Left Ventricular Volumes in Valvular Heart Disease

By John W. Jones, M.D., Charles E. Rackley, M.D., Robert A. Bruce, M.D., Harold T. Dodge, M.D., Leonard A. Cobb, M.D., and Harold Sandler, M.D.

Development of a satisfactory method for quantifying left ventricular chamber volumes in man has provided an opportunity to assess changes of volume that occur as a result of diseases of the aortic and mitral valves.1-3 These studies provide data concerning the relationships among chamber volume, stroke volume, and filling pressure of the left ventricle in patients with valvular heart disease.

Materials and Methods

Data were analyzed from 86 patients with aortic and mitral valve disease who were studied at the Seattle Veterans Administration Hospital or at the University of Washington Hospital. In 81 cases the valve lesions were secondary to rheumatic endocarditis; in the others they were due to healed bacterial endocarditis (two cases), aortic regurgitation secondary to cystic medionecrosis of the aorta (two cases), and congenital aortic valve disease (one patient). Sixty-four were men and 22 women; ages ranged from 25 to 65 years.

For this analysis, the valvular lesions were subdivided according to the final interpretation of all the available clinical and laboratory data into two groups: predominant aortic and predominant mitral valve disease. These major categories were subdivided arbitrarily on the basis of the volume of valvular regurgitation per heart beat, as determined by the angiographic method, where “minimal” indicated 0 to 20 ml., “moderate” 21 to 50 ml., and “marked” greater than 50 ml. of regurgitant flow per heart beat.

Left ventricular volumes were calculated from biplane angiocardiograms taken at four or six pairs of films per second following rapid injection of radiopaque contrast material into the right atrium, pulmonary artery, left atrium, or left ventricle. In some patients with aortic valve insufficiency, satisfactory opacification of the left ventricle followed injection of contrast material into the base of the aorta. Left ventricular end-diastolic and end-systolic volumes and stroke volume were calculated from films exposed at end-diastole and end-systole, as determined from a recording of the time of each film exposure with respect to the electrocardiogram. To calculate volume, the left ventricle is considered to be an ellipsoid with volume = \( \frac{4}{3} \pi \left( \frac{D_1}{2} \right) \left( \frac{D_2}{2} \right) \left( \frac{D_3}{2} \right) \), where \( D_1 \) is the longest observed diameter, and \( D_2 \) and \( D_3 \) are the lesser diameters.1-2 The margins of the ventricular chambers were traced on each film, the longest length or diameter on each film was directly measured, and the area (A) was determined by planimetry. The lesser diameters (D2 and D3) were calculated for both the anteroposterior and lateral projections by the formula for the area of an ellipse, where \( d = \frac{4A}{\pi} \). Each of these diameters was corrected for x-ray distortion by a method of triangulation. Following correction for x-ray distortion, the longest directly measured length, whether on the anteroposterior or lateral film, was used to represent the major axis (D1) of the ellipsoid for calculating volume. The calculated volume was adjusted by means of a regression equa-

![Figure 1](http://circ.ahajournals.org/)

Mean values for left ventricular volumes in patients with valvular heart disease grouped according to the volume of regurgitation. In each instance the whole bar represents end-diastolic volume in milliliters, which is divided into end-systolic and total stroke volume. Total stroke volume in turn is composed of the forward and regurgitant stroke volume.

From the Department of Medicine, University of Washington, and the Medical Service of the Veterans Administration Hospital, Seattle, Washington.

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Table 1

Physical Characteristics, Blood Flows, and Left Ventricular Volumes in Patients with Valvular Heart Disease

<table>
<thead>
<tr>
<th>Predominant valve lesion</th>
<th>Mitral</th>
<th>Aortic</th>
</tr>
</thead>
<tbody>
<tr>
<td>RF, ml./beat</td>
<td>0-20</td>
<td>21-50</td>
</tr>
<tr>
<td>No. of patients</td>
<td>12</td>
<td>15</td>
</tr>
<tr>
<td>Age, years</td>
<td>40</td>
<td>40</td>
</tr>
<tr>
<td>BSA, M²</td>
<td>±7</td>
<td>±6</td>
</tr>
<tr>
<td>Forward flow, liters/min.</td>
<td>±0.16</td>
<td>±0.18</td>
</tr>
<tr>
<td>Heart rate during</td>
<td>±1.60</td>
<td>1.75</td>
</tr>
<tr>
<td>forward flow</td>
<td>±11</td>
<td>±10</td>
</tr>
<tr>
<td>Heart rate during</td>
<td>±8</td>
<td>±6</td>
</tr>
<tr>
<td>angiogram</td>
<td>±13</td>
<td>±10</td>
</tr>
<tr>
<td>ESV, ml.</td>
<td>±28</td>
<td>±38</td>
</tr>
<tr>
<td>TSV, ml./beat</td>
<td>±31</td>
<td>±36</td>
</tr>
<tr>
<td>FSV, ml./beat</td>
<td>±18</td>
<td>±19</td>
</tr>
<tr>
<td>RSV, ml./beat</td>
<td>±16</td>
<td>±16</td>
</tr>
<tr>
<td>RF, liters/min.</td>
<td>±0.58</td>
<td>2.12</td>
</tr>
<tr>
<td>% Regurgitation</td>
<td>±0.53</td>
<td>±0.52</td>
</tr>
<tr>
<td>EDP, mm. Hg</td>
<td>±10</td>
<td>±8</td>
</tr>
<tr>
<td></td>
<td>±2</td>
<td>±2</td>
</tr>
</tbody>
</table>

* All values expressed as means ± one standard deviation.

BSA, body surface area; EDP, end-diastolic pressure; EDV, end-diastolic volume; ESV, end-systolic volume; FSV, forward stroke volume; RF, regurgitant flow; TSV, total stroke volume.

The data obtained for each group are presented in Table 1 and in Figure 1.

Eleven of the 12 patients with "minimal" mitral regurgitation were classified clinically as predominant stenosis; the twelfth patient was studied 7 months after a successful mitral annuloplasty. Four of these patients were thought to have "pure" mitral stenosis. In them the average end-diastolic volume was 108 and end-systolic volume was 48 ml.

All nine of the patients with "minimal" regurgitation at the aortic valve had predominant aortic stenosis of varying severity. The mean end-diastolic volume of 181 and end-systolic volume of 99 were considerably larger than seen in the group with "minimal" regurgitation at the mitral valve. Since regurgitation was only slight, it is presumed that the

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CREASES IN LEFT VENTRICULAR VOLUMES

Increases in left ventricular volumes were due to myocardial failure secondary to aortic stenosis or associated myocardial fibrosis. Moderate aortic stenosis did not necessarily lead to increase in left ventricular diastolic volume as demonstrated by an end-diastolic volume of 98 ml. in a patient with a calculated valve area of 0.8 cm².

Fifteen patients with mitral and eight with aortic valve disease with "moderate" regurgitant volumes were difficult to classify clinically as either predominant stenosis or insufficiency.

The two groups with regurgitant flow of greater than 50 ml. per beat had unequivocally "severe" aortic or mitral regurgitation. The average end-diastolic volumes were 250 and 293 ml. with maximum values of 440 and 443 ml., respectively. The average regurgitant stroke volumes of 92 and 95 ml. represented more than half the total left ventricular stroke.

In terms of minute volume, average regurgitant flow for these two groups of 7.4 liters per minute exceeded the forward effective flow of 4.1 and 4.6 liters. The magnitude of the left ventricular volume load produced by severe valve incompetence was illustrated by a 25-year-old man with free aortic regurgitation. The regurgitant stroke volume was 191 ml. per beat and the regurgitant flow of 19 liters per minute represented 77 per cent of the total cardiac output. Regurgitant values of a similar magnitude were also seen in several patients with mitral insufficiency.

Figure 2
Relationship of left ventricular end-systolic volume to end-diastolic volume in all patients. ▲ = Mitral 0-20 ml.; △ = Mitral 21-50 ml.; ○ = Mitral >50 ml.; □ = Aortic 0-20 ml.; ▼ = Aortic 21-50 ml.; O = Aortic >50 ml.

Figure 3
Relationship of total left ventricular stroke volume and end-diastolic volume in all patients studied. Symbols as in figure 2.

Figure 4
Relationship of total left ventricular stroke volumes to end-diastolic pressures in all patients studied. Symbols as in figure 2.

Figure 5
Relationship of left ventricular end-diastolic pressure to end-diastolic volume in all patients studied. Note the wide range of volumes for pressures of 10 mm. Hg or less. Symbols as in figure 2.
All three groups of patients with aortic valve disease had larger ventricular volumes than the corresponding groups of patients with mitral disease. Whether this represented primarily myocardial failure or the combined effects of aortic stenosis and regurgitation could not be determined from these data.

Discussion

There was marked variance in angiographic volume measurements within each group. This is not surprising, since the patients had disease of varying severity and duration. Correction of the derived volumes for differences in body surface area did not reduce the variance.

End-systolic volumes varied directly with end-diastolic volumes (R = +0.86) when data from all patients were considered (fig. 2). In a few subjects with minimal regurgitant flow, there was little or no elevation of end-diastolic volume, and the left ventricle appeared to maintain a large stroke volume by functioning with a small end-systolic volume. End-systolic volume averaged 45 per cent and stroke volume 55 per cent of the end-diastolic volume. The increased end-systolic volumes observed with increased end-diastolic volumes may be a manifestation of left ventricular failure. This increase of end-diastolic and end-systolic volume, however, might also be a consequence of the large values for left ventricular stroke volume resulting from the valvular insufficiency per se. The latter is suggested by the studies of Holt et al., 4 who found a linear relationship between end-diastolic volume and end-systolic volume in different species of animals that ranged in body size from 15.5 to 836 Kg. and had end-diastolic volumes from 44 to 1794 ml.; end-systolic volume averaged 57 per cent and stroke volume 43 per cent of the end-diastolic volume over the entire range of volumes. If these volume relationships are applicable to normal man, then the observations in our patients with valvular heart disease indicate a relatively greater systolic emptying with respect to end-diastolic volume. It is possible, however, that the differences in volume relationships noted in the present study and those described by Holt et al. 4 are due to differences in the methods for determining volumes. A comparison of left ventricular volumes in dogs determined by the indicator-dilution and angiographic technics showed good agreement for stroke volumes, but significant and consistent differences of end-diastolic volumes. 5 Since only the angiographic method gave values for volume that showed good agreement with measured volumes of chamber casts, volumes estimated by the indicator-dilution method were interpreted to be erroneously large.

Total left ventricular stroke volume was proportional to end-diastolic volume (fig. 3). Yet there was some scatter, as shown by patients with a total stroke volume that was normal or small despite a considerably elevated end-diastolic volume. These patients probably demonstrated left ventricular dilatation resultant from myocardial failure, rather than enlargement related primarily to valvular regurgitation.

Figure 4 illustrates the lack of relation between total stroke volume and end-diastolic pressure. The relationships between left ventricular stroke volume and diastolic volume and pressure indicate that in patients with valvular heart disease the level of total left ventricular stroke volume is more closely correlated with end-diastolic volume than with diastolic filling pressure.

Patients with end-diastolic pressures of 10 mm. Hg or less had end-diastolic volumes ranging from 75 to 430 ml., essentially over the entire range of diastolic volumes observed (fig. 5). These data demonstrate marked differences in pressure-volume relationships of the diastolic left ventricle of patients with chronic valvular heart disease. In general, for any given end-diastolic volume the end-diastolic pressure was higher in patients with aortic valve disease than it was in patients with mitral valve disease.

Summary

Left ventricular volumes were determined in 86 patients with aortic and mitral valve disease by use of a biplane angiographic technic.
in conjunction with cardiac catheterization. Total stroke output of the left ventricle was grossly increased by severe degrees of valvular incompetence. Although both end-systolic and total stroke volume varied directly with the end-diastolic volume, there was no correlation between end-diastolic pressure and either total stroke volume or end-diastolic volume.

References

William Withering

Withering struggled against illness during the last twenty-five years of his life. He seems to have been a man of reasonably good natural endowments as to health and constitution. He came of good stock physically, his mother having lived to the age of eighty-one. Until he was thirty-five years old Withering appears to have been remarkably well but in 1776 he had an attack of an irregular fever that probably marked the beginning of the tuberculous infection which eventually caused his death. He soon recovered from this attack but not a single winter season passed after that without some more or less serious pulmonary trouble.

In 1783 and again in the following year it was necessary for him to give up work entirely and go to the country to recruit his health. In 1790 he had a very serious attack of pleurisy. He began to suffer from shortness of breath, hemoptysis became more frequent, and the decline of strength so characteristic of chronic pulmonary tuberculosis was more apparent . . .

Withering’s health by 1796 had become so bad that he was incapable of any sustained effort whatever and he gradually sank into almost complete invalidism. He suffered from shortness of breath and attacks of hemoptysis . . .

One who had visited him a few days before his death made in all kindness and sympathy a pun that will always be classic: “The flower of English physicians is indeed Withering.”

Withering’s death occurred on the evening of October 6, 1799.

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