The Effects of the Valsalva-like Maneuver upon the Circulation in Normal Individuals and Patients with Mitral Stenosis

By H. Goldberg, M.D., E. I. Elsberg, M.D., and I. N. Katz, M.D.

A Valsalva-like maneuver was performed during cardiac catheterization by a normal group of individuals and a group of patients with acoustic evidence of mitral stenosis. The latter group was subdivided according to its response to the maneuver. The mild group, like the normal group, showed an "overshoot" of blood pressure and a bradycardia in the post-straining period, whereas the severe mitral stenosis group did not. The relatively undisturbed dynamics of the mild mitral stenosis group, in contrast to the marked disturbance in the severe group, was felt to be due to the amount of obstruction at the mitral valve. Additional data from the cardiac catheterizations of these patients is presented to substantiate this concept. This simple maneuver may prove to be a bedside method for the evaluation of the dynamic significance of mitral stenosis.

The dynamic effects of mitral stenosis have been the subject of many investigations. The circulatory disturbances produced by it have long been known, chiefly through animal experimentation. Recently, data accumulated by means of right heart catheterization have confirmed and expanded these observations. Clinically, patients with mitral stenosis may be seen with little, if any, changes in their cardiovascular systems. With the possibility of surgical intervention, the need of assaying the magnitude of the stenosis has become obvious recently. This was recognized some years ago, when it was pointed out that the mitral valve area must be reduced at least 50 per cent in animals before its diverse effects upon the systemic circulation become evident. Attempts have been made in man by elaborate formulas to arrive at a similar judgment. The simple method of measuring the stenotic index suggested by this department for pulmonary stenosis may also be applied to mitral stenosis.

In the course of a recent investigation of the relationship of respiration to cardiovascular dynamics, we studied the effects of a Valsalva-like maneuver upon the circulation. The response in patients with dynamically significant mitral stenosis was distinctly different from the response in patients with lesions of little or no dynamic significance and in normal individuals. These observations suggest that this Valsalva-like maneuver with certain simple measurements might be a useful adjunct in establishing the dynamic significance of a deformed mitral valve. The present report bears on this promising lead.

Material

The individuals comprising this study were selected from the Cardiac Clinic and Medical Wards of the Michael Reese Hospital. A complete history and physical examination was taken on each patient and examination by fluoroscopy, roentgenograms and electrocardiograms was included.

The individuals were divided into three groups: normal, mild mitral stenosis and severe mitral stenosis. The significant data are assembled in table 1.

1. Normal. Each of the three individuals in this group, cases 1, 2 and 3, 22, 26 and 34 years of age respectively, was considered to have a normal cardio-respiratory system.

2. Mitral Stenosis (Mild), Group 1. The three patients of this group had acoustic evidence of mitral stenosis. However, their resting pulmonary artery pressures, their response to exercise and to the Valsalva-like maneuver, showed that the mitral lesion had little effect on the cardiodynamics (table 1). These patients, cases 4, 5 and 6, 19, 30 and 35 years of age respectively, had known mitral lesions for one and one-half to five years. There were no known rheumatic episodes in childhood, though case

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Aided by a grant from the National Heart Institute (H 218C).
6 had joint symptoms one year prior to cardiac catheterization. Case 5 had a mitral valvulotomy several months before the postoperative catheterization reported here. Case 4 was completely symptom free, cases 5 and 6 showed dyspnea only on prolonged exertion. (In case 5 there was a considerable improvement from his clinical state before valvulotomy.) All patients had acoustic evidence of mitral stenosis, and case 4 had, in addition, acoustic evidence of mitral insufficiency and aortic regurgitation. Cases 4 and 5 showed only left auricular enlargement on fluoroscopy. In addition to this, case 6 showed slight enlargement of the outflow tract of the right ventricle. The electrocardiograms in this group were within normal limits.

3. Mitral Stenosis (Severe), Group 2. The four patients of this group, cases 7, 8, 9 and 10, 22, 23, 33 and 39 years of age respectively, had mitral disease with evidence in their resting pulmonary artery pressures and in their response to exercise and to the Valsalva-like maneuver which suggested a valve lesion severe enough to have led to demonstrable cardiodynamic alterations (table 1). Cases 8, 9 and 10 had had known episodes of rheumatic fever in childhood, and case 7 was known to have mitral stenosis for four years. Cases 8 and 9 had dyspnea on moderate exertion. Cases 7 and 10 had had episodes of congestive heart failure, and were dyspneic on slight exertion. However, none of these patients was in congestive heart failure at the time of this study. Acoustic evidence of mitral stenosis was present in all cases. In addition, case 9 showed acoustic evidence of aortic valvular deformity. Fluoroscopy revealed gross cardiac enlargement, including left auricular enlargement, except in case 10 in which there was only left auricular enlargement (the right ventricular outflow tract being at the upper limits of normal). Cases 8 and 9 had auricular fibrillation. The electrocardiograms in all these patients were definitely abnormal. Cases 7, 8 and 9 were taking digitoxin.

**METHOD**

All studies were done in the Florsheim Cardiovascular X-ray Research Unit in the morning without prior sedation. Under local anesthesia (Metycaine) a Courmand needle was placed in the brachial artery of the left arm for data on systemic arterial oxygen and pressure. Cardiac catheterization was performed according to the technic of Courmand and Ranges. The catheter was placed in the pulmonary artery in all instances except in case 3 in which it could not be advanced beyond the right ventricle. Simultaneous pressure tracings from the pulmonary artery (or right ventricle) and brachial artery, using electromanometers* recording on a four channel direct-writing polyoscillograph, were made together with lead II of the electrocardiogram.

* Sanborn Company, Cambridge, Massachusetts.

After a short control tracing, the patient was instructed to blow into a mercury manometer for as long as he was able, a Valsalva-like maneuver. Maximum sustained intracardiac pressure was recorded by the method recently described. Afterward the patient relaxed. Continuous tracings were made until the pressure in the pulmonary and brachial arteries had returned to normal.

The effect of the Valsalva-like procedure on the circulation was divided into four phases as suggested by Hamilton. The first phase occurs immediately after straining begins, the second during the course of the sustained straining, the third immediately after straining ends, and the fourth lasts until blood pressure reaches control levels. Tabulation of the systolic, diastolic, and pulse pressures and of the heart rate during each of the four phases of the Valsalva-like maneuver was made in the systemic and pulmonary circuit in the normal as well as in the two groups of mitral cases and the results are summarized in table 1.

**RESULTS**

1. *Effect of Straining on the Systemic Circulation and Heart Rate* (table 1, figs. 1, 2 and 4)

A. Normal. The Valsalva-like maneuver affected the systemic blood pressure, the contour of the pulse and the cardiac rate.

The average intraoral pressure for this normal group during the Valsalva-like maneuver was 55 mm. Hg, indicating that the average intrapleural pressure was about 48 mm. Hg. Pressures in the pulmonary artery or right ventricle were normal at rest. They were also normal on exercise, when measured. The cardiac index at rest was in the accepted normal range in two patients and somewhat above this rate in one due to anxiety. In one of the two patients in whom exercise was done the cardiac index doubled. In the other case, the patient, a normal, healthy Mexican male, apparently had considerable anxiety at rest, with a resultant cardiac output of 10 liters per minute, and a cardiac index of 6.1 liters per minute per square meter of body surface. During exercise there was no appreciable change in the cardiac output. Dexter and coworkers have published similar data on patients with a relatively high output at rest and Hickam and his associates have actually shown a fall of cardiac output during cardiac catheterization when exercise was performed in normal anxious patients.
Table 1---Summary of Data on the Valsalva-like Maneuver Compared with Pertinent Cardiac Catheterization Findings

<table>
<thead>
<tr>
<th>Case</th>
<th>Normal</th>
<th>Brachial Artery</th>
<th>Pulmonary Artery</th>
<th>IOP</th>
<th>ECG</th>
<th>CI REST</th>
<th>CI EX.</th>
<th>PA REST</th>
<th>PA EX.</th>
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<tr>
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<td>128/84</td>
<td>76/144/106</td>
<td>58 75/128/96</td>
<td>32</td>
<td>94</td>
<td>70/48</td>
<td>22 107/156/86</td>
<td>70</td>
<td>54</td>
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<tr>
<td>2.</td>
<td>122/94</td>
<td>83/152/106</td>
<td>46 83/78/74</td>
<td>4 107 30/46</td>
<td>4 135/222/124</td>
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<td>69</td>
<td>14/4</td>
<td>10 40/36</td>
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<tr>
<td>3.</td>
<td>100/64</td>
<td>83/116/80</td>
<td>36 83/96/74</td>
<td>22 107/86/46</td>
<td>22 107/146/86</td>
<td>56</td>
<td>71 20/0</td>
<td>20 40/18</td>
<td>22 32/13</td>
</tr>
<tr>
<td>Av.</td>
<td>120/71</td>
<td>80/144/97</td>
<td>47 80/101/81</td>
<td>20 103/69/53</td>
<td>16 117/175/102</td>
<td>71</td>
<td>62</td>
<td>18/7</td>
<td>11* 63/53</td>
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<tr>
<td>Mitral Stenosis (Mild) Group 1</td>
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<td>116/60</td>
<td>79/144/78</td>
<td>68 89/110/80</td>
<td>30 120/68/50</td>
<td>38 130/150/80</td>
<td>70</td>
<td>68 65/15</td>
<td>11 36/30</td>
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<td>110/70</td>
<td>80/144/48</td>
<td>48 85/130/100</td>
<td>30 88/100/76</td>
<td>24 94/136/80</td>
<td>56</td>
<td>60 31/16</td>
<td>15 48/40</td>
<td>8 43/34</td>
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<tr>
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<td>80/124/84</td>
<td>40 80/120/100</td>
<td>25 73/100/76</td>
<td>24 75/134/80</td>
<td>54</td>
<td>65 20/8</td>
<td>17 42/30</td>
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<tr>
<td>Av.</td>
<td>112/95</td>
<td>78/135/86</td>
<td>53 81/122/93</td>
<td>29 94/96/67</td>
<td>29 96/140/80</td>
<td>60</td>
<td>65 24/10</td>
<td>14 43/23</td>
<td>9 41/34</td>
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<td>Mitral Stenosis (Severe) Group 2</td>
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<td>34 85/110/80</td>
<td>30 83/66/58</td>
<td>28 88/100/64</td>
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<td>88 57/28</td>
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<td>83/166/106</td>
<td>60 68/139/109</td>
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<td>93 56/30</td>
<td>26 108/80</td>
<td>28 92/90</td>
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<td>97/116/84</td>
<td>32 97/76/64</td>
<td>12 103/60/56</td>
<td>4 107 89/67</td>
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<td>4 40/15</td>
</tr>
<tr>
<td>Av.</td>
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<td>90/146/97</td>
<td>49 84/124/91</td>
<td>33 94/105/71</td>
<td>34 97/124/77</td>
<td>47 97 58/27</td>
<td>31 86/59</td>
<td>27 67/52</td>
<td>15 48/21</td>
</tr>
</tbody>
</table>

* = Cases with pulmonary artery pressure
† = Cases with right ventricular pressure
‡ = Last 3 cases
BP = Blood pressure, mm. Hg
PP = Pulse pressure, mm. Hg
R = Cardiac rate, beats/min.

IOP = Intraosolar pressure, mm. Hg
ECG = Electrocardiogram
CI = Cardiac index, L/min./M²
PA = Pulmonary artery pressure, mm. Hg
EX. = Exercise
WNL = Within normal limits
PWNL = Probably within normal limits

RHS = Right heart strain
RBBSB = Right bundle branch system block
A.F. = Auricular fibrillation
B.C. = Borderline curve
M.P. = Mitral P waves

Data presented are averages of each phase.
Phase 1. With the onset of straining, both the systolic and diastolic blood pressures rose in all three patients while the pulse pressure remained close to the control value. The maximum pressure was attained very shortly after the straining was begun (within 4 beats). During this period the cardiac rate and pulse contour remained unchanged.

![Fig. 1. Systolic blood pressure and pulse pressure recorded from the brachial artery during the control period and the four phases of the Valsalva-like maneuver, for normal subjects and for patients with mitral stenosis (mild), group 1, and (severe), group 2. Note the lack of “overshoot” in phase 4 in patients with mitral stenosis, group 2.](image)

Phase 2. As the straining was sustained there was a gradual fall in the systolic and diastolic blood pressure, the former declining more than the latter. Consequently there was a decrease in pulse pressure. The latter became almost imperceptible in case 2. The systolic pressure in one instance fell below, and in two instances was at or near, the control level. In two instances the diastolic pressure remained above, and in one it fell below, the control level. The cardiac rate, in all instances, rose above the

![Fig. 2. Cardiac rate during the control period and four phases of the Valsalva-like maneuver for patients in normal group and in mitral stenosis, groups 1 and 2. Note relative constancy of pulse in severe mitral stenosis (group 2), and lack of bradycardia in phase 4 in this group.](image)

![Fig. 3. Systolic blood pressure and pulse pressure as recorded from the pulmonary artery in normal subjects and mitral stenosis, groups 1 and 2, during the control period and four phases of a Valsalva-like maneuver. Note “overshoot” in all groups in phase 4 and that this is not as marked for each group as in the systemic circuit (compare with figure 1).](image)
Fig. 4. Typical pressure records obtained with Valsalva-like maneuver from simultaneously registered brachial (upper) and pulmonary (lower) arteries. A is case 1, a normal individual; B is case 5, a patient with mild mitral stenosis (group 1); and C is case 7, a patient with severe mitral stenosis (group 2).

In the pressure records the Valsalva-like maneuver begins with a rapid rise in pressure, and ends with a sudden fall. The control period in A is the first 2½ beats, in B the first 1½ beats and in C the first 6 beats. Phase 1 of the Valsalva-like maneuver is the first few beats after the onset of the pressure rise, phase 2 occur during the steady level of the pressures, phase 3 is the first beat or two immediately after the end of the maneuver during which the pressure falls, and phase 4 occurs over the next 8 to 10 beats—with “overshoot” and bradycardia present in A and B at this time and not present in C. There are 2 premature systoles near the end of record C.

Ordinates are pressures in mm. Hg, and abscissas indicate time, each heavy line equaling 0.2 second. Discussed in text.
control rate. In two instances, a definite sinus tachycardia was present. The contour of the pulse was markedly changed. The dicrotic notch approached end diastole and eventually the dicrotic pressure equaled the end diastolic pressure. The pulse curve tended to become symmetric, with the anacrotic limb equal to the catacrotic limb.

Phase 3. With the cessation of straining there was an immediate drop in both systolic and diastolic pressures. The pulse pressure usually remained the same as in phase 2. The blood pressure in all instances fell below the control level. The contour of the pulse remained essentially similar to that in the preceding phase. The cardiac rate was somewhat faster than in the previous phase.

Phase 4. Soon after cessation of straining the systolic and diastolic blood pressure rose. There was a relatively greater increase in the former so that the pulse pressure increased rapidly. The control blood pressure level was quickly reached (within 5 to 8 beats). The pressure continued to rise so that the control systolic, diastolic and pulse pressures were definitely exceeded in all three patients. During this period, termed the "overshooting" phase, the pressure may rise to extremely high levels, with the heart rate at the same time becoming moderately or markedly slowed. In all three of our patients the heart rate fell below the control levels. The contour of the pulse returned to normal somewhat more slowly than the pressure. The dicrotic notch appeared higher on the catacrotic limb until it eventually assumed its normal position.

B. Mitral Stenosis (Mild), Group 1. As stated above, this group has much in common with the normal group. Though the cardiac index was slightly below normal in all three patients, there was a normal response of the cardiac output to exercise. The pulmonary artery pressures at rest were at the upper limits of normal, and were increased only slightly by exercise. The intraoral pressure was recorded in only one instance (case 6). It was 50 mm. Hg during the Valsalva-like maneuver, indicating that the average intrapleural pressure was about 43 mm. Hg.

The four phases of the Valsalva-like maneu-

ver showed essentially the same response in the systemic circulation as did the subjects composing the normal group. In phase 1, coincident with the increase in blood pressure, there was a decrease in pulse rate in two of the three patients. The "overshoot" and bradycardia in phase 4, while not as great as in those of the normal group, was definite.

C. Mitral Stenosis (Severe), Group 2. The patients in this group, with one exception (case 9), had low cardiac indexes at rest and all had fixed cardiac indexes on exercise. The pulmonary artery pressures, high at rest, became considerably increased during exercise. The average intraoral pressure during the Valsalva-like maneuver was 53 mm. Hg, indicating that the average intrapleural pressure was about 46 mm. Hg.

As in the normal group, the effect of straining upon the systemic circulation may be divided into four phases. With the onset of straining, the systolic and diastolic pressures rose, the maximum pressure being attained before the fourth beat in all cases. The pulse pressure remained close to control level. The cardiac rate slowed in two of the four patients during phase 1, but returned to essentially control rates during the other phases. As the strain was sustained (phase 2) the pressure fell somewhat below that observed in phase 1. Although the pulse pressure became smaller, it did not decrease to the extremes noted in the normal group, except in case 10. Similarly, the pulse contour did not exhibit the marked changes observed in the control group. The cardiac rate was close to control level in this phase.

With the release of straining (phase 3), both systolic and diastolic pressures fell to below control levels, but not to the extent seen in the normal group. The pulse pressure remained as in phase 2 and the cardiac rate was unchanged.

During recovery (phase 4) the blood pressure rose gradually, returning to normal levels within 4 to 9 beats. The contour of the pulse followed the recovery of the blood pressure. The pronounced "overshooting" of the pulse pressure was not observed in any of the patients in this group. The marked slowing of the pulse was also not observed in any case. In no in-
stance did the cardiac rate during this phase fall below control levels.

Except for phase 1, when there was slowing in two patients, the cardiac rate remained at approximately control levels during all phases of the Valsalva-like maneuver.

2. The Effect of Straining on the Pulmonary Circulation (table 1, figs. 3 and 4)

A. Normal. With the onset of the Valsalva-like maneuver (phase 1) the systolic and diastolic pressures in the pulmonary artery (or right ventricle) rose in all subjects. The pulse pressure remained at control level in two patients and below it in the third. As straining was maintained (phase 2) the blood pressure fell slightly below the preceding phase. In contrast to the systemic circulation, however, the systolic and diastolic pressures in the pulmonary artery (or right ventricle) remained considerably above control levels during phase 2. During this phase all pulse pressures were reduced below control values. As the strain was released, the pressure within the lesser circulation dropped precipitously to below control levels, though the pulse pressure quickly rose above that in the previous phase. During recovery (phase 4) the blood pressure rose quickly, returning to control levels somewhat earlier than in the systemic circuit. An "overshoot" with an increased pulse pressure was present in the pulmonary artery (or right ventricle) but the pressure change was not nearly as marked as that observed in the systemic circuit. In no case were the upper limits of normal values for the pulmonary artery (or right ventricular pressures) exceeded during this phase.

B. Mitral Stenosis (Mild), Group 1. The findings in this group were essentially similar to those of the normal group. The systolic and diastolic pressures in this group during the period of the "overshoot" (phase 4) in two of the three patients were definitely elevated above normal values for pulmonary artery pressure. However, the control pressures in this group, while within normal limits, were higher than in the normal group, so that the actual "overshoot" was quantitatively similar to that seen in the normal group.

C. Mitral Stenosis (Severe), Group 2. The quantitative findings were similar to those shown by the other groups. The control pressures were higher, and as a result the "overshoot" present in the pulmonary circulation (in contrast to its absence in the systemic circuit) reached higher levels than in the other groups.

Discussion

The pressures recorded from the brachial and pulmonary arteries are gross pressures during the straining procedure. While the diastolic brachial artery pressure and the systolic and diastolic pulmonary artery pressures are elevated during the sustained period of straining, the net blood pressure in these vessels—obtained by subtracting the simultaneous intrapleural pressure from the gross blood pressure—was actually found to be reduced, in confirmation of the findings of Courand and co-workers. This is particularly well seen in phase 3 where the blood pressure falls precipitously below control values when the support of the intrapleural pressure is removed.

Our observations on the effect of the Valsalva-like maneuver upon the systemic circuit in normal subjects are in agreement with those of Hamilton and co-workers. With the onset of straining, the intrapleural pressure rises. We have found that during the Valsalva-like maneuver intrapleural pressure curves closely simulate intrapleural pressure curves in contour and have concluded that the intrapleural pressure is an accurate measure of intrapleural pressure during this maneuver, being on the average 6 to 7 mm. Hg higher than the latter. With the onset of straining the pressure in the brachial artery rises. This undoubtedly is related to an increased output of the left ventricle, as a result of the blood forced into it from the lungs. As the strain is sustained, the blood pressure level and particularly the pulse pressure in the systemic circuit fall. This is attributable to the depletion of the pulmonary blood reservoir which is squeezed out by the increased intrapleural pressure and prevented from refilling by the simultaneous decrease in venous return to the heart resulting from the same cause. During these phases, reflexes may come
into play. These may explain the slowing of the heart in some cases during phase 1, when there is a sudden increase in blood pressure, and the increase in the heart rate in phase 2. The tachycardia persists into phase 3.

During straining the venous pressure becomes markedly elevated\textsuperscript{14} because of interference with venous return. With the cessation of straining and the sudden release of the elevated intrapleural pressure, the venous pressure, as expected, falls abruptly. This is associated with a marked and immediate increase in the venous return of blood to the heart from the blood stored in the veins during straining, which is facilitated by the hyperpnea following straining. It is this brisk augmentation in venous return that leads to the "overshoot" in the blood pressure in phase 4. This marked blood pressure "overshoot" in the systemic circuit in its turn leads reflexogenically to slowing of the heart and to a generalized vasodilation. The cardioregulatory reflexes appear to originate in the receptors of the carotid sinus and aortic arch and their effect may be eliminated by various drugs.\textsuperscript{17, 18}

The response to the straining procedure in the systemic circulation of patients with dynamically insignificant mitral stenosis was similar to that observed in normal individuals. In patients of this group the blood pressure "overshoot" and bradycardia of phase 4 were present and marked, but to a lesser degree than in normal subjects. This suggests that the factors which operate in the more severe mitral stenosis cases do so in these milder cases but to a lesser extent. In the patients with severe mitral stenosis the blood pressures "overshoot" and bradycardia of phase 4 was absent or minimal.

In the pulmonary circulation all groups of patients showed an "overshoot" of approximately the same degree in the post-straining period of the Valsalva-like maneuver (phase 4) regardless of the initial resting pulmonary artery pressure. This was unexpected on the assumption that in severe mitral stenosis the pulmonary vascular bed was reduced because of the marked pulmonary vascular engorge ment. However, apparently the pulmonary bed is so large\textsuperscript{19} and can be emptied sufficiently by straining even in patients with a tight mitral stenosis, that it can accommodate the added post-straining load as well as in the normal individual without an augmented blood pressure "overshoot" in phase 4 (or the distensibility is altered with mitral stenosis).

The differences in the systemic blood pressure "overshoot" and its associated bradycardia between the normal subjects and those with tight mitral stenosis is obviously due to the obstruction at the mitral orifice. The deformed valve causes an obstruction to the filling of the left ventricle which normally occurs almost entirely during early diastole (in the rapid inflow phase). The auricular systolic contribution ordinarily is of minor importance and equal to about 5 per cent of total filling. Mitral stenosis affects chiefly the rapid inflow phase of left ventricular filling.\textsuperscript{20} In mild degrees of stenosis, as in our mitral stenosis group 1, the tendency to impairment of filling during rapid inflow is compensated adequately by the elevation of left auricular pressure and by an augmented auricular contraction. The result will be a dynamically unimportant lesion but one with acoustic characteristics. When, however, the obstruction becomes severe, as in our mitral stenosis group 2, these compensatory mechanisms are inadequate and filling is impaired. As a result the cardiac index at rest tends to be low. The obstruction at the mitral valve not only operates to prevent filling at rest but does so during exercise. Hence the cardiac output cannot increase to the same degree in patients with "tight" mitral valves as in normal individuals. In fact, the disparity between normal subjects and patients with severe mitral stenosis is much more marked during exercise than at rest as we and others\textsuperscript{6-7} have found. A similar situation prevails during the Valsalva-like maneuver. The sudden increase in the quantity of blood entering the right heart from the blood pooled in the veins during the early post-straining period, as a result of the release of the increased intrapleural pressure, cannot readily pass on into the left ventricle because of the stenotic mitral valve. Hence, the blood is accommodated for a time behind the mitral valve until in the course of time it drains away. This
limitation to the augmentation of left ventricular filling and output is dependent on the severity of the degree of mitral stenosis. It determines in turn the presence and magnitude of the systemic blood pressure “overshoot” and its accompanying reflexogenic bradycardia. In mitral stenosis group 1, where the stenosis is mild, as evidenced by the high normal pulmonary arterial pressure at rest and the almost normal response of the pulmonary artery pressure and cardiac index to exercise, there was exhibited a definite but less than normal “overshooting” in systemic blood pressure and bradycardia in phase 4. In the severe cases, mitral stenosis group 2, with resting pulmonary hypertension, marked augmentation of pulmonary arterial pressure and fixed cardiac indexes on exercise, there was exhibited little or no “overshooting” in systemic blood pressure and little or no slowing in pulse rate in phase 4.

Since the reflexogenic bradycardia after straining in mitral stenosis is dependent on the magnitude of the “overshoot” in systemic arterial pressure and this is in turn inversely related to the severity of the mitral stenosis, it is possible that the presence or absence of the bradycardia and its degree after a standardized Valsalva-like maneuver may be used as a simple bedside index of the dynamic severity of mitral stenosis. It is obvious that this needs further testing in mitral stenosis. Furthermore, other circumstances which prevent the ready redistribution of blood on and after straining will have to be tested as well. Such studies are under way.

SUMMARY

1. The effect of the straining procedure upon the systemic and pulmonary circulations in normal individuals and patients with mitral stenosis of several grades is described.

2. The response of patients with severe mitral stenosis to this procedure differs from that of normal individuals and individuals in whom the mitral stenosis is only of acoustic significance. The difference is based upon the altered dynamics created by the stenotic mitral valve.

3. The value of this procedure in establishing the dynamic significance of mitral stenosis and in estimating the size of the mitral valve is indicated.

4. It is possible that the presence or absence of the reflexogenic post-straining bradycardia and its degree with a standardized Valsalva-like maneuver may be used as a clinical bedside index of the degree of mitral stenosis. Other factors which can have an effect similar to mitral stenosis are being examined.

ACKNOWLEDGMENTS

We are indebted to the physicians of Michael Reese Hospital for permission to study and report upon their patients. We are also indebted to the Cardiorespiratory and Catheterization Teams for their valuable assistance in obtaining these data.

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Circulation. 1952;5:38-47
doi: 10.1161/01.CIR.5.1.38

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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