Hemodynamic Sequelae of Sustained Elevation of Left Atrial Pressure

By Arthur Selzer, M.D.

A comparison of hemodynamic findings in 63 cases of mitral stenosis and in 35 cases of chronic left ventricular failure is presented. It is shown that circulatory dynamics of the 2 conditions are similar and only minor differences were detected.

SIMILARITIES between mitral stenosis and left ventricular failure, regarding their effects upon the pulmonary circulation and the right heart, have been known long before cardiac catheterization permitted their direct assessment. The advent of mitral valve surgery, a decade ago, stimulated widespread interest in the hemodynamics of mitral stenosis. On the other hand, circulatory changes associated with cardiac failure have been studied in considerably less detail. In 1955, Harvey et al.1 raised an important practical point regarding this similarity: are hemodynamic changes caused by mechanical factors (mitral stenosis) and those caused by myocardial factors (cardiac failure) distinctive enough to permit their differentiation? These authors answered this question in the affirmative, suggesting that venous catheterization of the heart is helpful in the selection of proper candidates for surgical treatment. The purely surgical aspect of this problem can now be approached in a more direct way, in that combined right and left heart catheterization permits the direct measurement of the diastolic gradient across the mitral valve. Other aspects of the similarity and dissimilarity between mitral stenosis and left ventricular failure are, however, of considerable importance, particularly in view of the increasing interest in the mechanism of pulmonary hypertension which may complicate these conditions. The current study was undertaken in an effort to compare circulatory dynamics of these 2 conditions in a larger series of cases, and to present them quantitatively by taking into consideration their respective severity.

MATERIAL AND METHODS

The material for this study consisted of 63 cases of mitral stenosis and 35 cases of left ventricular failure, a total of 98 cases. All patients underwent a detailed clinical scrutiny, and the diagnosis was established on the basis of clinical, electrocardiographic, phonocardiographic, and radiologic examinations. The group of cases with mitral stenosis included patients with pure mitral stenosis and those patients with combined mitral stenosis and insufficiency in whom mitral stenosis was considered the predominant lesion. All patients thus were "surgical" cases and in most of them mitral block was severe enough to subject them to mitral valvotomy. Patients underwent cardiac catheterization at the time of a stable clinical condition; those who entered the hospital in cardiac failure, with pulmonary edema, or with uncontrolled atrial fibrillation were studied only after these complications were eliminated or brought under complete control.

Patients with left ventricular failure were selected out of a larger group of patients with low-output cardiac failure,2 who, studied at the time cardiac failure was brought under control, showed significant elevation of left atrial pressure (pulmonary wedge pressure over 15 mm. Hg). The clinical diagnoses included hypertensive heart disease, arteriosclerotic (coronary) heart disease, and aortic valvular disease.

Cardiac catheterization was performed according to uniform standards used in this laboratory.3,4 The mode of study, the technic of recording pressures, the method for the study of cardiac output, and the exercise load have all been carefully standardized so as to make the findings comparable.

RESULTS

In order to assess properly the hemodynamic effects of mitral stenosis and of chronic left ventricular failure it was necessary to find

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means of making the severity of each condition comparable with each other. The immediate effect of mitral block and of left ventricular failure, the elevation of left atrial pressure, is in itself not an adequate yardstick of severity of each condition because of its dependence upon the magnitude of cardiac output. It was therefore considered that a ratio of cardiac output to left atrial pressure would be a more desirable indication of the severity of the circulatory derangement. This ratio has been first used by Wood as a simplified substitute for the calculation of the mitral valve area, and is derived from the formula

\[
\text{CO/LAP index} = \frac{\text{cardiac output (L./min.)}}{\text{pulmonary wedge pressure (mm. Hg)}} \times 100
\]

CO/LAP index, and is termed "mitral valve index." Actually, this index does not take into consideration the gradient across the mitral valve in diastole and therefore more properly expresses the resistance to diastolic filling of the left ventricle, being applicable equally well to left ventricular failure as to mitral stenosis, because it is immaterial whether this resistance is caused by mechanical obstruction or by incomplete emptying of the failing ventricle. In order to assess the value of this index in expressing the severity of mitral stenosis, it was compared with the mitral valve area (MVA) calculated from the formula of Gorlin and Gorlin in the series of 67 cases of mitral stenosis. This comparison (fig. 1) shows an excellent correlation. As suggested by Wood, severe mitral stenosis is shown by an index less than 15 (MVA less than 0.75 cm²); moderate mitral stenosis by an index between 15 and 25 (MVA 0.75 to 1.25 cm²); mild mitral stenosis by an index more than 25. Obviously, mild, moderate, and severe left
ventricular failure can be graded by a similar range of indices.

The material was divided into 2 groups reflecting the severity of mitral stenosis and left ventricular failure respectively, with the dividing line set arbitrarily at index of 16. Group I included cases of severe mitral stenosis and severe left ventricular failure with indices from 8 to 15. The second group included all other cases, the index being over 15. Table 1 presents a summary of the findings, showing the mean values and the ranges of the various hemodynamic measurements in group I. It is seen that the mean age of patients with left ventricular failure is higher and the mean value for the index slightly lower in left ventricular failure than in mitral stenosis. The mean value for left atrial pressure is similar in the 2 groups. Pulmonary artery pressure is slightly higher in mitral stenosis, both at rest and during exercise, although the per cent increase with exercise is not materially different in the 2 groups. The cardiac output is abnormally low in both groups and appears to be lower in the patients with cardiac failure than in those with mitral stenosis. However, the per cent increase of the output with exercise is identical in both groups, being abnormally low in both. Pulmonary vascular resistance is abnormally high in both groups, the mean values being elevated to the same extent.

Table 2 shows a summary of findings in the milder cases of mitral stenosis and of left ventricular failure. Here, the mean value for the index is similar in both classes. The age shows an even wider spread than in severe cases. Pulmonary wedge and resting pulmonary arterial pressure are similar in both classes and the increase with exercise, which is abnormal in both, is more marked in left ventricular failure. The mean resting cardiac output is slightly below normal in mitral stenosis, and is still lower in cardiac failure. There is, however, an appreciable difference in its response to exercise: the increase in output in mitral stenosis is normal, while in cardiac failure it is abnormally low. Pulmonary vascular resistance is at the upper limit of normal in mitral stenosis, but is abnormally elevated in left ventricular failure, where the mean value is twice that of the other group. The 2 most important hemodynamic findings, the resting cardiac output and the pulmonary vascular resistance are compared in the 2 classes in figures 2 and 3. Figure 2 shows the relationship between the resting cardiac output and the severity of mitral stenosis and left ventricular failure, respectively, expressed as the CO/LAP index. It is seen that a normal cardiac output (shaded area) is found, as a rule, in both conditions with indices larger than 26. In severe cases, cardiac output is abnormally

| Table 1.—Summary of Hemodynamic Findings in More Severe Cases of Mitral Stenosis and of Chronic Left Ventricular Failure (Index 8 to 15) |
|---|---|---|---|---|---|---|---|---|
| Mitral stenosis (19 cases) | Age | CO/LAP (L. min./M.²) | Pulmonary wedge pressure (mm. Hg) | Pulmonary capillary wedge pressure (mm. Hg) | Pulmonary arterial pressure (mm. Hg) | Cardiac output (L. min./M.²) | Cardiac output (L. min./M.²) | Pulmonary resistance (dynes sec. cm.⁻²) |
| Mean value | 40 | 12.1 | 31.4 | 56.2 | 76.6 | 2.1 | 2.5 | 533 |
| Range | 18-61 | 10-15 | 17-40 | 25-79 | 32-106 | 1.7-2.7 | 1.8-3.6 | 75-1050 |
| Left ventricular failure (15 cases) | Mean value | 50.7 | 10.5 | 29.2 | 45.1 | 62.5 | 1.7 | 2.0 | 513 |
| Range | 55-65 | 8-13.5 | 16-36 | 30-74 | 43-90 | 1.2-3.6 | 1.5-2.6 | 110-1020 |
TABLE 2.—Summary of Hemodynamic Findings in Milder Cases of Mitral Stenosis and of Chronic Left Ventricular Failure (Index Over 15)

<table>
<thead>
<tr>
<th>Condition</th>
<th>CO/LAP Index</th>
<th>Pulmonary wedge pressure (mm Hg)</th>
<th>Pulmonary arterial diastolic pressure (mm Hg)</th>
<th>Pulmonary arterial systolic pressure (mm Hg)</th>
<th>Cardiac output at rest (liters/min/M²)</th>
<th>Cardiac output at exercise (liters/min/M²)</th>
<th>Pulmonary vascular resistance (dynes sec cm⁻²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mitral stenosis (43 cases)</td>
<td>40.5</td>
<td>23</td>
<td>21.5</td>
<td>31.4</td>
<td>44.3</td>
<td>2.5</td>
<td>3.7</td>
</tr>
<tr>
<td>Mean value</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Range</td>
<td>28-56</td>
<td>16-35</td>
<td>16-27</td>
<td>21-54</td>
<td>25-72</td>
<td>2.0-3.1</td>
<td>2.6-5.2</td>
</tr>
<tr>
<td>Left ventricular failure</td>
<td>61.9</td>
<td>22.9</td>
<td>19.3</td>
<td>33.4</td>
<td>51.7</td>
<td>2.3</td>
<td>2.7</td>
</tr>
<tr>
<td>Mean value</td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Range</td>
<td>44-74</td>
<td>16-35</td>
<td>15-24</td>
<td>21-57</td>
<td>36-75</td>
<td>1.7-3.2</td>
<td>1.7-4.3</td>
</tr>
</tbody>
</table>

low in both conditions, but in the intermediate group of severity of 15 to 25, an abnormally low cardiac output is found more frequently in cardiac failure than in mitral stenosis.

Figure 3 shows pulmonary vascular resistance in mitral stenosis and in left ventricular failure plotted against the severity index. It is seen that abnormally high pulmonary vascular resistance is found only in moderately severe and severe cases of both conditions. It is also seen that in both conditions, an appreciable number of severe cases is found with normal or near-normal vascular resistance.

DISCUSSION

The object of this study was a comparison between the hemodynamic effects of mitral stenosis and of chronic left ventricular failure. Both these conditions have one common denominator: the sustained elevation of left atrial pressure.

The dynamics of mitral stenosis is well known. The series of cases included in this study represents a wide spectrum of severity of mitral valve block with hemodynamic findings similar to those of earlier investigators.⁷-¹⁰

In mitral stenosis, elevation of left atrial pressure is caused by valvular obstruction introducing resistance to flow through the mitral orifice. The level of left atrial pressure is thus an index of the severity of mitral stenosis for a given level of flow through the orifice in diastole. Chronic left atrial hypertension is an essential and constant feature of significant mitral stenosis.

In left ventricular failure, elevation of left atrial pressure reflects resistance to diastolic filling of the incompetent left ventricle and can be considered a physiologic adaptation, permitting the overloaded and overstretched left ventricle to maintain the necessary forward flow. This may be an acute response to temporary overload, such as during exercise, when it is reversible with cessation of exercise. Furthermore, it may be a more chronic finding as an expression of left ventricular failure at rest, but still reversible by cardiac therapy. Finally it may assume the form of irreversible, chronic left ventricular failure. The frequent occurrence of chronic left ventricular failure has been shown in another study from this laboratory.² The series of cases of left ventricular failure included in this study falls into the last category.

In both mitral stenosis and chronic left ventricular failure, the abnormally high value for left atrial pressure expresses the severity of the circulatory disturbance only in relation to the magnitude of the cardiac output; hence the index CO/LAP was used as a more accurate expression of the degree of mitral valve
obstruction and of severity of left ventricular failure, in preference to the level of left atrial pressure alone.

From the data presented here it can be concluded that a comparison of the hemodynamic effects of mitral stenosis and of left ventricular failure reveals predominant similarities. In both these conditions, pulmonary hypertension is the rule, either passively reflecting left atrial hypertension, or exaggerated by increased pulmonary vascular resistance. Severely elevated pulmonary vascular resistance, exceeding 600 dynes sec. cm.\(^{-5}\), appears as an occasional complication of severe mitral stenosis and, to a comparable extent, of severe left ventricular failure. The effect of sustained elevation of left atrial pressure upon the cardiac output is variable. In the more severe cases of mitral stenosis and of left ventricular failure alike, the resting cardiac output is almost always reduced. In both conditions, the rise of the cardiac output in response to exercise is subnormal, but most cases showed some increase, so that a "fixed" cardiac output was uncommon in this series. In milder cases, the resting cardiac output was at the low normal range, showing no difference between the 2 conditions.

Only 2 points could be noted in which the 2 conditions differed from each other: (a) significant increase in pulmonary vascular resistance was observed only in severe mitral stenosis, but occurred occasionally in milder cases of left ventricular failure (fig. 3) and by the higher mean value for pulmonary vascular resistance in milder cases of left ventricular failure versus mitral stenosis (table 2); (b) in milder cases of mitral stenosis, cardiac
output showed a normal rise with exercise, but in mild left ventricular failure this response was subnormal (table 2).

Harvey and her associates\(^1\) reported 8 cases of mitral stenosis and an equal number of cases of cardiac failure imitating mitral stenosis, stressing the importance of pulmonary hypertension in distinguishing between mitral block and myocardial weakness. However, the arguments of these authors are subject to criticism for the following reasons: (a) their series of cases was too small to draw definitive conclusions; (b) failure to show improvement after mitral valvotomy was considered to be proof of myocardial insufficiency; (c) left atrial pressures were not measured, whereby passive pulmonary hypertension could not be distinguished from that caused by elevated pulmonary vascular resistance.

Results of this study indicate clearly that pulmonary hypertension cannot be considered a feature helpful in separating mitral block from myocardial insufficiency, because its occurrence and severity appear to be similar in the 2 conditions. In general, it is shown here that sustained elevation of left atrial pressure due to mechanical block at the mitral valve is not associated with a distinctive hemodynamic pattern, inasmuch as all the hemodynamic abnormalities caused by it occur also in chronic left ventricular failure. The differences between the 2 conditions can only be considered minor, in view of the overwhelming similarity of all the findings. These differences, furthermore, are not helpful in evaluating...
ELEVATED LEFT ATRIAL PRESSURE

individual cases because of the wide overlapping of the findings in them.

Finally, it is necessary to take into consideration the appreciable higher age of patients in cardiac failure than those with mitral stenosis, so that the observed differences may altogether be caused by circulatory responses in different age groups rather than by differences between mechanical versus myocardial factors.

**Summary**

A series of 98 cases, consisting of 63 patients with mitral stenosis and 35 patients with chronic left ventricular failure, was examined in order to compare hemodynamic findings in these 2 conditions. Cardiac catheterization was performed at the time when all acute complications and reversible factors were eliminated. Both mitral stenosis and left ventricular failure were graded as to the severity of the respective conditions with the use of an index:

\[
\frac{\text{Cardiac output}}{\text{Left atrial pressure}} \times 100.
\]

The hemodynamic sequelae of the sustained elevation of the left atrial pressure, which is the common denominator of mitral stenosis and left ventricular failure, showed important similarities and minor differences. It was found in both groups that pulmonary arterial pressure is always elevated, but in many cases pulmonary hypertension is severe, out of proportion to left atrial hypertension, signifying abnormally high pulmonary vascular resistance. Such cases of severe pulmonary hypertension are found in more severe cases of mitral stenosis and of left ventricular failure, and its occurrence is not appreciably different in the 2 conditions. In both conditions, the resting cardiac output is, on the average, abnormally low in severe cases and normal or slightly reduced in milder cases. Inadequate rise of cardiac output with exercise is frequently observed in both groups.

On the basis of the overwhelming similarities and relatively minor differences observed in hemodynamic changes caused by chronic elevation of left atrial pressure in mitral stenosis on the one hand, and left ventricular failure on the other hand, it is concluded that mitral stenosis does not have a distinctive hemodynamic pattern that would permit a distinction between a "mechanical block" and a "myocardial factor" unless direct measurement of left atrial and left ventricular pressure permits the demonstration of a diastolic gradient across the mitral valve.

**Summario in Interlingua**

Novanta-octo patientes — 63 con stenosis mitral e 35 con chronic disfallimento sinistro-ventricular — esseva examineate con le objectivo de comparar le constatationes hemodynamic in le duo mentionate conditiones. Catheterisation cardiac esseva effectuate al tempore quando omne complicaciones acute e omne reversibile signos e symptomatas esseva eliminate. Ambe le conditiones—stenosis mitral e disfallimento sinistro-atrial—esseva graduare secundo lor severitate in le casos individual per medio del indice:

\[
\frac{\text{rendimento cardiac}}{\text{tension sinistro-atrial}} \times 100.
\]

Le sequellas hemodynamic del prolongate elevation del tension sinistro-atrial (le qual es le caracteristica commun de stenosis mitral e disfallimento sinistro-ventricular) esseva simile in importante respectos e differente in minores. Esseva constatate in ambe gruppos que le tension pulmono-arterial es semper elevate. Tamen, in multe cases iste hypertension pulmonar ha un grado de severitate foras de proportion con le hypertension sinistro-atrial, un facto que significa le presentia de un anormalmente alte resistentia pulmonovascular. Le indicentia de un tal sever forma de hypertension pulmonar es plus alte inter le casos de sever stenosis mitral e de sever disfallimento sinistro-ventricular, sed illo non differe appreciabilemente inter le duo conditiones. In ambes, le rendimento cardiac medie in stato de reposo es anormalmente basse in casos sever e normal o paquo reduceite in casos leve. Un inadequate augmento del rendimento cardiac post exercitio physic es observate frequentemente in ambe gruppos.
Super le base del frappante similaritates et del relativamente leve differentias observate in le alterationes del stato hemodynamic que es sausate per le elevation chronie del tension sinistro-atrial in (de un latere) stenosis mitral e (del altere) disfallimento sinistro-ventricular, le conclusion es formulate que stenosis mitral non se distigue per un specific configuration hemodynamic que permetterea un distinction inter "bloco mechanic" e le presentia de un "factor myocardial," excepte si le directe mesuration del tension sinistro-atrial e del tension sinistro-ventricular permette le demonstration de un gradiente diastolic a transverso le valvula mitral.

REFERENCES


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