Relationship of Left Atrial Volume to Pulmonary Artery and Wedge Pressures in Mitral Stenosis

By Louis A. Soloff, M.D., Jacob Zatuchni, M.D., and George E. Mark, Jr., M.D.

The significance of left atrial volume has been studied in 20 consecutive patients with mitral stenosis. Cardiac catheterization was performed and resting pressures in the right atrium, right ventricle, and pulmonary artery, as well as wedge pressure, were determined. Angiocardiography was performed immediately after cardiac catheterization. On the basis of these data the relationship between the estimated left atrial volume and these pressures is stated.

ENLARGEMENT of the left atrium is a characteristic and constant finding in mitral stenosis. The degree of left atrial enlargement, which varies considerably from patient to patient, may be due to a single cause or a combination of factors such as left intratrial pressure, intrinsic left atrial disease, and hypervolemia.

This study attempts to determine the significance of left intratrial pressures as reflected in the pulmonary artery and pulmonary artery wedge pressures, in the production of the abnormal and variable volumes of the left atria in individuals with mitral stenosis.

METHODS AND MATERIALS

Twenty consecutive unoperated individuals with isolated mitral stenosis were studied. There were 16 females and 4 males, 25 to 60 years old. None was in heart failure at the time of study. All had a late diastolic rumbling murmur with presystolic accentuation, of variable intensity, at the apex. None had a mitral systolic murmur with an intensity greater than grade 1; indeed, 13 had no systolic murmur at all. M1 and P2 were loud. There was normal sinus rhythm in 14 and atrial fibrillation in 6. None had a lifting thrust at the apex. All showed on roentgen-ray study a variable degree of prominence of the pulmonary artery and enlargement of the left atrium and right ventricle. None had a roentgen diagnosis of left ventricular enlargement. The over-all size of the cardiac silhouette was reported as normal in 10 individuals and variably enlarged but never greatly so in the remaining 10. There was no instance of leftward deviation of the QRS in the extremities nor signs of left ventricular hypertrophy in the precordial leads. Finally, the pressure pulse obtained by cardiac catheterization disclosed no evidence suggestive of mitral regurgitation.

All patients were studied in the postabsorptive state and without sedation. The technic of venous cardiac catheterization was performed with the patient in the supine position. Resting pressures in the right atrium, right ventricle, and pulmonary artery were obtained. For the purpose of this study, only the pulmonary artery pressure is given. Pressure was also obtained after wedging the pulmonary artery by catheter, according to the method of Hellem and his associates.2 Pressure so obtained was designated pulmonary capillary venous pressure, according to custom, but only if blood from this site had an oxygen content approximating that from the femoral artery. All pressures were recorded with a Statham strain-gage electromanometer and Cambridge direct-writing oscillograph.

The mean pulmonary artery pressure and the mean pulmonary capillary venous pressure were determined by planimetric integration. In our laboratory, the normal mean pulmonary artery pressure is 13 mm. Hg and the mean capillary venous pressure is no greater than 12 mm. Hg.

Immediately after cardiac catheterization, angiocardiography was done with the patient in the sitting position. We used a simultaneous biplane stereoscopic angiocardiographic apparatus devised by Dr. W. Edward Chamberlain.* This technic and the method of defining the left atrial volume have been previously described.1

RESULTS

The pertinent data are given in table 1. The subjects are listed according to increasing angiocardiographic volume of the left atrium.

In this series, the smallest left atrial volume was 270 ml. and the largest 918 ml. Three were less than 300 ml., 5 between 300 and 400 ml., 3 between 400 and 500 ml., 5 between 500 and 600 ml., and 4,680 ml. or more. The mean

From the departments of Medicine, Temple University School of Medicine and Hospital, and Episcopal Hospital, Philadelphia, Pa.

Supported in part by a grant from the Heart Association of Southeastern Pennsylvania.

TABLE 1.—Clinical Data and Pulmonary Vascular Pressures Listed According to Increasing Left Atrial "Volume"

<table>
<thead>
<tr>
<th>Subject</th>
<th>Sex</th>
<th>Age</th>
<th>Rhythm</th>
<th>From onset of R.F. (yr.)</th>
<th>Known heart disease (yr.)</th>
<th>Angio-cardiographic left atrial &quot;volume&quot; (ml.)</th>
<th>Pressure (mm. Hg)</th>
<th>Mean pulmo-venous pressure (mm. Hg)</th>
<th>Mean pulmonary venous pressure (mm. Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. W.O.</td>
<td>F</td>
<td>33</td>
<td>S</td>
<td>27</td>
<td>7</td>
<td>270</td>
<td>39</td>
<td>34</td>
<td>18</td>
</tr>
<tr>
<td>2. M.T.</td>
<td>F</td>
<td>39</td>
<td>S</td>
<td>29</td>
<td>4</td>
<td>281</td>
<td>39</td>
<td>18</td>
<td>15</td>
</tr>
<tr>
<td>3. C.P.</td>
<td>F</td>
<td>35</td>
<td>S</td>
<td>22</td>
<td>22</td>
<td>293</td>
<td>10</td>
<td>9</td>
<td>14</td>
</tr>
<tr>
<td>4. E.F.</td>
<td>F</td>
<td>35</td>
<td>S</td>
<td>3</td>
<td>3</td>
<td>318</td>
<td>25</td>
<td>19</td>
<td>14</td>
</tr>
<tr>
<td>5. G.I.</td>
<td>F</td>
<td>37</td>
<td>S</td>
<td>*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. L.S.</td>
<td>F</td>
<td>42</td>
<td>S</td>
<td>9</td>
<td>9</td>
<td>345</td>
<td>18</td>
<td>11</td>
<td>24</td>
</tr>
<tr>
<td>7. C.W.</td>
<td>F</td>
<td>45</td>
<td>S</td>
<td>40</td>
<td>22</td>
<td>351</td>
<td>28</td>
<td>24</td>
<td>34</td>
</tr>
<tr>
<td>8. J.C.</td>
<td>F</td>
<td>33</td>
<td>AF</td>
<td>23</td>
<td>15</td>
<td>399</td>
<td>42</td>
<td>20</td>
<td>38</td>
</tr>
<tr>
<td>9. M.H.</td>
<td>F</td>
<td>25</td>
<td>S</td>
<td>x</td>
<td>2</td>
<td>416</td>
<td>38</td>
<td>28</td>
<td></td>
</tr>
<tr>
<td>10. F.P.</td>
<td>F</td>
<td>47</td>
<td>S</td>
<td>25</td>
<td>7</td>
<td>422</td>
<td>68</td>
<td>33</td>
<td></td>
</tr>
<tr>
<td>11. M.G.</td>
<td>F</td>
<td>31</td>
<td>S</td>
<td>24</td>
<td>24</td>
<td>480</td>
<td>51</td>
<td>20</td>
<td></td>
</tr>
<tr>
<td>12. E.D.</td>
<td>F</td>
<td>60</td>
<td>AF</td>
<td>37</td>
<td>18</td>
<td>504</td>
<td>26</td>
<td>14</td>
<td></td>
</tr>
<tr>
<td>13. M.S.</td>
<td>F</td>
<td>46</td>
<td>AF</td>
<td>16</td>
<td>10</td>
<td>520</td>
<td>50</td>
<td>22</td>
<td></td>
</tr>
<tr>
<td>14. A.M.</td>
<td>M</td>
<td>41</td>
<td>S</td>
<td>1</td>
<td>1</td>
<td>520</td>
<td>34</td>
<td>24</td>
<td></td>
</tr>
<tr>
<td>15. J.S.</td>
<td>F</td>
<td>59</td>
<td>AF</td>
<td>x</td>
<td>47</td>
<td>570</td>
<td>20</td>
<td>12</td>
<td></td>
</tr>
<tr>
<td>16. A.N.</td>
<td>M</td>
<td>46</td>
<td>S</td>
<td>x</td>
<td>1</td>
<td>595</td>
<td>55</td>
<td>30</td>
<td></td>
</tr>
<tr>
<td>17. L.G.</td>
<td>F</td>
<td>44</td>
<td>AF</td>
<td>x</td>
<td>30</td>
<td>680</td>
<td>29</td>
<td>17</td>
<td></td>
</tr>
<tr>
<td>18. A.R.</td>
<td>F</td>
<td>37</td>
<td>AF</td>
<td>30</td>
<td>15</td>
<td>720</td>
<td>26</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>19. T.J.</td>
<td>M</td>
<td>49</td>
<td>S</td>
<td>38</td>
<td>38</td>
<td>756</td>
<td>72</td>
<td>31</td>
<td></td>
</tr>
<tr>
<td>20. D.L.</td>
<td>M</td>
<td>39</td>
<td>S</td>
<td>30</td>
<td>1</td>
<td>918</td>
<td>26</td>
<td>18</td>
<td></td>
</tr>
</tbody>
</table>

S = normal sinus rhythm
AF = atrial fibrillation
* = No history of rheumatic fever.

pulmonary artery pressure varied from 10 to 72 mm. Hg. All but one exceeded the normal. This patient (C.P.) was a 35-year-old Negro female in the ninth month of pregnancy. She had no cardiac symptoms since her last bout of dyspnea 7 years ago. Five had a mean pulmonary artery pressure of 51 mm. Hg or more. In these 5, the left atrial volume varied from 322 to 756 ml. The largest left atrial volume (918 ml.) was associated with a mean pulmonary artery pressure of 26 mm. Hg. This patient (D.L.) was a 39-year-old man who was admitted into the hospital because of pneumonia. For 1 year he had experienced recurrent bouts of tachycardia. The highest pressure (61 mm. Hg) of those with a relatively small left atrial volume was found in patient G.I. who entered the hospital because of a bout of nocturnal dyspnea. A similar but more prolonged episode of dyspnea had occurred 3 years previously during the sixth month of pregnancy. She had no dyspnea in the intervening 3 years.

The correlation coefficient between left atrial volume and mean pulmonary artery pressure and its probability as a test of independence of the observed measurements of these 2 variables are shown in table 2.

The mean pulmonary capillary venous pressure exceeded our normal of 12 mm. Hg in 16 of the 20 patients. The greatest pressure (34 mm. Hg) was found in patient W.O. with the smallest left atrial volume. She had 2 episodes of acute dyspnea in the recent past and her effort tolerance was subnormal. A similar high pressure was found in patient T.J. with a left atrial volume of 756 ml. The largest left atrial volume (918 ml.) was associated with a mean pulmonary capillary venous pressure of only 18 mm. Hg.

The correlation coefficient between left atrial volume and mean pulmonary capillary venous pressure and its probability as a test of independence of the observed measurements of these 2 variables are also shown in table 2.

**DISCUSSION**

Our data fail to reveal any significant relationship between left atrial volume and either the mean pulmonary artery pressure or the mean pulmonary capillary venous pressure. Nor is there any association of rising pressures
with increasing volumes. Any relationship that may be present is completely obscured by other more potent factors determining the degree of left atrial enlargement.

The finding of markedly different left atrial volumes in individuals with approximately equal pulmonary vascular pressures suggests that factors other than pressure are the immediate cause of left atrial enlargement. One such factor may be change in elasticity of the left atrial wall. Decrease in elasticity of the left atrial wall, however, could be due either to the effect of an abnormally elevated pressure operating over a prolonged time or to factors independent of the back pressure produced by mitral block.

The effect on left atrial volume of chronically elevated pressures can be determined definitively only by longitudinal studies over a prolonged time. Such studies are, as yet, not available. Our preliminary studies of a few patients, studied for slightly more than 3 years, fail to reveal any significant change in left atrial volume. Indeed, we have conventional roentgenograms of individuals with mitral stenosis that reveal no detectable change in over-all cardiac size or shape for over 2 decades, but admittedly the left atrial volume is difficult to estimate by such relatively crude studies.³

An indirect method of attempting to assess the effect of time is to determine the relationship of left atrial volume and pulmonary vascular pressures to the age of patient and to known duration of rheumatic state and of rheumatic heart disease. This is possibly an oversimplification of the problem because of the uncertainty of the duration of the level of the pulmonary vascular pressures found at the time of study. Nevertheless, a positive correlation would suggest a definite relationship, whereas very wide variations in opposite directions point very strongly to factors other than pressure due to mitral block as more important in determining the degree of left atrial enlargement. We have therefore analyzed our data with respect to age of patient and to duration both of the rheumatic state and of known rheumatic heart disease.

Duration of the rheumatic state is defined as the difference between the present age and the age at onset of rheumatic fever. Because of the frequent absence of a history of rheumatic fever and because we are concerned specifically with its cardiac component, duration of known heart disease was also determined. This is defined as the difference between the present age and the age at which heart disease was first recognized and thereafter constantly present.

No significant relation was found between these factors and either the left atrial volume or the pulmonary vascular pressures (table 2). An additional 18 cases, not included in this study, were analyzed to determine any trend toward possible relation between age and left atrial volume and again no significant relationship was found (r = 0.495, p > 0.80). Likewise, in an additional 11 cases, no significant relationship was found between mean pulmonary capillary venous pressure and duration of known heart disease (r = −0.183, p > 0.50).

This lack of correlation between volume and pressure is most clearly indicated by those patients (C.P., L.S., J.S., and A.R.) who have an enlarged left atrium and normal pulmonary capillary venous pressure. Under such circumstances, one might assume either that there never was preceding mitral block due to mitral stenosis or that the pressure dropped without restoration of the left atrial volume to normal. The latter appears untenable because there are individuals with enlarged left atria who have a normal cardiac output. Had the hypothetical pulmonary capillary venous hypertension actually been due to structural mitral stenosis, its fall to normal would result in a diminished cardiac output inasmuch as the structural stenosis in the absence of surgery is permanent.

Two other causes for left atrial enlargement, among other possibilities, are rheumatic atrial myocarditis and cardiac failure of any cause.

Rheumatic atrial myocarditis was previously the dominant explanation for left atrial enlargement in rheumatic heart disease. This hypothesis has abundant support in anatomic studies of the heart.⁴ Indeed, Bramwell and Duguid,⁵ in a study of aneurysmal dilatation of the left atrium, concluded that the only obvious cause for such enlargement was "fibrosis of the auricular wall... due to an ischemic
necrosis associated with a rheumatic infection..." They discounted the mechanical influence of any mitral valvular lesion because stenosis or regurgitation may be present and marked enlargement of the left atrium is rare, while both stenosis and regurgitation are relatively common. We ourselves have found no correlation between the size of the left atrium during life and the degree of mitral stenosis as reported by the surgeon, regardless of the age of the patient or the duration of the rheumatic state. Indeed, the finding of very large left atria with but mildly elevated pressures in young people makes it difficult to consider anything but localized left atrial disease independent of pressure as the major cause of the enlargement. In those with normal pressure and normal cardiac output, atrial disease unrelated to mitral block appears to be the only possible cause.

Cardiac failure is a cause for left atrial enlargement in individuals with mitral stenosis. Our unpublished data indicate that the left atrium is larger during a bout of failure than after compensation. Our studies also show that the pulmonary vascular pressures are higher during failure than after compensation. This increase in pressure is not due to structural mitral stenosis, which is irreversible by medical therapy, but is due rather to hypervolemia, myocardial failure, or both. It is possible that prolonged myocardial failure leads to irreversible increase in volume of the left atrium that is already the seat of intrinsic disease. Indeed, numerous studies in the past indicate that the largest hearts occur in individuals who have had the most frequent and prolonged bouts of failure.

**SUMMARY**

The left atrial volume, pulmonary artery and pulmonary capillary venous pressures were measured in 20 consecutive unoperated individuals with isolated mitral stenosis. No significant relationship was found between left atrial volume and pulmonary artery wedge pressures. No significant relationship was found between left atrial volume and pulmonary vascular pressures and the age of the patient or the duration of the rheumatic state or of known rheumatic heart disease. Any relationship present between these parameters is obscured by other more potent factors.

**Acknowledgment**

We are grateful to Mr. Herman Siplet, A.B., of the Samuel S. Fels Research Institute, Temple University School of Medicine, for the statistical analysis.

**SOMMARIO IN INTERLINGUA**

Le volumine sinistro-atrial e le pressiones pulmono-arterial e pulmono-capillari venose eseva mesurata in 20 consecutiva non-operate cases de isolata stenosis mitral. Nulle significative relationes eseva trovate inter le volumine sinistro-atrial e le pressiones cuneate de arteria pulmonar. Nulle significative relationes eseva trovate inter le volumine sinistro-atrial e le pressiones pulmo-vascular e le etate del patiente o le duration de su stato rheumatic o cognoscite morbo cardiac rheumatic. Si il existe relationes inter le factores investigate, ille relationes es obseurate per alte re factores de plus grande potentia.

**REFERENCES**

Relationship of Left Atrial Volume to Pulmonary Artery and Wedge Pressures in Mitral Stenosis
LOUIS A. SOLOFF, JACOB ZATUCHNI and GEORGE E. MARK, JR.

Circulation. 1957;15:430-433
doi: 10.1161/01.CIR.15.3.430

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1957 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/15/3/430

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/