as mentioned above, is that phase of diastole which is characterized by a rising atrial pressure before atrial contraction. Thus all of the left atrial pressure curves before valvulotomy were characterized by a continuous decline in pressure during diastole until atrial contraction.

**Mitrail Stenosis with Abnormal Rhythms**

In patients with atrial fibrillation or other arrhythmias producing diastolic periods of varying durations, the magnitude of the left atrioventricular pressure gradient at the end of diastole varies inversely with the duration of the preceding diastole (figs. 3 & 4). A longer diastolic period presumably allows more effective emptying of the left atrium.

Among the other features of interest in the patients with arrhythmias are the following: the dynamics of "frustrate" ventricular contractions are well demonstrated in figure 3. The third beat in the upper tracing, a ventricular premature contraction deliberately induced by tapping the ventricle with a hemostat, does not raise the ventricular pressure to the level of the aortic pressure. Consequently, the aortic valves do not open, and the aortic pressure continues to fall uninterruptedly. The entire period between mitral valve closure and opening is, therefore, an isometric one. There is no ejection phase for the ventricular premature contraction, isometric relaxation following isometric contraction immediately. The presence in such a cycle of an atrial c wave, a finding we have confirmed in other cases, is further evidence that this wave is a function of ventricular contraction alone, and not related to the aortic pressure pulse.

In both A. B., a patient without mitral stenosis and, I. M., a patient with mitral stenosis, nodal beats were characterized in the atrial tracing by a greater pressure rise at the onset of ventricular contraction, presumably the result of summation of the a and e waves.

**Changes Following Mitrail Valvulotomy**

Just as the presence of a filling pressure gradient between the left atrium and ventricle is the most significant finding in mitral stenosis, so does the abolition of this gradient constitute the chief object of mitral surgery. However, changes in the filling pressure gradient brought about by valvulotomy must also be analyzed in the light of possible variation in the amount and character of the blood flow across the mitral valve. All other factors remaining unchanged, the left atrioventricular filling pressure gradient is dependent not only on the size of the mitral orifice, but also on the mitral valvular flow rate at the time the gradient is measured. If the mitral valvular flow rate increases, the pressure gradient required to force this additional volume of blood across the stenotic mitral orifice must likewise increase. The value of the knowledge of the alterations in the mitral valvular flow rate in evaluating the significance of changes in the left atrioventricular filling pressure gradient following mitral valvulotomy is, therefore, apparent. It is possible to estimate the direc-
Fig. 5. Pressure recordings in a patient before and after mitral valvulotomy. Note that the elevated left atrioventricular filling pressure gradient (shaded in black in the third beat of the upper figure) has been completely abolished following the procedure in spite of the fact that the left atrial pressure remained elevated. Paper speed is 50 mm. per second in the upper, 25 mm. per second in the lower tracing.
tion and degree of change in cardiac output by applying Swan and associates\textsuperscript{38} modification of the pressure pulse method to the aortic pulses obtained from our patients before and after valvulotomy. We are unwilling to draw any final conclusions from such calculations until the validity of the method has been firmly established for mitral commissurotomy with its numerous and profound hemodynamic alterations.

Flow across a narrowed orifice, such as a stenotic mitral valve, is believed to be turbulent. The pressure gradient would then be expected to be directly proportional to the square of the flow, and relatively small changes in flow could produce significant alterations in the left atrioventricular filling pressure gradient. It is possible, however, that following surgical correction of the stenotic mitral valve the turbulent flow may be converted to laminar flow, and the pressure gradient would then vary with flow in a simple linear fashion.

If the postvalvulotomy left atrioventricular filling gradient is 0 or very close to 0 (as in patients S. K., E. S. (fig. 5) and M. G.), then the obstruction to blood flow offered by the mitral valve cannot be significant, no matter what the mitral valve flow or the heart rate may be. If there is a significant fall in the gradient, but this does not fall to or close to 0 and there is no fall in cardiac output or in heart rate, this fall in gradient may be related to the relief of the mitral stenosis.

It is apparent that the left atrioventricular filling gradient is a function of both the left atrial and left ventricular diastolic pressures. Following mitral valvulotomy, significant elevations in the left ventricular diastolic pressures were noted in several of the patients. This may represent a temporary effect on the left ventricle before it has adjusted itself to the altered dynamics produced by valvulotomy. In any event, it is apparent that the magnitude of the left atrial pressure does not clearly reflect the magnitude of the left atrioventricular pressure gradient, and that changes in the left atrial pressure following commissurotomy do not necessarily indicate identical changes in the left atrioventricular filling pressure gradient. For example, in E. S. (fig. 5), the left atrial mean pressure fell from 24 to 20 mm. Hg after valvulotomy, while the corresponding left atrial mean diastolic pressure fell from 23 to 16 mm. Hg. Since the left ventricular mean diastolic pressure rose from 12 to 16 mm. Hg, the left atrioventricular filling pressure gradient fell from 11 to 0 mm. Hg. Had the left atrial pressure alone been obtained, one could not have determined that the pressure gradient had been completely abolished by valvulotomy since the postvalvulotomy mean left atrial pressure of 20 mm. Hg and left atrial mean diastolic pressure of 16 mm. Hg are still elevated.

**Summary**

The hemodynamic events of the left side of the heart have been studied in detail in 14 patients. Needles were introduced into the left atrium, left ventricle, and aorta through the open chest, and the pressures recorded simultaneously on identical baselines at identical sensitivities.

In the six patients without mitral stenosis, the left ventricular mean diastolic pressure ranged between 3 and 10 mm. Hg. The mean left atrioventricular filling pressure gradient in these patients measured between 0 and 1 mm. Hg.

The elevated left atrioventricular filling pressure gradient is the fundamental hemodynamic expression of mitral stenosis, and its abolition constitutes the chief object of mitral surgery. In the eight patients with mitral stenosis this gradient ranged from 4 to 20 mm. Hg and following valvulotomy fell significantly in every instance, being almost entirely abolished in three patients.

The technique employed in these studies appears to be safe and makes possible the registration of three simultaneous pressure pulses with relatively little distortion.

**Summario in Interlingua**

Le hemodynamica del corde sinistre esseva studiate in detalia in 14 patientes subjicite a interventiones chirurgic. Quando le thorace habeva esseva aperiite, agulias esseva introducite a in le atri sinistre, a in le ventriculo, e a in le aorta, e le pressiones in iste tres locos esseva
registrazione simultaneamente per medio di manometri del medesimo grado di sensibilità con identiche linee di base.

Le gruppo studiato includeva 6 pazienti che non soffrivano di stenosi mitrale. In tali casi la valore medio del pressione diastolica sinistro-ventricolare variava inter 3 e 10 mm Hg; le valori medio del gradiente atrio-ventricolare sinistro di pressione replenitori mesurava inter 0 e 1 mm Hg.

Un'elevar gradiente atrio-ventricolare sinistro di pressione replenitori es la expression hemodynamic fundamental de stenosis mitral. Su abolition es le obiettivo principale del chirurgo mitral. In le 8 paziente con stenosis mitral, iste gradiente variava inter 4 e 20 mm Hg. Post valvulotomia illo se abassava significativamente in onne casi; in 3 casi illo esesva quasi integremente abolite.

Le tecnica usate in iste studio pare non riscoso. Illo rende possibile le registration simultanee de tres pressione con un distortion che es relativamente negligibile.

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The Hemodynamics of the Left Side of the Heart as Studied by Simultaneous Left Atrial, Left Ventricular, and Aortic Pressures; Particular Reference to Mitral Stenosis

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