Hemodynamics in Ruptured Chordae Tendineae and Chronic Rheumatic Mitral Regurgitation

By William A. Baxley, M.D., J. Ward Kennedy, M.D., Bolling Feild, M.D., and Harold T. Dodge, M.D.

SUMMARY
Twenty-five patients with proven ruptured mitral chordae tendineae and 25 patients with chronic rheumatic mitral regurgitation were studied with quantitative biplane left-heart angiography. Compared to the rheumatic patients, the ruptured chordae group had strikingly smaller left atrial maximum volume (mean: 119 ± 47 vs 267 ± 131 ml/m² mean) and less compliant left atrium (mean: 1.61 ± 1.52 vs 5.14 ± 4.31 ml/mm Hg-m²); less impressively but significantly smaller left ventricular end-diastolic volume (mean: 142 ± 43 vs 188 ± 75 ml/m²) and mass (mean: 133 ± 27 vs 166 ± 50 mm/m²), and relatively insignificant tendency toward smaller left ventricular stroke volume and regurgitant flow per stroke. Ejection fractions, ventricular pressures, and forward flow per stroke were not significantly different. Within the ruptured chordae group, the duration of heart failure was not significantly correlated with any hemodynamic variable. These results indicate quantitatively the spectrum of abnormal hemodynamics in these two different etiologic forms of mitral insufficiency. The most striking differences between the two groups are in atrial dynamics. In spite of the usually more acute nature of ruptured chordae, significant compensatory dilatation and hypertrophy of the left ventricle is common in those patients. In both groups, large regurgitant flows with relatively normal ventricular function as expressed by ejection fraction is the usual finding.

Additional Indexing Words:
Left ventricular volumes Quantitative angiography Myocardial function
Valvular heart disease

Rupture of the mitral chordae tendineae may occur spontaneously and idiopathically or in association with cardiac disease, particularly bacterial endocarditis.1-7 The result is usually sudden mitral valve incompetence in a heart which, unless there were pre-existing disease, may have inadequate chronic compensatory mechanisms for tolerating a left ventricular volume overload. The clinical course is often marked by the abrupt onset of congestive heart failure. Previous clinical studies have revealed reasonably characteristic clinical findings in patients with mitral insufficiency from ruptured chordae. In addition to the systolic murmur, there is lack of severe cardiomegaly or left atrial enlargement by X-ray, and there is sinus rhythm and large “V” waves in the left atrial or pulmonary artery wedge pressure curve.6-9

Rheumatic mitral insufficiency in the adult, in contrast, usually has its beginnings in childhood or adolescence and is characteristically more chronic and indolent in its progression. Severe cardiomegaly, marked left atrial enlargement, and atrial fibrillation are common accompaniments of the advanced form of this disease.10 Furthermore, the left atrial or pulmonary artery wedge pressure “V” waves are often less abnormal in rheumatic patients with mitral regurgitation and severe cardiomegaly than in those patients with mitral regurgitation and less cardiac enlargement.9,11 In spite of these differences, either form of mitral regurgitation may cause the same end result: severe and possibly fatal congestive heart failure.
HEMODYNAMICS IN MITRAL REGURGITATION

The clinical and pathological features of these two distinct forms of mitral regurgitation have been well described. However, little hemodynamic data, other than pressure and cardiac output data, have been reported in patients with these specific etiologic forms of mitral regurgitation. In the past decade, quantitative angiocardiography has been utilized to measure left atrial volumes and left ventricular chamber volumes and masses in man with reasonable accuracy. In the present study, quantitative biplane angiocardiography was employed to measure these values in patients with congestive heart failure and pure mitral regurgitation. Additional parameters describing cardiac function were also calculated including left ventricular ejection fraction, forward and regurgitant flows, and left atrial compliance. Data from 25 adults with proven ruptured mitral chordae were thus compared to those of 25 adults with isolated chronic rheumatic mitral regurgitation, and both groups were compared to previously determined normal values. The results characterize the major hemodynamic features of these two etiologically separate forms of mitral incompetence in a more quantitative manner than has been heretofore reported.

Methods

The records of adults who had quantitative biplane angiocardiography at the University of Alabama Hospital, the University of Washington Hospital, or the Seattle V.A. Hospital were reviewed. Two groups of patients with pure mitral regurgitation and class III or IV congestive heart failure (New York Heart Association classification) were selected. Twenty-five patients (Group A) had isolated mitral incompetence due to ruptured mitral chordae tendineae, anatomically proven at either surgery (24 patients) or autopsy (one patient). Although three patients gave past histories of possible acute rheumatic fever, the appearance of the valves indicated chordal rupture to be the major pathological abnormality. Seven patients had histories of bacterial endocarditis 2–36 months previously as possible predisposing factors to chordal rupture; none had active endocarditis at the time of surgery. None had evidence of coronary disease or other cardiac or systemic disease. There were ten females and fifteen males. Age ranged from 21–69 years (mean 50.5). Sixteen were in sinus rhythm, and nine were in atrial fibrillation.

In contrast to Group A, Group B consisted of the first 25 adults reviewed who met the following criteria: 1) Isolated mitral valve incompetence with a definite past history of acute rheumatic fever. None had evidence of coronary disease, bacterial endocarditis, or other valve disease; none had mitral stenosis (no calculated valve areas < 2.0 cm²). 2) Evidence of chronicity as shown by a history of cardiac signs or symptoms dating back at least five years prior to study.

In Group B there were six females and 19 males. Age ranged from 14 to 65 years (mean 42.0). Seven patients were in sinus rhythm, and 18 had atrial fibrillation.

Each patient in both groups had right and transseptal and/or retrograde left heart catheterization with quantitative biplane left-heart angiocardiography. Pressures were recorded through fluid filled catheters with Statham transducers. Cardiac output was measured by the Fick method. Left ventricular end-diastolic pressure could not be accurately recorded in one patient in each group, and left atrial peak pressure and pulse pressure were not recorded in one patient in Group B.

Fifty to 75 ml of contrast material were then power-injected over 2–3 sec into the left atrium or left ventricle with filming at 6 or 12 frames/sec in the antero-posterior and lateral projections. From the films, left atrial volumes and left ventricular volumes and masses were calculated and expressed per m² body surface area. Because of occasional isolated difficulty in visualizing portions of angiograms the following were omitted: left atrial minimal volume in one group A patient, left ventricular end-diastolic volume and mass in one group A patient, and left ventricular mass in four group B patients. Because some patients were in atrial fibrillation, at least three cardiac cycles, excluding any premature beat or immediate post-premature beat, were averaged for the volume data in each patient. Furthermore, heart rates did not change more than 10% during the angiography or between the cardiac output measurements and the angiography. From these data, left ventricular ejection fraction and regurgitant flow were calculated. Average left atrial compliance was estimated as volume change/pressure change per cycle.

The hemodynamic data were punched on IBM cards and submitted to a digital computer for statistical analysis. In addition to the hemodynamic variables, the duration of heart failure symptoms in months was entered as a separate variable for Group A patients; this was then compared to the hemodynamic variables within that group. The Student's t-test and cumulative distribution plots were utilized to identify significant differences between the two groups for each set of variables.

Results

In table 1 are presented the major hemodynamic data for the two patient groups together with previously determined normal values. Differences in the two groups which were significant at the P < 0.01 level were limited to left atrial volumes (lower in group A), pressures (higher in group A), and compliance (lower in group A); and left ventricular end-diastolic volume and mass (lower in group A). Although left ventricular stroke volume and regurgitant flow per stroke tended to be larger in group B compared to group A, these differences were not statistically significant.

Circulation, Volume XLVIII, December 1973
### Table 1

**Hemodynamic Data**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Normal values* M ± sd</th>
<th>Ruptured chordae</th>
<th>Rheumatic group</th>
<th>t</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>Group A M ± sd</td>
<td>Range</td>
<td>N</td>
<td>Group B M ± sd</td>
</tr>
<tr>
<td>LA maximum volume (ml/m²)</td>
<td>35 ± 9</td>
<td>25 119 ± 47</td>
<td>62–277</td>
<td>25</td>
<td>267 ± 131</td>
</tr>
<tr>
<td>LA minimum volume (ml/m²)</td>
<td>17 ± 6</td>
<td>24 80 ± 44</td>
<td>33–240</td>
<td>25</td>
<td>207 ± 122</td>
</tr>
<tr>
<td>LA volume change (ml/m²)</td>
<td>18 ± 7</td>
<td>24 39 ± 15</td>
<td>11–70</td>
<td>25</td>
<td>60 ± 36</td>
</tr>
<tr>
<td>LA maximum pressure (mm Hg)</td>
<td>&lt;20</td>
<td>25 45 ± 16</td>
<td>10–80</td>
<td>24</td>
<td>30 ± 13</td>
</tr>
<tr>
<td>LA mean pressure (mm Hg)</td>
<td>&lt;12</td>
<td>25 22 ± 7</td>
<td>8–40</td>
<td>25</td>
<td>17 ± 8</td>
</tr>
<tr>
<td>LA pulse pressure (mm Hg)</td>
<td></td>
<td>25 34 ± 16</td>
<td>4–60</td>
<td>24</td>
<td>18 ± 11</td>
</tr>
<tr>
<td>LA compliance (ml/m²/mm Hg)</td>
<td>24 1.61 ± 1.52</td>
<td>0.57–6.00</td>
<td>24 5.14 ± 4.31</td>
<td>0.76–16.00</td>
<td>3.79</td>
</tr>
<tr>
<td>LV mass (gm/m²)</td>
<td>92 ± 16</td>
<td>24 133 ± 27</td>
<td>96–183</td>
<td>21</td>
<td>166 ± 50</td>
</tr>
<tr>
<td>LV end-diastolic volume (ml/m²)</td>
<td>70 ± 20</td>
<td>24 142 ± 43</td>
<td>54–256</td>
<td>25</td>
<td>188 ± 75</td>
</tr>
<tr>
<td>LV stroke volume (ml/m²)</td>
<td>45 ± 13</td>
<td>24 86 ± 29</td>
<td>32–157</td>
<td>25</td>
<td>109 ± 50</td>
</tr>
<tr>
<td>LV ejection fraction</td>
<td>0.67 ± 0.08</td>
<td>24 0.61 ± 0.14</td>
<td>0.36–0.82</td>
<td>25</td>
<td>0.58 ± 0.10</td>
</tr>
<tr>
<td>LV peak systolic pressure (mm Hg)</td>
<td>25 119 ± 19</td>
<td>80–155</td>
<td>25 114 ± 20</td>
<td>90–180</td>
<td>0.92</td>
</tr>
<tr>
<td>LV end-diastolic pressure (mm Hg)</td>
<td>&lt;12</td>
<td>24 14 ± 5</td>
<td>5–22</td>
<td>24</td>
<td>11 ± 4</td>
</tr>
<tr>
<td>Regurgitant flow/stroke (ml/m²)</td>
<td>0</td>
<td>24 59 ± 29</td>
<td>19–132</td>
<td>25</td>
<td>81 ± 49</td>
</tr>
<tr>
<td>Regurgitant flow/minute (l/m²)</td>
<td>0</td>
<td>24 5.19 ± 2.93</td>
<td>1.90–12.90</td>
<td>25</td>
<td>6.37 ± 388</td>
</tr>
<tr>
<td>Forward flow stroke (ml/m²)</td>
<td>45 ± 13</td>
<td>25 27 ± 10</td>
<td>9–47</td>
<td>25</td>
<td>28 ± 11</td>
</tr>
<tr>
<td>Cardiac index (l/m²)</td>
<td>&gt;2.50</td>
<td>25 2.30 ± 0.72</td>
<td>0.90–3.90</td>
<td>25</td>
<td>2.30 ± 0.58</td>
</tr>
</tbody>
</table>

Abbreviations: LA = left atrium; LV = left ventricle; N = number of patients; M = mean; sd = standard deviation.

*Normal volume and mass data were previously reported. 17,18

†Student t-test.
Figure 1 shows a comparison of the variables used to calculate average left atrial compliance: pulse pressure and volume change. Group A has the less compliant atria with higher pressure changes with less volume changes as compared to Group B.

Figure 2 reveals the comparison of left ventricular stroke and end-diastolic volumes with the two groups depicted differently. Ejection fraction is revealed by the distance of each point from the diagonal line, and the general similarity of ejection fraction for the two groups is evident. It is also evident that end-diastolic volumes in the ruptured chordae group tended to be smaller than in the chronic rheumatic group as is also demonstrated in table 1.

The cumulative distribution plots in figures 3-7 more graphically depict the differences between the two groups for the variables left atrial maximum volume, peak pressure, and compliance; and left ventricular end-diastolic volume and mass. In this form of data display, each individual point represents a measurement from one patient, and the horizontal distance between the two curves on each display represents the difference between the groups at various cumulative magnitudes of the variable. Clearly, the atrial parameters exhibit a more striking difference between the two groups than do the ventricular parameters.

It is of interest that for left atrial maximum volume as shown in figure 3, only one subject with ruptured chordae had a maximum volume exceeding 200 ml/m², whereas over 50% of those in the chronic rheumatic group had left atrial volumes in excess of 200 ml/m².

As illustrated in figure 5, left atrial compliance was greater than 4.0 ml/m²/mm Hg in more than half the subjects in the rheumatic group. Only three of the ruptured chordae group, or less than 15%, had left atrial compliance values exceeding 4.0 ml/m²/mm Hg.

Within the ruptured chordae group, the duration of heart failure in months did not correlate significantly with any of the hemodynamic variables ($P > 0.10$ for all). Heart failure duration ranged from 1-72 months (mean 17.6) within this group.
**Discussion**

The results of this study describe in quantitative fashion the hemodynamic abnormalities of mitral regurgitation of two distinctively different pathologic etiologies: ruptured chordae tendineae and chronic rheumatic heart disease. In spite of the general impression that most patients with ruptured mitral chordae do not have significant cardiomegaly, mean left ventricular end-diastolic volume for the patients in this group was 142 ml/m², over twice the normal value. Furthermore, all but one subject had end-diastolic volumes more than one standard deviation above normal. Also, some left ventricular hypertrophy had occurred, as shown by an elevated average mass value of 133 gm compared to normal mean of 92. These changes in mass and end-diastolic volume represent compensation for the large stroke volume (mean 86 ml/m²), caused by an average of 59 ml/m² per stroke, or 5.19 liters/min, regurgitation back into the left atrium. Thus, over two-thirds of the left ventricular stroke volume reflexes across the incompetent valve in this group. In spite of this reflux, most patients had normal myocardial function at least as revealed by mean ejection fraction of 0.61, with only five patients below 0.50. In this regard, Mitchell et al. reported that acute mitral regurgitation in animal models produces an increase in ejection fraction. In the present study, mean left ventricular end-diastolic pressure was elevated (14 mm Hg) and cardiac index diminished (2.30 liters/min/m²), reflecting the heart failure clinically present in all the patients selected for study. Thus, although cardiac index and ventricular filling pressure are abnormal, heart failure in this group of patients is due primarily to volume overload from mitral incompetence and not to poor muscle function.

Left atrial dynamics in ruptured chordae tendineae have been the subject of recent interest. Attention has focused on characteristic large left atrial "V" pressure waves and apparent lack of atrial enlargement on chest X-rays. Indeed, in comparing the hemodynamic data of the rheumatic patients with those of the ruptured chordae group as seen in table 1, the most striking differences are noted in atrial dynamics. The atrial maximal volume is significantly larger in the rheumatic
HEMODYNAMICS IN MITRAL REGURGITATION

Circulation, Volume XLVIII, December 1973

Figure 7
Cumulative distribution plot for left ventricular mass.

group as shown in figure 3, with the average of 267 ml/m² being over seven times average normal. Values as high as 605 ml/m² were noted. This is significantly more than in the chordae group. However, left atrial volumes in the ruptured chordae group were also increased greatly when compared to normal, the mean of 119 ml/m² being over three times normal mean, and the smallest atrial volume in this group was nearly two times normal. Also in this group, left atrial pulse pressure or pressure-change per beat averaged 34 mm Hg with values up to 60. When atrial pressure and volume data are combined to calculate compliance values, the rheumatic patients have significantly greater values, as shown in figures 1 and 5. In figure 1, the rheumatic patients are seen to have greater atrial volume change with less pressure change. All patients with atrial volume change greater than 70 ml/m² had rheumatic disease; all those with atrial pulse pressure greater than 44 mm Hg had ruptured chordae. In figure 5 the difference in the compliance values for the two groups is depicted by the cumulative distribution plot. Thus, these data quantitate the previously reported impression that rheumatic hearts have more left atrial dilatation and can “cushion” a given regurgitant flow with less pressure-rise than can hearts with ruptured chordae. In contrast, the relative “stiffness” of the left atra in ruptured chordae may cause higher pulmonary capillary pressure and hence more pulmonary congestive symptoms for a given regurgitant flow value than would be present with a more compliant atrium. On the other hand, a “stiffer” left atrium may, with its higher pressures, partially retard regurgitation through a given size valve defect by creating higher resistance to the regurgitant flow. It is of interest that Roberts et al. noted increased thickness of left atrial walls in hearts with ruptured mitral chordae.5

In the chronic rheumatic patients the mitral defect resulted in an average regurgitant flow of 81 ml/m² per stroke with values up to 238 ml/m². Expressed as regurgitant flow per minute, values ranged up to 16.52 liters/min/m² with an average of 6.37. Though these figures are larger than those of the ruptured chordae group, they are not significantly so. These large mitral regurigitant flows result in an increased stroke volume of 109 ml/m³ (mean), ranging up to 269. This figure is larger than that for the ruptured chordae group, but not significantly so. It is of interest that the mean ejection fraction, or ratio of stroke volume to end-diastolic volume as a measure of myocardial function,12 is normal in the rheumatic group. This value of 0.58 is similar to that in the ruptured chordae patients and the values extend down to a low of only 0.43. These relationships between left ventricular end-diastolic volume, stroke volume, and ejection fraction for the two groups are shown in figure 2. This finding of relatively normal ejection fraction for most patients with chronic rheumatic mitral regurgitation may be an unexpected finding, since these patients might have been anticipated as having more diminished myocardial function from old rheumatic myocarditis, or from many years of left ventricular volume overload. It may be that the low resistance to ejection or afterload is a significant factor in maintaining the ejection fraction within the normal range in patients with mitral insufficiency.

Mean left ventricular end-diastolic volume in the rheumatic group was 188 ml/m², over two and one-half times normal mean. Average mean values for left ventricular mass were 166 gm/m² compared to average normal of 92. Both parameters are significantly greater than those in the ruptured chordae group, as shown in figures 6 and 7. Thus, the rheumatic disease had dilated and hypertrophied the ventricles in these patients to a greater extent than had the ruptured chordae phenomenon in the other group of patients. There are several explanations for this observation. The tendency to larger regurgitant flows and stroke volumes in the rheumatic group may be a factor. Chronic left ventricular dilatation and hypertrophy apparently involve sarcomere replication, though the exact mechanism and time sequence are unknown.21 The
duration of the disease would appear to be an important factor in the adaptive changes noted in the two groups. The rheumatic patients all had had heart failure for over five years; the ruptured chordae patients had a mean duration of failure of only 17.6 months. Klughaupt et al. reported that patients with ruptured mitral chordae with longer duration of symptoms had more evidence of cardiomegaly than those with shorter symptom duration. However, in the present ruptured chordae group there was no correlation of duration of symptoms with any hemodynamic parameter. An additional factor may be the patients’ age at the onset of the volume overload, as previous animal studies have shown that the hearts of younger animals may hypertrophy more than those of older animals. Thus, rheumatic mitral regurgitation that develops in childhood may be more adequately compensated than adult-onset ruptured chordae. Also, the anatomic orifice-size of the mitral valve defect, through which the regurgitation occurs, may progress slowly over the years in rheumatic patients, allowing compensatory dilatation and hypertrophy to keep pace with the severity of mitral regurgitation to a greater extent in that group.

In spite of these compensatory changes and apparent good myocardial function, cardiac output and forward flow/minute stroke are both abnormally low and are virtually the same as the values for the ruptured chordae group. Furthermore, mean left atrial pressure is elevated in both groups. This low peripheral blood flow and elevated mean left atrial pressure reflect the clinical heart failure present in all the patients studied.

References

Hemodynamics in Ruptured Chordae Tendineae and Chronic Rheumatic Mitral Regurgitation
WILLIAM A. BAXLEY, J. WARD KENNEDY, BOLLING FEILD and HAROLD T. DODGE

Circulation. 1973;48:1288-1294
doi: 10.1161/01.CIR.48.6.1288

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1973 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/48/6/1288

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/