Angiocardiographic and Physiologic Correlations in Mitral Stenosis

By Daniel S. Lukas, M.D., Peter R. Mahrer, M.D., and Israel Steinberg, M.D.

With the use of the technics of angiocardiography and cardiac catheterization the relation of specific hemodynamic abnormalities to alterations in cardiovascular anatomy in mitral valvular disease was investigated in 56 patients.

It is well known that the left atrium and pulmonary artery are characteristically enlarged in mitral stenosis. However, the quantitative relationship of these anatomic abnormalities to the concomitant disturbances in cardiovascular hemodynamics has not been defined. The few anatomic-physiologic studies that are available are semiquantitative, since they are based on rough estimates of left atrial and pulmonary arterial size derived from fluoroscopy and conventional roentgenograms of the heart. Since angiocardiography delineates the cardiovascular structures and permits precise measurements, a quantitative method for comparison with the data obtained by cardiac catheterization in patients with mitral stenosis is available. Correlation of the findings derived from the 2 technics forms the basis of this report, an abstract of which has been published previously.

Materials and Methods

Fifty-six patients with rheumatic mitral valvular disease who were undergoing evaluation for mitral valvuloplasty were selected on the basis of technically adequate angiocardiograms and the completeness of the data obtained by cardiac catheterization. In all instances cardiac catheterization and angiocardiography were performed within a short interval, usually within a week. Clinically each patient was in an optimum state, induced when necessary by vigorous medical therapy. Thirty-three were women; 23 were men. The ages varied from 21 to 56 years; the majority were in the fourth decade.

Mitral stenosis was the predominant lesion in 51 patients and was confirmed by operation in 47. Five patients had associated mitral insufficiency of a marked degree; this was verified by operation in 3 instances. The 56 patients were divided into 4 groups; group I consisted of 13 patients with pure mitral stenosis as determined by the most stringent criteria, notably, absence of an apical systolic murmur, absence of evidence of mitral insufficiency in the pulmonary "capillary" pressure tracing, and absence of a regurgitation jet at mitral valvuloplasty. The 13 patients in group II also had no palpable regurgitant jet at operation but had either an apical systolic murmur or some evidence of mitral insufficiency in the pulmonary "capillary" tracing. Group III was composed of 25 patients who exhibited a small regurgitant jet at operation or, if not operated upon, had an apical systolic murmur and significant evidence of mitral insufficiency in the pulmonary "capillary" tracing. Mitral stenosis, however, was the predominant lesion, clinically, hemodynamically, and at surgery. Group IV was composed of 5 patients with evidence of severe mitral insufficiency in addition to significant stenosis.

Cardiac catheterization was performed by methods previously described. Frontal and left lateral angiocardiograms were made at a distance of 48 inches, usually at 1-second intervals with an exposure time of 1/30 to 1/10 second. The Fairchild roll-film cassette and the F-X-R 12 by 12 inch roll-film magazine permitted multiple serial exposures. Immediately prior to angiocardiography the circulation time was determined by rapid injection of 3 ml. of Decholin in 15 ml. of saline via the Robb-Steinberg needle and stopcock unit; 50 ml. of sodium acetrizoate (Sodium Urokon) were then injected rapidly and the central cardiovascular system was visualized.

The outline of the opacified left atrium (fig. 1) was traced and the enclosed area was measured with a self-compensating polar planimeter. Left atrial volume could be calculated from measurements of the horizontal, superior-inferior and anteroposterior axes of the left atrium as defined in the posterior-anterior and lateral angiocardiograms by applying the formula for an ellipsoid.
eter of the mechanical action of the heart was not available, it was not possible to select a specific phase of the cardiac cycle for these measurements. In the 35 patients with atrial fibrillation variation in size of the left atrium from one film to another, however, was minimal. In those with normal sinus rhythm difference in size ranged from 1 to 8.1 cm$^3$ (mean: $1.8 \pm 2.4$ cm$^3$). The largest area was chosen for inclusion in the data. In half this group no change in area occurred during the entire series of exposures.

The diameter of the main stem pulmonary artery was measured at the midpoint of the vessel perpendicular to its axis (fig. 2). The diameter of the midpoint of the ascending aorta also was measured. The pulmonary artery was assumed to be cylindrical and the cross-sectional area was calculated on that basis.

Normal values for left atrial area, cross-sectional area of the main pulmonary artery and aortic diameter (table 1) were obtained from the angiograms of 24 subjects without clinical evidence of cardiovascular disease. Their ages ranged from 18 to 60 years. Most of them had peripherally located pulmonary or anterior mediastinal lesions. No involvement or distortion of the main cardiovascular structures was demonstrated in the angiograms.

**RESULTS**

**Hemodynamic Data.** The range and distribution of the physiologic measurements can be seen in figures 3 to 6. Mitral valve area was calculated in 52 patients. In 30 the area was less than 1 cm$^2$. In only 2 patients was valve area larger than 2 cm$^2$. Mean resting pulmonary artery pressure was normal in only 2 cases. There was no marked difference in the average mean pressures in groups I, II, and III, but in group IV, the pressures were generally lower. Pulmonary vascular resistance varied widely within each group. It was normal in only 3 patients. Cardiac index was normal in a significant number of group I patients only. Among the other groups no more than 4 patients had a normal cardiac output. Pulmonary "capillary" pressure at rest in all groups clustered about the level of plasma protein osmotic pressure. The pressure was normal in 1 patient but increased to abnormal levels during exercise.

**Anatomic Data (Table 1).** Left atrial size was beyond the normal range in every case.

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**Fig. 1.** Top. Frontal angiocardiogram at 9 seconds of 21-year-old woman from group 1 with pure mitral stenosis demonstrating opacification of left atrium and aorta. Bottom. Tracing with border of left atrium heavily outlined. The enclosed area was measured by planimeter. Note elliptical shape of atrium.

In a preliminary survey good correlation between the volume determined by this method and the frontal area was observed. For this reason and because in many instances the configuration of the atrium deviated significantly from an ellipsoid, it was decided to use the frontal area as the best objective index of left atrial size.

Since synchronization of x-ray exposures with either the electrocardiogram or a suitable param-
HEMODYNAMIC ABNORMALITIES AND MITRAL STENOSIS

Table 1.—Measurements of Left Atrium, Pulmonary Artery, and Aorta in Normal Subjects and Patients with Mitral Stenosis

<table>
<thead>
<tr>
<th></th>
<th>Mean ± S.D.</th>
<th>Range</th>
<th>Mean ± S.D.</th>
<th>Range</th>
<th>Significance of difference between means: p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frontal area of left atrium (cm.²)</td>
<td>27 ± 4</td>
<td>18–32</td>
<td>72 ± 27</td>
<td>41–190</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td></td>
<td>16 Normals</td>
<td></td>
<td>51 Patients, groups I-III</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>157 ± 55</td>
<td>79–238</td>
<td>5 Patients, group IV</td>
<td></td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Cross-sectional area of main pulmonary artery (mm.²)</td>
<td>530 ± 101</td>
<td>313–707</td>
<td>909 ± 285</td>
<td>491–1810</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td></td>
<td>14 Normals</td>
<td></td>
<td>35 Patients, groups I-IV</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diameter of ascending aorta (mm.)</td>
<td>23 ± 3</td>
<td>25–35</td>
<td>27 ± 4</td>
<td>23–36</td>
<td>&lt;0.1 &gt; 0.05</td>
</tr>
<tr>
<td></td>
<td>24 Normals</td>
<td></td>
<td>44 Patients, groups I-IV</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The mean atrial size in group I (60 ± 12 cm.²) was somewhat smaller than in group II (80 ± 39 cm.²) and in group III (78 ± 19 cm.²), but in general there was a marked degree of overlap of atrial size among the first 3 groups. In group IV the atrial size was significantly larger than in the other groups. There were 7 patients with "giant" atria (greater than 115 cm.²). Two were in group II, 1 in group III, and 4 in group IV. The main pulmonary arterial cross-sectional area was not as consistently enlarged as was the atrium. The areas of 3 patients in group I, 3 in group II, 9 in group III, and 2 in group IV fell within the normal range. There was little difference in the mean size and distribution among the 4 groups. Aortic diameter, contrary to general opinion, did not differ significantly from normal. The aorta appeared small in relation to the other enlarged cardiovascular structures.

Anatomic-Physiologic Correlations. Left Atrial Size. Only a low degree of inverse correlation (r = –0.34; p < 0.05 > 0.01) existed between the left atrial area per cm.² body surface area and the mitral valve area index (fig. 3). A similar and equally low relation was present between the left atrial index and the cardiac index (r = –0.34; p < 0.05 > 0.01) (fig. 4) and the left atrial size and mean resting pulmonary arterial pressure (r = 0.36; p < 0.01). The degree of these correlations as measured by the correlation coefficient, was similar among the individual groups, though statistical significance was assumed only when the groups were combined. The size of the atrium was not related to the pulmonary "capillary" pressure at rest (r = 0.24; p < 0.1 > 0.05) or during exercise; to the pulmonary vascular resistance (r = –0.07; p < 0.1) or to the cross-sectional area of the pulmonary artery (r = –0.13; p > 0.1) (fig. 5).

In group IV a high paradoxical inverse correlation (r = –0.90; p < 0.05 > 0.02) was present between atrial and pulmonary arterial size. The angiocardiograms in these patients (fig. 8) frequently showed the main pulmonary artery to be compressed and displaced to the left by the very large atrium.

When patients with mitral valve areas above and below 1 cm.² were separated without regard to group, there was a significant difference (p < 0.05) in mean atrial size: 66 ± 15 cm.² with valves greater than 1 cm.² and 81 ± 31 cm.² with valves smaller than 1 cm.². The scatter within each category, however, was wide.

In each group except the first, patients with fibrillation had larger left atria than patients with sinus rhythm although there was considerable overlap in size (table 2). In group IV, the group with the largest mean atrial
size, all patients were in atrial fibrillation. Giant atria occurred only in patients with fibrillation. The proportion of patients with fibrillation increased directly with the increasing evidence of mitral insufficiency (table 2). In each group patients with fibrillation generally had smaller mitral valve areas, smaller cardiac outputs and, therefore, more severe hemodynamic impairment than patients with normal sinus rhythm. Atrial size did not correlate with cardiac index, mitral valve area, pulmonary vascular resistance, or pulmonary arterial pressure in patients with atrial fibrillation or in those with sinus rhythm.

Cross-Sectional Area of Main Pulmonary Artery. The cross-sectional area of this vessel correlated closely \((r = 0.66; p < 0.01)\) with the mean pressure in the artery both at rest and during exercise in combined groups I, II, and III (fig. 6). Pulmonary arterial area correlated but less well with pulmonary vascular resistance \((r = 0.31; p < 0.05 > 0.01)\) (fig. 7) and with the mitral valve area \((r = -0.47; p < 0.01)\). These correlations were not demonstrable in each of the individual groups. In group I the lack of correlation was due to inclusion of 2 cases with areas differing widely from the mean. In group IV distortion and lateral displacement of the pulmonary artery by a very large atrium appeared to limit enlargement of the pulmonary artery. The pulmonary arterial cross-sectional area of a number of patients in this group was normal despite marked pulmonary arterial hypertension. The right branch of the artery

**Table 2.** Frontal Area of Left Atrium (cm.\(^2\)) in Mitral Stenosis as Related to Cardiac Rhythm

<table>
<thead>
<tr>
<th>Group</th>
<th>Statistic</th>
<th>Normal sinus rhythm</th>
<th>Atrial fibrillation</th>
<th>Significance of difference between means: (p)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Mean ± S.D.</td>
<td>57 ± 12</td>
<td>67 ± 14</td>
<td>&gt;0.5</td>
</tr>
<tr>
<td></td>
<td>Range</td>
<td>41-86</td>
<td>48-74</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Number</td>
<td>10</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>II</td>
<td>Mean ± S.D.</td>
<td>62 ± 14</td>
<td>98 ± 47</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td></td>
<td>Range</td>
<td>43-91</td>
<td>58-190</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Number</td>
<td>7</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>III</td>
<td>Mean ± S.D.</td>
<td>60 ± 7</td>
<td>82 ± 19</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td></td>
<td>Range</td>
<td>54-70</td>
<td>58-122</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Number</td>
<td>4</td>
<td>21</td>
<td></td>
</tr>
<tr>
<td>IV</td>
<td>Mean ± S.D.</td>
<td>157 ± 55</td>
<td>79-238</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Range</td>
<td>0</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>I, II, III</td>
<td>Mean ± S.D.</td>
<td>59 ± 12</td>
<td>84 ± 27</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td></td>
<td>Range</td>
<td>41-91</td>
<td>48-190</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Number</td>
<td>21</td>
<td>30</td>
<td></td>
</tr>
</tbody>
</table>
HEMODYNAMIC ABNORMALITIES AND MITRAL STENOSIS

Fig. 3 Top. Relation of left atrial frontal area in cm.$^2$/M.$^2$ body surface (ordinate) to area of mitral valve orifice in cm.$^2$/M.$^2$ body surface (abscissa) in mitral stenosis. Correlation coefficient: $-0.34$.

Fig. 4 Middle. Relation of frontal area of left atrium in cm.$^2$/M.$^2$ body surface (ordinate) to cardiac output in L./min./M.$^2$ body surface (abscissa) in mitral stenosis. Correlation coefficient: $-0.34$.

Fig. 5 Bottom. Relation of cross-sectional area of main pulmonary artery in cm.$^2$ (ordinate) to frontal area of left atrium in cm.$^2$ (abscissa) in mitral stenosis. Correlation coefficient: $-0.13$.

was similarly compressed and stretched out as it coursed over the left atrium (fig. 8).

Fig. 6 Top. Relation of cross-sectional area of main pulmonary artery in cm.$^2$ (ordinate) to mean pulmonary arterial pressure in mm. Hg during rest and exercise (abscissa) in mitral stenosis. Correlation coefficient: 0.66.

Fig. 7 Bottom. Relation of cross-sectional area of main pulmonary artery in cm.$^2$ (ordinate) to pulmonary vascular resistance in 100 dynes-sec.-cm.$^3$ (abscissa) in mitral stenosis. Correlation coefficient: 0.31.

DISCUSSION

In no other form of acquired heart disease is elevation of left atrial pressure as chronic and as pronounced during rest and exercise as in severe mitral stenosis. Ostensibly it is for this reason that the left atrium in this disease is enlarged more consistently and more strikingly than in other conditions that cause left atrial hypertension.

In this study, nevertheless, it was not possible to demonstrate a significant correlation between left atrial size and pulmonary 'capillary' pressure, a pressure which on the basis
of ample demonstration in the past can be regarded as equivalent to that in the left atrium. In most of the patients left atrial pressure fell within the range of plasma protein osmotic pressure (i.e., 25 to 30 mm. Hg) whereas left atrial area varied as much as 6-fold. The tendency for left atrial pressure at rest to be fixed at a “ceiling” equivalent to plasma protein osmotic pressure in patients with mitral orifice areas less than 0.8 cm.²/M.² has been noted previously. The majority of our subjects had such marked restriction of the mitral orifice. Although a significant negative correlation between atrial size and orifice area was found, it was of low order partially, no doubt, because of fixation of atrial pressure.

It is significant that the majority of our subjects had relatively advanced mitral disease and were under consideration for valvuloplasty. Had the entire spectrum of severity of this disease been represented more adequately, especially by mild and asymptomatic mitral stenosis, it may have been possible to identify more clearly the effects of pressure and orifice size on atrial dimensions.

When the response of a vascular structure to alterations in pressure is considered, the physical properties of the structure, notably distensibility (compliance) and elasticity, must be taken into account. In mitral stenosis these properties not only vary normally among individuals but are subject to considerable modification by such pathologic processes as mural thrombosis, rheumatic involvement of the left atrial endocardium and myocardium, mural calcification, and thickening and adhesion of the overlying pericardium. Variation in physical properties of the left atrium from patient to patient will tend to obscure a correlation between chamber size and pressure.

Larger atria in patients with atrial fibrillation as compared to those with normal sinus rhythm regardless of orifice size suggests that this rhythm also alters left atrial distensibility and elasticity. It appears likely that loss of a coordinated atrial contraction converts this chamber into a flaccid structure that is more susceptible to dilatation.

In patients with normal sinus rhythm, it was not possible to demonstrate a significant dilating effect of coexisting mitral insufficiency on the left atrium. The range and mean of atrial size was almost identical in those patients with no evidence whatsoever of mitral insufficiency, those with auscultatory and hemodynamic evidence of insufficiency but no regurgitant jet at operation, and those with auscultatory, hemodynamic, and operative signs of mitral regurgitation. In patients with atrial fibrillation, on the other hand, insufficiency was associated with significantly larger atria.

The regurgitant stream of insufficiency produces increased turbulence within the atrium, impinges on its wall and is associated with a positive systolic pressure wave, which in the presence of associated stenosis may attain very high levels. All these factors are expected to contribute to dilatation of the atrium; the
present evidence, however, indicates that their effect is more manifest in atrial fibrillation than sinus rhythm. This may be due not only to the facecidity of the atrium in atrial fibrillation but also to the more sustained nature of the insufficiency wave in the atrium with this rhythm. The effect of insufficiency on left atrial size may be assessed more completely when reliable methods for quantification of valvular regurgitation become available.

Controversy exists over the significance of a markedly enlarged or "giant" left atrium. It is regarded by some as the result of overwhelmingly predominant mitral insufficiency. Among the 7 of our patients with giant atria (area greater than 115 cm.\(^2\) — 4 times greater than the normal mean) 2 had surgically pure mitral stenosis. The atrium of 1 of these was the second largest in the series and contained a volume of blood calculated to be approximately 1.7 L. A third patient had a small regurgitant jet at operation but also very tight stenosis. Only 4 patients had marked evidence of mitral insufficiency but in the 3 treated surgically significant stenosis, which could be relieved, was found. This evidence indicates that although it is more common when insufficiency coexists, a giant atrium connotes underlying mitral stenosis which may be of pure form. In a carefully selected series of 30 cases of pure mitral insufficiency, studied by Bridgen and Leatham\(^1^2\) the "... left auricle was moderately dilated but was never aneurysmal ..." In the evaluation of an insufficient mitral valve for associated stenosis, it should be remembered that the subvalvular mitral apparatus may be the site of very significant obstruction (chordal stenosis of Rusted, Scheifley, and Edwards\(^1^3\)), which may be overlooked if not sought.

In contrast to the left atrium, enlargement of the pulmonary artery is more directly related to the severity of the hemodynamic abnormalities, particularly the degree of pulmonary arterial hypertension, which in turn depends on the severity of the stenosis and the secondary alteration in the pulmonary vascular bed.\(^6\) \(^1^0\) However, considerable scatter in the correlation occurred and in some cases of severe stenosis with pulmonary arterial pressure up to 3 times normal the main pulmonary artery was of normal size. Some of the scatter can be attributed to intrinsic alterations in the arterial wall produced by factors such as rheumatic involvement and atherosclerosis. In addition to elevated pressure, increased blood flow in the artery may increase its size as it does in atrial septal defect and in hyperthyroidism. Pulmonic valvular insufficiency also may produce considerable increase in blood flow in the main pulmonary artery and occurs with moderate frequency in severe mitral stenosis. The largest pulmonary artery encountered in this series occurred in a patient with marked pulmonary arterial hypertension and advanced pulmonic valvular insufficiency.

In a practical vein it is apparent from our data that the degree of stenosis and hemodynamic abnormality in mitral stenosis can be estimated only roughly from roentgenograms of the heart. Larger atria and main pulmonary arteries in general indicate severe disease; however, modest enlargement of these structures cannot be regarded as excluding tight stenosis. In an assessment of the size of the left atrium roentgenographically the overpenetrated frontal view of the chest offers many advantages over the lateral and oblique views even when taken with barium in the esophagus. In this view with the proper degree of penetration it is usually possible to trace the outline of the atrium almost as reliably as in the angiocardiogram, whereas in the other views only the posterior border of the chamber as it impinges on the esophagus can be delineated with assurance. Knowing the full extent of the chamber in at least 1 plane it is possible to quantify the degree of enlargement by comparison with normal data. Planimetry of the area is the preferred method of measurement; however, determination of the vertical and horizontal axes of this elliptical chamber provides a satisfactory index of the enlargement. In the normal adult these axes do not exceed 5 cm. by 8 cm. Enlargement of the atrium in the frontal plane is invariably
more prominent than in the sagittal plane. We have encountered instances in which enlargement was questionable in the lateral and oblique views but unequivocal in the frontal view.

Estimating the diameter of the main pulmonary artery from conventional roentgenograms of the heart is not so readily accomplished. As indicated previously, a large left atrium may displace the pulmonary artery to the left and cause it to project prominently along the left border of the heart and thus give a false appearance of enlargement, even though in some instances the diameter is normal. The expert can guess the diameter of the artery with reasonable accuracy, but a precise evaluation requires angiocardiography.

**SUMMARY**

The frontal area of the left atrium and the cross-sectional area of the main pulmonary artery were measured angiocardiographically in 56 patients with mitral stenosis and compared with hemodynamic data obtained by cardiac catheterization. Left atrial size was related inversely to the area of the mitral orifice and the cardiac output and directly to the pulmonary arterial pressure. These correlations although statistically significant were low grade.

Associated mitral insufficiency and atrial fibrillation appeared to increase the size of the left atrium, but a dilating effect of mitral insufficiency could not be detected in patients with sinus rhythm. Giant atria occurred only in patients with atrial fibrillation, frequently in association with mitral insufficiency, but occasionally in patients with surgically pure mitral stenosis or minimal insufficiency.

The cross-sectional area of the main pulmonary artery correlated closely with the pulmonary arterial pressure and to a lesser degree with the pulmonary vascular resistance and the size of the mitral orifice. Considerable scatter in the various anatomic-physiologic correlations suggested the existence of wide interpatient variability in the intrinsic elastic properties of the atrium and artery.

**LUKAS, MAHRER, AND STEINBERG**

The diameter of the mid-ascending aorta did not differ from normal.

**ACKNOWLEDGMENT**

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**SUMMARIO IN INTERLINGUA**

Le area frontal del atrio sinistre e le area del section transverse del major arteria pulmonar esseva mesurate angiocardiographica mente in 56 subjectos con stenosis mitral. Le resultatos esseva comparate con datos hemodynamic obtenite per catheterisation cardiace. Le dimension del atrio sinistre esseva relacionate inversemente al area del orificio mitral e al rendimento cardiac e directamente al pression del arteria pulmonar. Iste correlaciones esseva de basse grados ben que statisticamente significative.

Le association de insufficientia mitral e de fibrillation atrial pareva augmentar le dimension del atrio sinistre, sed nulle effecto dilatale del insufficientia mitral esseva detegibile in patientes con rhythm sinusal. Atrios gigante occurreva solmente in patientes con fibrillation atrial, frequentemente in association con insufficientia mitral, sed a vices in patientes con chirurgicamente pur stenosis mitral o grados minimal de insufficientia.

Le area del section transverse del major arteria pulmonar se correlationava netamente con le pression del arteria pulmonar e minus pronunciatemente con le resistentia pulmono-vascular e le dimension del orificio mitral. Un grado considerabile de dispersion in le varie correlationes anatomico-physiologic suggereva le existentia de un extense variabilitate ab un patiente al altere in le intrinsec proprietates elastic del atrio e del arteria.

Le diametro del aorta centro-ascendente non differeva ab le norma.

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HEMODYNAMIC ABNORMALITIES AND MITRAL STENOSIS


Study of the clinical course of 87 young patients with severe free aortic regurgitation due to rheumatic heart disease showed that at the end of 10 years 37 per cent were still able to lead quiet, nearly normal lives, and at the end of 20 years, 26 per cent still remained in this category. On the other hand, 38 per cent died within 10 years and 50 per cent within 20 years. The causes of death were recurrent rheumatic fever with congestive heart failure, angina pectoris, or subacute bacterial endocarditis. The appearance of congestive failure or angina pectoris, in particular, nocturnal angina at rest, was of ominous import with the duration of life after the onset of angina pectoris, in the majority of cases being 1 to 2 years. In these patients the anginal syndrome had certain characteristic features. The most severe attacks occurred at night and without provocation. The recumbent position appeared to be a predisposing factor. Sinus tachycardia, palpitation, generalized flushing of the skin, profuse sweating, and difficult respiration were essential features of the attack usually preceding the onset of pain. Because of intractable congestive heart failure and angina pectoris surgical relief with the plastic valve of Hufnagel was attempted in 14 patients. Favorable results were obtained in 40 per cent with extraordinary benefits in a few, so that this physiologic compromise is recommended for those patients in serious difficulty until a more promising procedure is available.

SAGALL
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