Cardiac Chamber Volumes and their Significance in Rheumatic Heart Disease with Isolated Mitral Stenosis

By Louis A. Soloff, M.D., and Jacob Zatuchni, M.D.

The total capacity of the heart and the volume of each of its chambers were determined by the technic of simultaneous biplane stereoscopic venous angiocardiography in 18 persons with surgically confirmed, isolated severe mitral stenosis. Although cardiac capacity and chamber volumes varied greatly, the left atrium was almost always the largest chamber. The left ventricle was not always the smallest chamber. In fact, it was larger than the right ventricle in 7 of the 18 and was the only chamber to be significantly correlated with total cardiac capacity greater than 1,000 ml. The right ventricular volume was found to be significantly correlated with its systolic pressure, or with the pulmonary artery systolic or mean pressures. Disproportionate enlargement of the right atrium can occur in the absence of tricuspid stenosis. Finally, atrial fibrillation was commonly present in patients with a large total cardiac capacity.

Roentgenologic measurements of the heart should ideally provide information concerning the volume of its chambers and the thickness of its walls during various phases of the cardiac cycle. Such information is as yet not available. Nevertheless, all measurements, whether linear, planigraphic, or volumetric, have meaning only insofar as they approximate this ideal information. Measurements of the heart on conventional roentgenograms are concerned with over-all size of the cardiac silhouette. These measurements combined with the shape of the cardiac silhouette are used to judge size of the chambers and to a lesser extent thickness of its walls. Such deductions are not inherent in the roentgenogram itself but are based upon years of study by physicians who correlated roentgenographic findings with anatomic specimens. There are, however, gross differences between the anatomic specimen and the cardiac silhouette. There are changes in position and in elasticity, but most important the anatomic specimen is devoid of the intracardiac pressures that are so important in molding the shape and size of the cardiac chambers. It is equally difficult to visualize the interlocking relationships of the chambers in the anatomic specimen. It is for these reasons that precise knowledge of the size of the cardiac chambers is not obtainable from study of either the conventional roentgenogram or the anatomic specimen. For instance, we have previously demonstrated that esophageal displacement fails to define the degree of left atrial enlargement, that the small aorta in mitral stenosis may be an illusion, and that the left atrium may be strikingly enlarged in diseases affecting predominantly the left ventricle. This knowledge was obtained by angiocardiography, a technic that makes possible visualization of individual chambers and the thickness of their walls.

We now propose to employ this technic to determine the volume of the cardiac chambers in rheumatic heart disease with isolated mitral stenosis.

Materials and Methods

Eighteen persons with inactive rheumatic heart disease and isolated mitral stenosis were studied preoperatively. The criteria for the diagnosis of isolated mitral stenosis have been described elsewhere.4 Severe mitral stenosis was found in each person at surgery. Mitral regurgitation and other valvular lesions were not detected. There were 15 females, 20 to 49 years old, and 3 males, 33 to 48 years old. Ten had normal sinus rhythm and 8 had atrial fibrillation. None had clinical evidence of cardiac failure when studied. Cardiac catheterization was done in 17 and revealed no evidence of a gradient across the tricuspid valve.

From the Department of Medicine, Temple University Medical School and Hospital, Philadelphia, Pa. Supported in part by a grant from the Heart Association of Southeastern Pennsylvania.
### Table 1.—Volume of Each Cardiac Chamber in Persons with Rheumatic Heart Disease and Isolated Mitral Stenosis Arranged According to Total Cardiac Capacity

<table>
<thead>
<tr>
<th>Subject</th>
<th>Sex</th>
<th>Age</th>
<th>NSR*</th>
<th>AF†</th>
<th>Mitral orifice</th>
<th>Right atrium</th>
<th>Right ventricle</th>
<th>Pulmonary artery</th>
<th>Chamber volume (mL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. W.D.</td>
<td>F</td>
<td>33</td>
<td>+</td>
<td></td>
<td>5 x 2 mm.</td>
<td>5</td>
<td>85</td>
<td>64</td>
<td>96</td>
</tr>
<tr>
<td>2. J.P.</td>
<td>F</td>
<td>22</td>
<td>+</td>
<td>2 x 3 mm.</td>
<td>2</td>
<td>34</td>
<td>4</td>
<td>30</td>
<td>17</td>
</tr>
<tr>
<td>3. M.D.</td>
<td>F</td>
<td>20</td>
<td>+</td>
<td></td>
<td>Pencil</td>
<td>5</td>
<td>30</td>
<td>11</td>
<td>24</td>
</tr>
<tr>
<td>4. F.K.</td>
<td>F</td>
<td>42</td>
<td>+</td>
<td>5</td>
<td>Match-stick</td>
<td>10</td>
<td>50</td>
<td>8</td>
<td>52</td>
</tr>
<tr>
<td>5. E.S.</td>
<td>M</td>
<td>41</td>
<td>+</td>
<td>&lt; 1 finger</td>
<td>10</td>
<td>55</td>
<td>8</td>
<td>52</td>
<td>24</td>
</tr>
<tr>
<td>6. E.H.</td>
<td>F</td>
<td>42</td>
<td>+</td>
<td>Cigarette</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>7. C.P.</td>
<td>F</td>
<td>28</td>
<td>+</td>
<td></td>
<td>String-like bands</td>
<td>1</td>
<td>64</td>
<td>2</td>
<td>53</td>
</tr>
<tr>
<td>8. L.N.</td>
<td>F</td>
<td>45</td>
<td>+</td>
<td>Tip</td>
<td>10</td>
<td>49</td>
<td>12</td>
<td>43</td>
<td>30</td>
</tr>
<tr>
<td>9. M.T.</td>
<td>F</td>
<td>39</td>
<td></td>
<td>8 x 3 mm.</td>
<td>4</td>
<td>65</td>
<td>2</td>
<td>56</td>
<td>39</td>
</tr>
<tr>
<td>10. T.H.</td>
<td>F</td>
<td>32</td>
<td></td>
<td>7 x 2 mm.</td>
<td>10</td>
<td>50</td>
<td>8</td>
<td>52</td>
<td>41</td>
</tr>
<tr>
<td>11. S.N.</td>
<td>F</td>
<td>36</td>
<td></td>
<td></td>
<td>Markedly contracted</td>
<td>13</td>
<td>68</td>
<td>14</td>
<td>72</td>
</tr>
<tr>
<td>12. I.A.</td>
<td>F</td>
<td>45</td>
<td></td>
<td></td>
<td>Markedly contracted</td>
<td>6</td>
<td>25</td>
<td>6</td>
<td>26</td>
</tr>
<tr>
<td>13. M.I.</td>
<td>F</td>
<td>26</td>
<td></td>
<td>5 mm.</td>
<td>10</td>
<td>45</td>
<td>0</td>
<td>48</td>
<td>30</td>
</tr>
<tr>
<td>14. J.C.</td>
<td>F</td>
<td>33</td>
<td></td>
<td>4 x 4 mm.</td>
<td>7</td>
<td>48</td>
<td>10</td>
<td>67</td>
<td>42</td>
</tr>
<tr>
<td>15. T.J.</td>
<td>M</td>
<td>48</td>
<td></td>
<td>2 matches</td>
<td>7</td>
<td>121</td>
<td>7</td>
<td>112</td>
<td>72</td>
</tr>
<tr>
<td>16. L.D.</td>
<td>M</td>
<td>33</td>
<td></td>
<td>Tip</td>
<td>10</td>
<td>32</td>
<td>8</td>
<td>28</td>
<td>18</td>
</tr>
<tr>
<td>17. E.M.</td>
<td>F</td>
<td>41</td>
<td></td>
<td></td>
<td>11</td>
<td>43</td>
<td>0</td>
<td>53</td>
<td>33</td>
</tr>
<tr>
<td>18. K.C.</td>
<td>F</td>
<td>49</td>
<td></td>
<td></td>
<td>7</td>
<td>86</td>
<td>8</td>
<td>86</td>
<td>59</td>
</tr>
</tbody>
</table>

*Normal sinus rhythm.
†Atrial fibrillation.

Simultaneous biplane (posteroanterior and left lateral) stereoscopic venous angiocardiograms, at a tube-to-film distance of 100 cm., were obtained every 0.7 second for at least 30 exposures. The duration of each exposure was 0.1 second.

The maximal size of each of the cardiac chambers was obtained by determining its outermost limits on the sequential venous angiocardiograms and reconstruction by tracing on onionskin paper.

Area of each of the cardiac chambers in the frontal plane was determined by use of a compensating polar planimeter. Depth is the largest diameter of the chamber in the lateral projection, perpendicular to the frontal plane. Volume of each cardiac chamber is the product of the area in the frontal plane and the depth in the lateral plane.

Other methods for determination of volume were also used. These included (1) the product of the area in the lateral plane and the diameter in the frontal plane, (2) the product of the areas in the frontal and lateral planes raised to the 3/4 power, and (3) the sum of volumes of small cylindrical components of each of the chambers in both planes. These technics were discarded, following study of all subjects, in favor of the method given because of (1) difficulty in determining the frontal plane a single representative diameter, (2) prejudice in using product of areas of an assumed common diameter, and (3) unequal projection of areas in both planes, which makes the creation of equivalent cylinders impossible in this type of cardiac disease. The method used is similar to that conventionally employed to determine volume of the cardiac silhouette.5

No correction factor for distortion produced by a tube-to-film distance of 100 cm. was used because of difficulty, if not impossibility, of obtaining...
equally important measurements inherent in its calculation. A factor to correct for distortions of volume produced by shape was also not employed because either the chamber did not conform significantly with any known geometric figure that could be analyzed mathematically or, regardless of similarity to a geometric structure in 1 plane, its entire shape and uniformity could not be determined even from 2 projections. Parenthetically, it can be stated that even for the heart as a whole, we have not encountered a mathematician who agrees with the usual statement that the heart is suggestive of a "combined sphere and a transversally placed paraboloid" and who is willing to accept Rohrer's correction factor of 0.63 or any other mathematical equation based on similar assumptions of shape.

For all these reasons, we have preferred to report each chamber volume as the product of its area in the frontal plane and greatest linear diameter perpendicular to it. Total cardiac volume, as used in this study, is the sum of the volumes of the 4 chambers. Obviously, it is representative of capacity rather than total cardiac volume, for solid mass is not included in its measurement.

RESULTS

Table 1 gives the actual volume of each of the cardiac chambers arranged according to total cardiac capacity. Total cardiac capacity varied greatly and showed no significant relationship either to age or to duration of rheumatic heart disease. Atrial fibrillation was correlated best with total cardiac capacity and was more frequent the larger the total cardiac capacities.

Chamber volumes and their relationship to each other also varied greatly. No significant relationship was found between the right atrial volume and its pressure, or between the right ventricular volume and either its end-diastolic pressure or the mean pulmonary capillary venous pressure. A suggestive relationship was found between the right ventricular systolic pressure and its volume (r = 0.4657, p > 0.05). Further study, however, showed that there was no relationship between these 2 variables with observed values of right ventricular volume less than 165 ml. (r = 0.1655, p > 0.70) and a significant one with observed values of 165 ml. or more (r = 0.7716, p < 0.01). Such right ventricular volumes were also significantly correlated with the pulmonary artery systolic pressure (r = 0.7771, p < 0.01) and with the pulmonary artery mean pressure (r = 0.7857, p < 0.01).

The volume of the left atrium was almost always larger than that of any other cardiac chamber. It constituted 28.9 to 59.9 per cent of the total cardiac capacity. There was a tendency for the larger total cardiac capacities to be associated with larger left atrial volumes that were frequently disproportionately increased. However, a small total cardiac capacity did not exclude a large left atrium. Conversely, a large total cardiac capacity did not exclude a relatively small left atrium.

The left ventricular volume was the smallest of the chamber volumes in only 55.6 per cent and was larger than the right ventricular volume in 38.9 per cent.

Finally, a significant relationship was demonstrated between the total cardiac capacity and the volume of each chamber except the left ventricle. However, if the total cardiac capacity was less than 1,000 ml., it was significantly correlated only with the left atrial volume and if greater than 1,000 ml. only with the left ventricular volume. The correlation coefficients and their probabilities may be seen in table 2.

DISCUSSION

These findings indicate that the left atrium is almost always the largest chamber of the heart of a person with rheumatic heart disease and isolated mitral stenosis. This fact has been generally assumed but has not actually been previously demonstrated during life. This chamber may at times occupy more than half the total capacity of the heart. The corollary of this statement is that the left atrium does not empty completely in systole and must indeed have the greatest residual systolic volume of all the cardiac chambers, a fact readily demonstrable by angiocardiology. Indeed, if this chamber is very large, there may be no appreciable change in its outer contour during various phases of the cardiac cycle. In fact, we have been unable to detect, by high-speed cinematographic stud-
ies, any motion of the lateral and cephalic borders of the left atrium in the lateral projection. Emptying of the left atrium under these circumstances is accomplished for practical purposes entirely by the piston-like action of the atrioventricular valves.

There is, however, no constant relationship in isolated mitral stenosis between the volume of the left atrium and that of the other cardiac chambers. Thus, the ratio of the volume of the left atrium to that of the right ventricle varied from 1.2 to 8.9, a 7-fold difference. Wide variation was also found when the left atrial volume was related to that of the right atrium or to the combined volumes of the right atrium and right ventricle. Such marked variation strongly supports the view that no single factor is equally effective in the production of enlargement of the left atrium, right ventricle, and right atrium. Back pressure from mitral block is obviously inadequate by itself to explain this variation.

We have previously reported that left atrial volume in isolated mitral stenosis cannot be correlated significantly with any pressure parameter, or with age or duration of the rheumatic state. On the other hand, we have also reported that pulmonary artery size does correlate significantly with the mean pulmonary artery pressure. We report now a significant correlation between the right ventricular systolic pressure and its volume for observed values of 165 ml. or more, and also between such volumes and the pulmonary artery systolic and mean pressures.

The demonstration of disproportionate enlargement of the right atrium in the absence of tricuspid stenosis raises serious doubt of the reliability of this sign which has been so used by others. Conceivably, it may be due to tricuspid incompetency not ordinarily detectable but suggested by the finding of an elevated right atrial pressure. It may also represent change in elasticity either primary or secondary to bouts of hypervolemia or both, for a significant correlation was found between the volume of the right atrium and total cardiac capacity but not between it and its pressures.

The interplay of these various factors in the production of chamber enlargement is also demonstrated by their relationship to right ventricular volume. Here, a significant relationship was found between the total cardiac capacity and the volume of the right ventricle. Yet, increase in the latter above 165 ml. is, as stated before, also significantly related to its pressure.

For these reasons, it appears that pressure, change in elasticity, and hypervolemia act variably and do not uniformly affect the entire heart. Moreover, they do not necessarily act together during all phases of the life cycle of rheumatic heart disease. Thus, the fact that the total cardiac capacity when less than 1,000 ml. correlates only with the left atrial volume is readily understandable, if enlargement of the left atrium occurs first and is, thereby, the major cause for increasing the total capacity of the heart. Right ventricular enlargement, on the other hand, is partly dependent upon

<table>
<thead>
<tr>
<th>Total cardiac capacity</th>
<th>Right atrium</th>
<th>Right ventricle</th>
<th>Left atrium</th>
<th>Left ventricle</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cardiac capacity</td>
<td>r = 0.7083*</td>
<td>r = 0.6845*</td>
<td>r = 0.8748*</td>
<td>r = 0.4108</td>
</tr>
<tr>
<td>less than 1,000 ml.</td>
<td>p &lt; 0.01</td>
<td>p &lt; 0.01</td>
<td>p &lt; 0.001</td>
<td>p &gt; 0.05</td>
</tr>
<tr>
<td>Total cardiac capacity</td>
<td>r = 0.0949</td>
<td>r = 0.2399</td>
<td>r = 0.8521*</td>
<td>r = 0.0175</td>
</tr>
<tr>
<td>greater than 1,000 ml.</td>
<td>p &gt; 0.70</td>
<td>p &gt; 0.40</td>
<td>p &lt; 0.001</td>
<td>p &gt; 0.90</td>
</tr>
<tr>
<td></td>
<td>r = 0.4124</td>
<td>r = 0.4479</td>
<td>r = 0.0534</td>
<td>r = 0.9581*</td>
</tr>
<tr>
<td></td>
<td>p &gt; 0.50</td>
<td>p &gt; 0.50</td>
<td>p &gt; 0.90</td>
<td>p &lt; 0.05</td>
</tr>
</tbody>
</table>

*Significant.
pulmonary artery pressure, which is not related to left atrial volume. Finally, the left ventricle may not be significantly enlarged until hypervolemia is or has been present. Hypervolemia may also disproportionately distend the left ventricle because the elasticity of the other chambers has been seriously compromised. This may be the reason that the total cardiac capacity when greater than 1,000 ml. correlates only with the left ventricular volume.

Perhaps for these reasons, left ventricular volume in severe mitral stenosis is not invariably decreased. In one half of the group, it was 150 ml. or more. Moreover, it is not always the smallest chamber of the heart in mitral stenosis, for in 7 of the 18, it was larger than the right ventricle. It is also possible that the left ventricle may be slightly enlarged in mitral stenosis because mitral regurgitation was present during the initial phase of rheumatic heart disease and reappears or increases whenever resistance to left ventricular forward flow is temporarily increased.

SUMMARY AND CONCLUSIONS

A study was made of 18 persons with rheumatic heart disease and isolated severe mitral stenosis, confirmed at surgery, to determine the volume of each of the cardiac chambers opacified by the technic of simultaneous biplane stereoscopic venous angiocardiography.

The volume of each chamber is represented by the product of the area in the frontal plane and the greatest linear diameter perpendicular to it. Total cardiac capacity is the sum of the chamber volumes.

Each of the chambers varied greatly in volume, right atrium 96 to 395 ml., right ventricle 63 to 311 ml., left atrium 209 to 582 ml., and left ventricle 41 to 207 ml. Total cardiac capacity varied from 595 to 1,341 ml.

Almost always the volume of the left atrium exceeded that of the other chambers, and constituted 28.9 to 59.9 per cent of the total cardiac capacity.

Wide variation was found between the volume of the left atrium and that of the right ventricle or right atrium or both.

Disproportionate increase in volume of the right atrium not due to tricuspid stenosis may occur in severe mitral stenosis.

The volume of the left ventricle varied 5 fold, was not invariably small, and, indeed, was not always the smallest chamber.

A significant relationship was found between the right ventricular volume for observed values of 165 ml. or more and its systolic pressure, and also with the pulmonary artery systolic and mean pressures.

A significant correlation was demonstrated between the total cardiac capacity and the volume of each chamber except the left ventricle. However, if the total cardiac capacity was less than 1,000 ml., it was significantly correlated only with the left atrial volume, and, if greater than 1,000 ml., only with the left ventricular volume.

Finally, the larger total cardiac capacities were commonly associated with atrial fibrillation.

ACKNOWLEDGMENT

We thank Professor Walter G. Lawton, Head of the Department of Mathematics, Temple University, for his mathematical guidance, and Herman Siplet, B.S., Fels Foundation, Temple University, for his statistical analysis. We also thank George E. Mark, Jr., M.D., Associate Professor of Medicine, Temple University School of Medicine, for the data obtained by cardiac catheterization.

SUMARIO IN INTERLINGUA

Esseva effectuate un studio de 18 patientes con rheumatic morbo cardiac e isolate stenosis mitral de grado sever, chirurgicamente confirmate, con le objectivo de determinar le volumine de cata un del cameras cardiac opacificate per le technica de veno-angiocardio graphia biplan-stereoscopic simultanea.

Le volumine de omne camera individual es representate per le producto del area in le plano frontal con le plus grande diametro linear perpendicular a ille plano. Le capacitate cardiac total es le summa del volumines del cameras.

Le volumine de cata un del cameras variava grandemente: le atrio dextere ab 96 a 395 ml, le ventriculo dextere ab 63 a 311 ml, le atrio sinistre ab 209 a 582 ml, e le ventriculo sinistre
ab 41 a 207 ml. Le capacitate cardiac total variava ab 595 a 1341 ml.

In quasi omne casos, le volumine del atrio sinistre excedeva le volumine del altere cameras. Illo constitueva inter 28,9 e 59,9 pro cento del total capacitae cardiac.

Extensio variaciones esseva constatate in le relation inter le volumine del atrio sinistre e le volumine del ventriculo dextere o del dextere o de ambes.

Augmentos disproportional in le volumine del atrio dextere, non causate per stenosis tricuspidal, pote occurrer in casos sever de stenosis mitral.

Le volumine del ventriculo sinistre variava usque a un maximo 5 vices plus grande que le minimo. Illo non esseva invariabilemente mi-
cre. De facto, illo non esseva le plus micre del cameras in omne le casos studiate.

Esseva constatate un relation significative inter le volumine dextero-ventricular a nivellos de 165 ml o plus e le correspondente pression systolic o, similmente, le pressione pul-
mono-arterial systolic e medie.

Esseva demonstrate un correlation significative inter le capacitae cardiac total e le volumine del cameras individual, con le exception del ventriculo sinistre. Tamen, in casos in que le capacitae cardiac eseva minus que 1000 ml, illo esseva correlationate significative con solmente le volumine del atrio sinistre, e quando illo esseva plus que 1000 ml, illo es-
seva correlationate significative con solmente le volumine del ventriculo sinistre.

Finalmente, le major capacitae cardiac total eseva communemente associate con fibrilla-
lazione atrial.

REFERENCES

1. SOLOFF, L. A., AND ZATUCHNI, J.: The rel-

2. — —, MARK, G. E., JR., AND STAUFFER, H. M.: The size of the pulmonary artery in rheu-

3. — —, STAUFFER, H. M., AND KELLY, E. W.: Angiocardiographic observations of intra-

4. — —, AND MARK, G. E., JR.: Relationship of left atrial volume to pulmonary artery and wedge pressures in mitral stenosis. Cir-
culation 15: 430, 1957.

5. FRIEDMAN, C.-E.: Heart volume, myocardial volume and total capacity of the heart cavi-


9. WILSON, M. G., AND LIM, W. N.: The natural history of rheumatic heart disease in the third, fourth and fifth decades of life. 1. Prognosis with special reference to survivor-
Cardiac Chamber Volumes and their Significance in Rheumatic Heart Disease with Isolated Mitral Stenosis
LOUIS A. SOLOFF and JACOB ZATUCHNI

Circulation. 1959;19:269-274
doi: 10.1161/01.CIR.19.2.269

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1959 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/19/2/269

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/