Systolic and Diastolic Abnormalities of the Left Ventricle in Coronary Artery Disease

Studies in Patients with Little or No Enlargement of Ventricular Volume

By J. D. Bristow, M.D., Bruce E. Van Zee, M.D., and Melvin P. Judkins, M.D.

SUMMARY

Left ventricular volume and circumference were calculated from cineangiocardiograms at 60 frames/sec in 15 patients with arteriographically proven coronary artery disease (CAD) and five control subjects. The patients with CAD had no mitral regurgitation and had an average end-diastolic volume equivalent to that reported by others in normal subjects and to that of the control group. The average end-diastolic pressure was higher in CAD and was often abnormal, despite lack of increase in end-diastolic volume. The ejection was lower in the CAD group as was the extent of circumferential fiber shortening. Maximal and mean rates of fiber shortening correlated positively with ejection fraction and were low in some individuals with CAD.

We conclude that our patients without significant increase in left ventricular end-diastolic volume had abnormalities of diastolic compliance and contractile performance.

Additional Indexing Words:
Ejection fraction       Diastolic compliance       End-diastolic pressure
Circumferential fiber shortening

SEVERAL RECENT STUDIES of coronary disease have demonstrated abnormal end-diastolic pressure in the left ventricle with stress, such as electrical pacing or exercise-induced angina pectoris.1–5 In patients with coronary disease who have no obvious cardiomegaly, it is important to learn whether the left ventricle is chronically on the verge of acute dilatation or manifests an abnormality of diastolic compliance, or both, to account for the elevated pressure found. Depressed myocardial function and alterations of the properties of the ventricular wall are both tenable. Although hemodynamic study by traditional means may allow a conclusion that the ventricle in coronary disease without distinct cardiomegaly functions normally at rest, it is possible that abnormalities of performance at rest are present which are detectable by more sensitive tests. The demonstration by cineangiocardiography of poorly contracting segments of the left ventricular wall confirmed the possibility that important muscle dysfunction without gross dilatation could occur clinically.6

The present studies, then, were designed to test two simple hypotheses. The first was that patients with coronary artery disease have abnormalities of left ventricular end-diastolic volume-pressure relationships. The second was that patients with coronary disease without definite left ventricular dilatation often have abnormalities of ventricular performance at
rest, without the stress of exercise or induced tachycardia.

**Methods**

Twenty patients are included in this report and are listed in table 1. Fifteen with coronary artery disease were chosen consecutively from a larger group undergoing coronary arteriography, because their left ventricular cineangiograms met two criteria. These were the absence of mitral regurgitation and the presence of a sufficient number of normal beats to allow determinations of left ventricular volume. Three of the patients had akinetic segments of the ventricular wall. The results were compared with those of five subjects whose left ventricular volumes and coronary arteriograms were normal and who were judged to be free of heart disease.

The procedure began with catheterization of the left ventricle by the retrograde percutaneous femoral arterial approach. Immediately after measurement of left ventricular pressure, ventriculography was performed. From 25 to 40 ml of methylglucamine diatrizoate (Renografin 76) was injected at a rate of 20 ml/sec into the left ventricle with a power injector. Filming was in the right anterior oblique projection at 60 frames/sec. Coronary arteriograms were then accomplished by technics previously described. The left ventriculograms were performed before the coronary arteriograms to avoid the hemodynamic and cardiac effects of accumulated contrast material.

The left ventricular cineangiograms were analyzed by a combination of the technic of Greene and Bunnell and the area-length method for calculation of volume of Dodge and Sandler, as previously reported. When premature ventricular contractions were induced by injection of contrast material, the second normal cycle following an extrasystole was the first one employed in the volume calculations. During normal rhythm, the ventricular cavity on each frame was traced and the area measured by planimetry. One to three complete cardiac cycles were available for each subject. Volumes were calculated and plotted by computer. From the ventriculograms, heart rate, end-systolic and end-diastolic volumes, and ejection fraction were calculated. The end-diastolic volume was compared with end-diastolic pressure obtained immediately before the filming. An equatorial

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### Table 1

**Clinical Data and Results of Coronary Arteriograms**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>History</th>
<th>Controls</th>
<th>Coronary arteries</th>
<th>Right</th>
<th>Main Left</th>
<th>Anterior Descending</th>
<th>Circumflex</th>
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<tbody>
<tr>
<td>1</td>
<td>47</td>
<td>F</td>
<td>Atypical chest pain</td>
<td>-</td>
<td>S</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
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<tr>
<td>2</td>
<td>45</td>
<td>M</td>
<td>? Angina</td>
<td>-</td>
<td>S</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
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<tr>
<td>3</td>
<td>35</td>
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<td>S</td>
<td>-</td>
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<td></td>
</tr>
<tr>
<td>4</td>
<td>48</td>
<td>F</td>
<td>Atypical chest pain</td>
<td>-</td>
<td>S</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
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<tr>
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<td>60</td>
<td>F</td>
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<td>S</td>
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<td>-</td>
<td>-</td>
<td></td>
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<tr>
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<td>-</td>
<td>S</td>
<td>-</td>
<td>O</td>
<td>S</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>48</td>
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<td>Nocturnal angina</td>
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<td>-</td>
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<td>-</td>
<td>S</td>
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<td>-</td>
<td>-</td>
<td></td>
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<tr>
<td>9</td>
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<td>M</td>
<td>Angina, previous infarction</td>
<td>O</td>
<td>S</td>
<td>-</td>
<td>-</td>
<td>-</td>
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<tr>
<td>10</td>
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<td>Severe angina, Vineburg operation</td>
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<td>Previous infarction</td>
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<td>13</td>
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<tr>
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<td>M</td>
<td>Previous infarction</td>
<td>S</td>
<td>S</td>
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<td>-</td>
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<tr>
<td>17</td>
<td>59</td>
<td>M</td>
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<td>O</td>
<td>S</td>
<td>-</td>
<td>S</td>
<td>S</td>
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<tr>
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<td>39</td>
<td>M</td>
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<td>O</td>
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</tbody>
</table>

Abbreviations: O = occlusion; S = stenosis.
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Table 2
Heart Rate, Pressure, and Volume Results

<table>
<thead>
<tr>
<th>Patient</th>
<th>Heart rate</th>
<th>End-diastolic pressure (mm Hg)</th>
<th>End-diastolic volume (ml/m²)</th>
<th>End-systolic volume (ml/m²)</th>
<th>Ejection fraction (%)</th>
<th>Atrial contribution to stroke volume (%)</th>
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<td>77</td>
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<td>66</td>
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<td>3</td>
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<td>10</td>
<td>109</td>
<td>44</td>
<td>60</td>
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<tr>
<td>4</td>
<td>90</td>
<td>10</td>
<td>54</td>
<td>14</td>
<td>75</td>
<td>23.5</td>
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<td>8</td>
<td>75</td>
<td>25</td>
<td>67</td>
<td>23.3</td>
</tr>
</tbody>
</table>

Controls

| 6       | 64         | 9                              | 64                          | 21                          | 68                    | 27.4                                     |
| 7       | 69         | 16                             | 89                          | 54                          | 40                    | 38.6                                     |
| 8       | 63         | 12                             | 91                          | 41                          | 55                    | 27.0                                     |
| 9       | 67         | 11                             | 93                          | 44                          | 53                    | 20.8                                     |
| 10      | 80         | 7                              | 84                          | 42                          | 50                    | 54.1                                     |

Coronary disease

| 11      | 78         | 13                             | 52                          | 23                          | 56                    | 27.2                                     |
| 12      | 66         | 22                             | 92                          | 53                          | 43                    | 12.5                                     |
| 13      | 81         | 20                             | 76                          | 18                          | 76                    | 11.9                                     |
| 14      | 100        | 9                              | 63                          | 36                          | 43                    | -*                                       |
| 15      | 93         | 31                             | 95                          | 50                          | 47                    | -*                                       |

Mean ± se

<table>
<thead>
<tr>
<th>Controls</th>
<th>77 ± 4.5</th>
<th>10 ± 0.7</th>
<th>77 ± 10.1</th>
<th>26 ± 5.6</th>
<th>68 ± 2.8</th>
<th>20.8 ± 1.6</th>
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<tr>
<td>Coronary disease</td>
<td>76 ± 4.0</td>
<td>15 ± 7.2</td>
<td>80 ± 4.7</td>
<td>33 ± 3.8</td>
<td>57 ± 3.1</td>
<td>26.7 ± 3.5</td>
</tr>
</tbody>
</table>

P value, comparison of means

| NS       | <0.02    | NS       | <0.02    | NS       |          |

*Could not be measured.

The increase of left ventricular volume during left atrial contraction was calculated. The effect of atrial systole was usually easily detectable from the volume curves, but when not, the volume change during the last 0.2 sec of diastole was used. Ventricular filling during atrial systole was expressed as a proportion of all filling (proportion of total left ventricular stroke volume).

Differences between the groups were compared by a t-test.

Results

The complete results are found in tables 2 and 3. The average end-diastolic volume was similar in the two groups at 77 and 80 ml/m². The end-diastolic volume (EDV) in the normal group ranged from 54 to 109 ml/m².
and from 52 to 119 ml/m² in the patients with coronary artery disease. Representative volume plots are shown in figure 1. Despite the similarity of EDV, end-diastolic pressure (EDP) was significantly different in the two groups, averaging 5 mm Hg lower in the normal subjects (P < 0.02). With coronary disease, values as high as 31 mm Hg were found with normal EDV and an example at 22 mm is shown in figure 2. No consistent relationship between the EDV and the EDP was discernible when raw volume data were used (fig. 3). However, when EDV was corrected for body size and expressed per square meter of body surface area, a significant correlation in the coronary artery disease group was found between EDV/m² and EDP (fig. 3). The correlation coefficient was 0.52 with P < 0.05. The slope of this distribution is very steep, with a rise of 1 mm Hg of EDP for each 5 ml/m² of EDV.

The change in left ventricular volume during the last 0.2 sec of diastole (during atrial systole) was divided by the increment in left ventricular diastolic pressure provided by left atrial contraction to provide a rough approximation of ventricular compliance. A significant negative correlation was found when the logarithm of compliance was plotted against left ventricular end-diastolic pressure.
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Figure 1
Plots of left ventricular volume in one control subject (A) and three patients with coronary artery disease. The scale on the ordinate is for actual volume in milliliters, not corrected for body size. The end-diastolic volume, corrected for body surface area, is indicated below each tracing (EDV/m²). (A) Normal end-diastolic volume and end-diastolic pressure. (B) Similar end-diastolic volume in a patient with coronary artery disease and definitely abnormal end-diastolic pressure. (C and D) Records from two patients with coronary artery disease with almost identical end-diastolic volumes/m² body surface area. End-diastolic pressure is very high in one and normal in the other.

Figure 2
Amplified left ventricular pressure record in patient 12, who has coronary artery disease. Large a waves with elevated ventricular end-diastolic pressure were common in the coronary disease group. Ordinate is in mm Hg.
0.52 (P < 0.05). It is clear that high end-diastolic pressure was observed without markedly elevated end-diastolic volume. (fig. 4). Those with highest EDP had the lowest compliance. The volume-pressure relationship was evaluated during this brief segment of diastole because an artifact in pressure tracings was least likely to be a factor with the low frequencies of the atrial contraction wave.

Ejection fraction (stroke volume/EDV) was significantly lower in the patients with coronary artery disease, averaging 57%, as compared to 68% in the controls (P < 0.02). Four patients were below 50%, distinctly abnormal values. Ejection fraction bore no predictable relationship to EDV or EDP in either group, as depicted in figure 5. The extent and percentage of shortening of the circumferential band of the ventricle were significantly lower in coronary artery disease. Thus, 11 of the 15 patients had values for the percentage of circumferential shortening below the lowest of the five normal subjects. The maximal and mean rates of circumferential shortening were not significantly different when the group averages were assessed, but very low values were found in individuals with coronary disease, for example in patients 7 and 14. As shown in figure 6, a significant positive correlation was present between ejection fraction and the maximal and mean rates of circumferential fiber shortening. Those with the lowest ejection fractions had the slowest shortening rates.

The contribution to left ventricular filling during atrial systole was not significantly different in the two groups (table 2). The volume plots were sufficiently clean to allow this calculation in four normal subjects and 12 with coronary artery disease; average values were 21 and 27%, respectively.

Discussion

The patients with coronary artery disease in this study were a selected group without mitral regurgitation, so chosen to avoid any influence on ventricular function of altered outflow load or abnormal filling volume. Dear
LV ABNORMALITIES IN CORONARY ARTERY DISEASE

Plot of left ventricular end-diastolic pressure and the logarithm of compliance during the last 0.2 sec of diastole. The distribution indicates that those with low compliance filled their ventricles to the point of high end-diastolic pressure. A semi-logarithmic plot was chosen because it gave the best distribution.

and associates\textsuperscript{17} previously observed that patients with this disease who do not have mitral regurgitation have relatively normal or modestly elevated left ventricular EDV, whereas those with EDV greater than 250 ml (uncorrected for body size) invariably have mitral regurgitation. Thus, mean values for EDV in our normal and coronary artery disease groups were similar. Despite this, EDP was remarkably higher in several of the patients with coronary artery disease, and the group average was higher. A similar conclusion can be reached from the results of recent studies by Falsetti and coworkers\textsuperscript{18} and Kasparian and Wiener.\textsuperscript{19} Additional corroboration comes from group averages for these two variables in patients studied by Herman and Gorlin.\textsuperscript{6} Twenty-five patients with coronary disease had an average EDV of 114 ml/m\textsuperscript{2} and an EDP of 17 mm Hg. A normal group had EDV of 100 ml/m\textsuperscript{2} and EDP of eight. The minor average difference of EDV (14\%) is associated with more than double EDP in the coronary artery disease patients. It seems certain that many patients with coronary artery disease have abnormally low ventricular diastolic compliance at rest, requiring a higher than normal driving force for filling. In our patients, there was no evident relationship between a past history of myocardial infarction and the combination of high EDP and normal EDV. The mechanism producing reduced compliance is not clear.

The relationship between EDP and diastolic
The abnormal EDP-EDV relationships at rest in our patients with coronary disease do not demonstrate a predictable relationship with ejection fraction. A high ventricular filling pressure is chosen, associated with a normal EDV, rather than a lower EDP and the resultant subnormal EDV and stroke volume. The choice presumably would be controlled by those mechanisms which influence blood volume and its distribution.

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not show whether the high EDP associated with angina or after pacing is due to left ventricular dilatation or an acute change in compliance. It is evident, however, that a small increase in EDV can be associated with considerable increases of EDP in those with coronary disease, as the distribution of volume-pressure values was very steep (fig. 3). Thus, the acute rise in EDP with angina pectoris, or after induced tachycardia, could be due to a slight increase of EDV in a ventricle which is stiff and noncompliant, without necessarily the need to invoke an acute change in compliance.

We have previously studied the influence of left atrial contraction on ventricular filling in aortic stenosis,15 with reduced left ventricular diastolic compliance due to hypertrophy.21, 22 The proportion and volume of filling occurring during atrial systole were considerably higher than in controls, showing dependence on atrial function for adequate ventricular filling at a lower mean left atrial pressure than otherwise would have been possible.23 In our patients with coronary artery disease the average contribution to ventricular filling during atrial contraction was 27%, compared to 21% in the controls and 26% in a previous control group. It appears that a potential, desirable adaptation to reduced left ventricular compliance is not commonly employed in coronary disease. It follows that hemodynamic needs for maintenance of sinus rhythm might not be as crucial in coronary disease as in aortic stenosis.

The failure to find a predictable relationship between EDP and ejection fraction was not surprising in view of the compliance abnormalities described. The variations in EDV were relatively small in our patients, and this variable also did not predict ejection fraction. Ejection fraction was abnormally low in several patients with the coronary artery disease, with values in nine of the 15 patients below the lowest one of the controls. In addition, the averages for the two groups were substantially different. The extent of muscle shortening of the idealized band at the endocardium of the ventricle also demonstrated abnormality of contraction, with values in most of the patients below the lowest found in the controls. Although the control subjects are few in number, the average shortening of the internal circumference of the ventricle (36%) is very near the results for a similar group reported by Gault and associates (38%).24 The degree of abnormality shown in coronary disease was usually not profound but was surprisingly frequent.

The ejection fraction and extent of shortening do not completely characterize the integrity of the ventricular myocardium, however.25 The rates of muscle fiber and contractile element shortening and their relationship to ventricular wall tension provide more sensitive judgments of the strength of the muscle.16, 24, 25 Our analysis provides maximal and mean circumferential fiber shortening rates, and these were markedly reduced in several individuals with coronary disease, despite absence of statistically significant differences between the means for the groups. Mean and maximal fiber shortening rates correlated well with ejection fraction for all patients studied.

Conclusions

We conclude that significant abnormalities of performance of the myocardium were often present in our patients with coronary artery disease, despite absence of significant left ventricular dilatation. We demonstrated a depression of ejection fraction, and the extent and rate of circumferential fiber shortening, variables which are reasonable indicators of the competence of the contractile elements.16, 25

Our patients also showed frequent abnormalities of left ventricular diastolic pressure-volume relationships and often had raised end-diastolic pressure. These results would conspire to produce further increases in left ventricular EDP if physiologic stress produced any increase in ventricular size.

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