Quantitative Determination of Regional Left Ventricular Wall Dynamics by Roentgen Videometry

By Jean G. Dumesnil, M.D., Erik L. Ritman, M.B., Ph.D., Robert L. Frye, M.D., Gerald T. Gau, M.D., Barry D. Rutherford, M.D., and George D. Davis, M.D.

SUMMARY

The use of roentgen videometry as a means of studying regional left ventricular wall dynamics and performance was evaluated in 32 patients undergoing coronary arteriography. Nine patients had normal coronary arteriograms and hemodynamic findings (group 1); 8 patients had generalized decrease in left ventricular contraction and abnormal hemodynamic findings (group 2), and 15 patients with coronary artery disease had regional wall dynamics abnormalities (group 3). Sixty-second determinations of wall thickness were performed in selected sites of the left ventricle. Measurements performed included end-diastolic wall thickness (EDTw), mean and peak rates of systolic wall thickening (m and p dTw/dt), and fractional systolic increase in wall thickness (ΔTw/EDTw). In patients with uniformly performing ventricles (groups 1 and 2), these parameters correlated well with other parameters of ventricular function. Best distinction between the “normal” group (group 1) and the “abnormal” group (group 2) was achieved when the rates of thickening (m and p dTw/dt) were utilized (P < 0.001). In patients of group 3, three types of abnormal regional wall dynamics could be determined and quantified objectively: hypokinesia (decreased p dTw/dt), akinesia (p dTw/dt = 0), and dyskinesia (p dTw/dt < 0). The severity of the abnormality of the wall dynamics correlated well with the presence or absence of a previous infarction on the electrocardiogram, and the anatomic location was strongly correlated with the distribution and severity of coronary artery lesions within a given ventricle.

Additional Indexing Words:

Wall thickness Regional ventricular performance Coronary artery disease Electrocardiogram

THE IMPORTANCE of abnormal regional myocardial performance as a cause of ventricular dysfunction has been emphasized by many observers.1,2 While measurements of left ventricular volumes and ejection fraction have been utilized in evaluating the performance of ventricles with normal or uniformly depressed functions, these do not provide an adequate expression of regional dysfunction nor location nor extent of such dysfunction when the ventricles have regional myocardial disease, such as occurs with coronary artery disease.3,4

Recent attempts at evaluating differences in regional myocardial performance have included outlining of ventricular contours,5 estimates of regional changes in ventricular contours,6 use of epicardial markers,7,8 radarkymography,9,10 and echocardiography.11 In particular, Eber et al.11 found that the extent of wall thickening in diseased portions of the left ventricle was markedly reduced. To date, however, a routine, objective evaluation of regional myocardial performance in both relative and absolute terms has not been feasible, most methods proposed being either qualitative only or too time consuming for frequent measurements during the cardiac cycle. The present study was designed to evaluate regional left ventricular wall dynamics by means of roentgen videometry.

Methods

Left ventricular angiograms from 32 patients undergoing coronary arteriography were analyzed. The clinical and laboratory data of these 32 patients are listed in table 1. Cardiac catheterization was performed with the patient in the fasting state after the intramuscular administration of 100 mg of sodium pentobarbital. Left heart catheterization, left ventriculography, and coronary arteriography were performed by the Sones technique.12 In three patients,
ROENTGEN VIDEOMETRY

Figure 1

A) Right anterior oblique and left anterior oblique projections of biplane left ventricular angiogram recorded as a single split-image on video tape. Bright spots are videometric recognition points of endocardial and epicardial borders. B) Postero-lateral wall as seen in left anterior oblique projection. Positioning of horizontal brightened lines determines upper and lower limits of wall segment intended for analysis. The video image contrast has been adjusted at the time of analysis to provide maximal signal-to-noise ratio of the epicardial and endocardial outlines for optimal border recognition. This adjustment is critical for reliable analysis and must be performed for each selected region.

Opaqueification of the left ventricle adequate for wall thickness studies was obtained after the injection of the contrast material into the main pulmonary artery. Left ventricular injections were performed before coronary arteriography by utilizing a No. 7 or 8 NIH catheter attached to a three-way stopcock system.

The patient was placed in the right anterior oblique position. While the patient was in deep inspiration, 50 ml of contrast material (76% Renografin) were injected continuously with the use of a power syringe during a 3- to 4-second period. A biplane video image-intensifier system (9-inch GE fluorcon, 300), with special electronic modifications for enhanced fluoroscopy, was used.

At ventriculography, the kilovoltage of the X-ray generator was held stable, and the milliamperage was increased by four to six times the normal fluoroscopic dose and held stable in order to obtain optimal signal-to-noise characteristics at angiography. The biplane video images were recorded as a single split-image (fig. 1) on 2-inch video tape at the rate of 60 video fields per second.

On completion of contrast material injection, left ventricular pressure tracings were obtained by use of the three-way stopcock system, although in patients with pulmonary artery injection, pressure tracings were available continuously throughout ventriculography. During the angiography, up to seven channels of data, including the electrocardiogram, low- and high-sensitivity left ventricular pressure, and brachial arterial pressures were recorded on the same video tape at the rate of 1,000 samples/sec by means of a specially developed video pulse-height modulator system.

The ventricular angiograms were analyzed with the use of an operator interactive border-recognition system interfaced with a computer (CDC 3500) as described previously. The bright spots in figure 1 represent the endocardial and epicar-
dial recognition points and are transmitted to the computer as digital values of their relative positions.

Calibrations were obtained from biplane video images of an object of known dimensions recorded on the same system under similar X-ray magnification conditions. Each video field was analyzed so that 60/sec determinations of left ventricular shape and wall thickness were possible. Notably, even with accurate localization of the epicardial and endocardial borders in the angiogram, the shortest distance between these borders is not necessarily the true wall thickness at that point. Thus, we have used the term "wall dynamics" rather than "dynamic wall thickness," but for convenience, wall thickness is at times used in the manuscript. The electrocardiographic and pressure values corresponding to each field were computed simultaneously. Left ventricular volumes were calculated by a computer program based on Simpson's rule. Left ventricular wall thickness was determined in sites selected by the operator and where border recognition of the epicardium was desirable and optimal. As illustrated in figure 1, the position of the horizontal bright lines determined the upper and lower limits of the wall segments being used for thickness determinations. Wall thickness was computed as the least distance between the endocardium and the epicardium and averaged out for the particular wall section being studied. Depending on the dimensions of the cardiac silhouette and on the extent over which the epicardium can satisfactorily be identified, one to three sections of the anterior wall and one to two sections of the posterolateral wall were generally studied. Plots of the 60/sec determinations of wall thickness were then displayed along with the electrocardiogram and, if so desired, the corresponding pressure values. Diastolic wall thickness, fractional increase in wall thickness, and mean and peak rates of wall thickening (dTw/dt in cm/sec) were then calculated. Multiple determinations of wall thickness in different regions of the same ventricle permitted comparison of cyclic thickness values computed during the same beat.

Validation of Methods

The accuracy and reliability of the videometric techniques have been previously established. When the computer program for thickness determination was used for an object of known dimensions, under the same X-ray magnification conditions, values obtained were within 1% of predicted values. Under constant beat-to-beat conditions (sinus rhythm and unchanged end-diastolic volume), mean and peak dTw/dt were reproducible in 11 patients within 8.5% and 10%, respectively. Furthermore, when the analysis of one ventricular segment was repeated at different times using renewed operator settings, values were reproducible to within 1%.

Results

During a three month period, from November 1972 to January 1973, the angiograms of 32 patients were studied. Two criteria were used for patient selection: (1) satisfactory identification of the epicardium and endocardium on the angiogram and (2) the possibility of including the patient into one of three groups. Group 1 patients had normal left ventricular function and pattern of contraction as determined by visual inspection of the ventriculogram; group 2 patients had generalized decrease in left ventricle performance and generalized decrease in vigor of contraction by visual inspection of the ventriculogram; and group 3 patients had more than 75% narrowing of the left anterior descending, circumflex, or marginal branches of the coronary arteries delineated by coronary arteriography and (in some patients) evidence of abnormal regional left ventricular wall motion on visual inspection of the left ventricle angiogram.

The study was designed to permit assessment of the quantitative rather than the qualitative regional ventricular function. Data from groups 1 and 2 were used to establish a relationship between wall dynamics and the functional status of uniformly performing ventricles as defined by conventional parameters. Criteria derived from this relationship were then used for the study of ventricles with regional abnormalities of contraction (group 3).

Group 1, Normal Ventricles (Table 1)

The first group consisted of nine patients who had normal findings on coronary arteriograms and normal hemodynamics (left ventricular end-diastolic pressure less than 16 mm Hg, ejection fraction above 55%, and no regional dyskinesynergy on visual inspection). A typical computer-generated plot of 60/sec left ventricular wall thickness determination is shown in figure 2. In most cases, four distinct phases were identified during one cardiac cycle: A, a transient presystolic "dip," presumably the result of atrial contraction; B, a phase of rapid thickening during ventricular contraction; C, a phase of rapid thinning corresponding to relaxation and rapid diastolic filling; and D, a phase of slow diastolic thinning corresponding to slow diastolic filling. The end-diastolic wall thickness averaged 1.16 cm (range, 0.51 to 1.82 cm), and the mean percentage increase in wall thickness was 82% (range, 46 to 156%). Peak rate of wall thickening (peak dTw/dt) averaged 7 cm/sec (range, 4.96 to 9.80 cm/sec), and mean rate of wall thickening was 3.99 cm/sec (range, 3.06 to 5.32 cm/sec). These values were obtained during regular beats in all patients. In six patients, postextrasystolic cycles also were present during ventriculography. Individual wall thickness values for postectopic cycles are listed in table 2. In all instances, the wall thickness at end-diastole was less than that observed preceding regular cycles, and the values of percentage increase in wall thickness and of mean and peak rates of wall thickening were markedly increased when compared to the values obtained during the regular cycles. Three patients (cases 1, 3, and 8) in this group had two or more different sites in the same ventricle which could be studied, as indicated in table 1. The maximal difference in peak dTw/dt between different sites was 0.31 cm/sec or 5.6% of the least...
value. Additional observations in normal patients may well indicate a wider range of values for rates of wall thickening in different areas of the ventricle, but thus far no consistent pattern of variation has been noted.

Group 2. Decrease in Left Ventricular Function (Table 1)

This group was composed of eight patients with generalized decrease in left ventricular function. Two patients had idiopathic congestive cardiomyopathy with normal coronary arteriograms. The remaining six patients had severe involvement of the three major coronary vessels and generalized reduction in left ventricular wall motion on conventional visual inspection. In this group, peak dTw/dt averaged 2.68 cm/sec (range, 1.07 to 4.39 cm/sec), and mean dTw/dt averaged 1.67 cm/sec (range, 0.80 to 2.95 cm/sec). All determinations were performed during regular cycles. Both peak and mean rates of wall thickening were significantly less than values obtained in patients of the normal group (P < 0.001, fig. 3). Although values of percentage of wall thickening in the two groups differed significantly (P < 0.02), considerable overlap of the distribution of values was present. End-diastolic wall thickness had a tendency to be less in this group, but it did not differ statistically from that of the normal group.

Group 3. Regional Wall Dynamics Abnormalities (Table 1)

The third group consisted of 15 patients with more than 75% narrowing of the left anterior descending, circumflex, or marginal branches on coronary arteriography. This group was further subdivided on the basis of the videometric data analysis into three subgroups: (a) regional decrease in the rate of wall thickening (hypokinesia); (b) no cyclic change in wall thickness (akinesia); and (c) regional systolic wall thinning (dyskinesia).* The sites where wall thickness measurements were performed are identified in table 1. Only the representative values are included in table 1, and in instances in which determinations from two regions within the same ventricle are similar and within normal range, only one region is mentioned.

Hypokinesia

A typical computer-generated plot of measurements of regional wall thickness in a ventricle (case 20) illustrating regional hypokinesis is shown in figure 4. The extent and rate of thickening of the low anterior wall were markedly decreased when compared to the high anterior wall. This patient had an 80% narrowing of his left anterior descending coronary artery immediately proximal to the origin of its diagonal branch.

A comparison of the mean and peak dTw/dt observed in the anterior and postero-lateral portions of a ventricle with regional hypokinesia is shown in figure 5. Anatomic sites of coronary artery narrowings are identified. All six patients with narrowing of 75% or greater (grade 3 or 4) of the left anterior descending coronary artery had relative hypokinesia of the

*These definitions of wall thickening abnormalities must not be confused with definitions of wall motion abnormalities as measured by movement of the endocardium alone.

---

**Figure 2**

Computer-generated plot of cyclic wall-thickness changes as measured from a left ventricular angiogram in patient with normal left ventricle. A, atrial contraction; B, ventricular contraction; C, relaxation and rapid diastolic filling; D, slow diastolic filling; E, ectopic beat.

**Figure 3**

Left) Fractional increase in wall thickness expressed as a percentage of end-diastolic wall thickness in patients with normal left ventricular function and in patients with generalized decrease in left ventricular performance. Right) Mean and peak rates of wall thickening (mean and peak dTw/dt expressed in cm/sec) in same two groups of patients. Peak dTw/dt values provided the most consistent discrimination between the two groups of patients.
Table 1

Clinical and Laboratory Data for 32 Patients Undergoing Coronary Arteriography

<table>
<thead>
<tr>
<th>Case</th>
<th>Age, sex</th>
<th>ECG</th>
<th>Coronary angiography†</th>
<th>Pressure (mm Hg)</th>
<th>EF (%)</th>
<th>EDV (ml)</th>
<th>EDTw (cm)‡</th>
<th>ΔTw/EDTw (%)§</th>
<th>dTw/dt (cm/sec)¶</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>LAD</td>
<td>LCx</td>
<td>R</td>
<td>LV syst.</td>
<td>LV ED</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>45 M</td>
<td>N</td>
<td>0 0 0</td>
<td>143</td>
<td>16</td>
<td>64</td>
<td>136</td>
<td>(a) 1.27</td>
<td>68</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(b) 1.17</td>
<td>105</td>
</tr>
<tr>
<td>2</td>
<td>35 M</td>
<td>N</td>
<td>0 0 0</td>
<td>150</td>
<td>6</td>
<td>66</td>
<td>106</td>
<td>(a) 0.91</td>
<td>84</td>
</tr>
<tr>
<td>3</td>
<td>40 F</td>
<td>N</td>
<td>0 0 0</td>
<td>123</td>
<td>12</td>
<td>58</td>
<td>153</td>
<td>(a) 0.63</td>
<td>(0.56) 155 (250)</td>
</tr>
<tr>
<td>4</td>
<td>46 F</td>
<td>N</td>
<td>0 0 0</td>
<td>160</td>
<td>2</td>
<td>66</td>
<td>113</td>
<td>(a) 1.51</td>
<td>(1.41) 46 (69)</td>
</tr>
<tr>
<td>5</td>
<td>18 M</td>
<td>N</td>
<td>0 0 0</td>
<td>121</td>
<td>15</td>
<td>75</td>
<td>200</td>
<td>(a) 1.26</td>
<td>139</td>
</tr>
<tr>
<td>6</td>
<td>31 M</td>
<td>N</td>
<td>0 0 0</td>
<td>118</td>
<td>12</td>
<td>70</td>
<td>99</td>
<td>(a) 1.82</td>
<td>(1.71) 56 (85)</td>
</tr>
<tr>
<td>7</td>
<td>50 M</td>
<td>N</td>
<td>0 0 0</td>
<td>100</td>
<td>9</td>
<td>59</td>
<td>125</td>
<td>(a) 1.47</td>
<td>52</td>
</tr>
<tr>
<td>8</td>
<td>55 F</td>
<td>N</td>
<td>0 0 0</td>
<td>149</td>
<td>13</td>
<td>66</td>
<td>78</td>
<td>(a) 0.51</td>
<td>(0.43) 156 (202)</td>
</tr>
<tr>
<td>9</td>
<td>53 M</td>
<td>N</td>
<td>0 0 0</td>
<td>138</td>
<td>11</td>
<td>68</td>
<td>129</td>
<td>(a) 1.05</td>
<td>(0.84) 93 (150)</td>
</tr>
<tr>
<td></td>
<td>Average</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>11</td>
<td>65</td>
</tr>
<tr>
<td>Group 1: Normal left ventricle (9 patients)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Congestive cardiomyopathy

<table>
<thead>
<tr>
<th>Case</th>
<th>Age, sex</th>
<th>ECG</th>
<th>Coronary angiography†</th>
<th>Pressure (mm Hg)</th>
<th>EF (%)</th>
<th>EDV (ml)</th>
<th>EDTw (cm)‡</th>
<th>ΔTw/EDTw (%)§</th>
<th>dTw/dt (cm/sec)¶</th>
</tr>
</thead>
<tbody>
<tr>
<td>10</td>
<td>63 M</td>
<td>LBB</td>
<td>0 0 0</td>
<td>146</td>
<td>27</td>
<td>27</td>
<td>411</td>
<td>1.25</td>
<td>52</td>
</tr>
<tr>
<td>11</td>
<td>44 M</td>
<td>N</td>
<td>0 0 0</td>
<td>95</td>
<td>39</td>
<td>4</td>
<td>380</td>
<td>1.32</td>
<td>27</td>
</tr>
</tbody>
</table>

Severe coronary artery disease

<table>
<thead>
<tr>
<th>Case</th>
<th>Age, sex</th>
<th>ECG</th>
<th>Coronary angiography†</th>
<th>Pressure (mm Hg)</th>
<th>EF (%)</th>
<th>EDV (ml)</th>
<th>EDTw (cm)‡</th>
<th>ΔTw/EDTw (%)§</th>
<th>dTw/dt (cm/sec)¶</th>
</tr>
</thead>
<tbody>
<tr>
<td>12</td>
<td>47 M</td>
<td>Inf. post. infarct</td>
<td>3 4 4</td>
<td>138</td>
<td>15</td>
<td>33</td>
<td>145</td>
<td>0.88</td>
<td>91</td>
</tr>
<tr>
<td>13</td>
<td>68 M</td>
<td>LBB</td>
<td>2 4 2</td>
<td>141</td>
<td>19</td>
<td>5</td>
<td>412</td>
<td>0.88</td>
<td>32</td>
</tr>
<tr>
<td>14</td>
<td>60 M</td>
<td>LBB</td>
<td>3 2 4</td>
<td>107</td>
<td>18</td>
<td>38</td>
<td>284</td>
<td>0.73</td>
<td>77</td>
</tr>
<tr>
<td>15</td>
<td>42 M</td>
<td>Ant. infarct</td>
<td>4 2 4</td>
<td>122</td>
<td>40</td>
<td>7</td>
<td>263</td>
<td>0.80</td>
<td>36</td>
</tr>
<tr>
<td>16</td>
<td>54 M</td>
<td>ST-T abnorm.</td>
<td>4 3 4</td>
<td>124</td>
<td>14</td>
<td>32</td>
<td>183</td>
<td>0.95</td>
<td>53</td>
</tr>
<tr>
<td>17</td>
<td>57 M</td>
<td>Ant. infarct</td>
<td>4 3 4</td>
<td>124</td>
<td>18</td>
<td>33</td>
<td>175</td>
<td>0.84</td>
<td>41</td>
</tr>
<tr>
<td></td>
<td>Average</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>21</td>
<td>20</td>
</tr>
</tbody>
</table>

Group 2: Generalized decrease in LV function (8 patients)

Hypokinesia

<table>
<thead>
<tr>
<th>Case</th>
<th>Age, sex</th>
<th>ECG</th>
<th>Coronary angiography†</th>
<th>Pressure (mm Hg)</th>
<th>EF (%)</th>
<th>EDV (ml)</th>
<th>EDTw (cm)‡</th>
<th>ΔTw/EDTw (%)§</th>
<th>dTw/dt (cm/sec)¶</th>
</tr>
</thead>
<tbody>
<tr>
<td>18</td>
<td>61 M</td>
<td>N</td>
<td>0 3 0</td>
<td>135</td>
<td>11</td>
<td>63</td>
<td>172</td>
<td>(c) 0.84</td>
<td>150</td>
</tr>
<tr>
<td>19</td>
<td>52 M</td>
<td>ST-T abnorm.</td>
<td>4 0 1</td>
<td>148</td>
<td>32</td>
<td>58</td>
<td>272</td>
<td>(d) 1.33</td>
<td>50</td>
</tr>
<tr>
<td>20</td>
<td>62 M</td>
<td>LBB</td>
<td>3 0 4</td>
<td>153</td>
<td>7</td>
<td>36</td>
<td>437</td>
<td>(a) 0.51</td>
<td>215</td>
</tr>
</tbody>
</table>

Group 3: Regional wall dynamics abnormalities (15 patients)
<table>
<thead>
<tr>
<th>No</th>
<th>Age</th>
<th>Gender</th>
<th>Location</th>
<th>QRS</th>
<th>R</th>
<th>S</th>
<th>QT</th>
<th>QTc</th>
<th>RP</th>
<th>RR</th>
<th>Average</th>
<th>Akinesia</th>
<th>Dyskinesia</th>
</tr>
</thead>
<tbody>
<tr>
<td>21</td>
<td>43 M</td>
<td>Ant. infarct</td>
<td>4</td>
<td>2</td>
<td>1</td>
<td>170</td>
<td>22</td>
<td>47</td>
<td>249</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>22</td>
<td>39 M</td>
<td>N</td>
<td>0</td>
<td>4</td>
<td>0</td>
<td>106</td>
<td>16</td>
<td>32</td>
<td>237</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>23</td>
<td>62 M</td>
<td>Ant. infarct</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>118</td>
<td>20</td>
<td>50</td>
<td>228</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>24</td>
<td>61 M</td>
<td>N</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>115</td>
<td>6</td>
<td>40</td>
<td>147</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>25</td>
<td>43 M</td>
<td>Ant. infarct</td>
<td>3</td>
<td>3</td>
<td>4</td>
<td>165</td>
<td>23</td>
<td>18</td>
<td>417</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>26</td>
<td>53 M</td>
<td>N</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>193</td>
<td>19</td>
<td>65</td>
<td>135</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>27</td>
<td>63 F</td>
<td>ST-T abnorm.</td>
<td>3</td>
<td>4</td>
<td>4</td>
<td>238</td>
<td>19</td>
<td>70</td>
<td>110</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>28</td>
<td>43 M</td>
<td>N</td>
<td>0</td>
<td>4</td>
<td>0</td>
<td>131</td>
<td>19</td>
<td>68</td>
<td>145</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Average**

- Less hypokinetic areas
- Most hypokinetic areas

- **Akinesia**
  - Ant. infarct: 4, 0, 1, 96, 25, 32, 214
  - N: 0, 0, 0

- **Dyskinesia**
  - RBB, ant. infarct: 4, 0, 3, 143, 16, 38, 162
  - Ant. infarct: 4, 0, 4, 147, 18, 34, 143
  - Inf. post. infarct: 2, 4, 4, 106, 12, 11, 229
  - N: 0, 0, 0

**Abbreviations:** LAD, left anterior descending; LCx, left circumflex; R, right coronary artery; LV syst., left ventricular systolic pressure; LVED, left ventricular end-diastolic pressure; EF, ejection fraction; EDV, end-diastolic volume; EDTw, end-diastolic wall thickness; ATw/EDTw, percent wall thickening; and dTw/dt, rate of wall thickening.

†Coronary angiographic findings: 0, normal; 1, irregularities; 2, <75% stenosis; 3, ≥75% stenosis; and 4, occlusion.

†Values in parentheses are for postectopic beats.

§(a), High anterior wall; (b), mid-anterior wall; (c), lower anterior wall; and (d), mid-postero-lateral wall.
The patient's left ventricular wall dynamics were studied using simultaneous regional wall-thickness changes in a 62-year-old man with 85% narrowing in the middle third of the left anterior descending coronary artery. Extent and rate of thickening were markedly decreased in the normally perfused area normally perfused by this vessel.

Furthermore, hypokinesia was most severe toward the apex in patients in whom two determinations in the anterior wall were feasible. In three patients with narrowing of the left circumflex coronary artery of 75% or greater, values of mean and peak dTw/dt recorded for the posterolateral wall were significantly lower than values recorded in other regions of the ventricle. The two remaining patients had significant narrowings of the left anterior descending and left circumflex coronary arteries and exhibited hypokinesia of both the anterior and posterolateral ventricular walls. Electrocardiographic evidence of a previous myocardial infarction tended to be associated with lower values of peak and mean dTw/dt in corresponding hypokinetic regions, when compared with values obtained in hypokinetic regions of patients without evidence of prior infarction. Study of the posteroseptal wall thickness was not performed in the present study because the angiographic projection used precluded adequate delineation of the epicardial surface.

**Akinesia**

The one patient in this group had a complete occlusion of his left anterior descending coronary artery and electrocardiographic signs of a previous

### Table 2

Comparison of Left Ventricular Wall Dynamics in Six Patients With Regular and Postectopic Beats During Left Ventricular Angiography

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Regular</th>
<th>Postectopic</th>
</tr>
</thead>
<tbody>
<tr>
<td>EDTw (cm)</td>
<td>1.13</td>
<td>1.03</td>
</tr>
<tr>
<td>ΔTw/EDTw (%)</td>
<td>95</td>
<td>141</td>
</tr>
<tr>
<td>Mean dTw/dt (cm/sec)</td>
<td>3.96</td>
<td>5.13</td>
</tr>
<tr>
<td>Peak dTw/dt (cm/sec)</td>
<td>6.98</td>
<td>10.0</td>
</tr>
</tbody>
</table>

* *Averages: end-diastolic wall thickness (EDTw), fractional increase in wall thickness (ΔTw/EDTw), and mean and peak rates of wall thickening (mean and peak dTw/dt).*
ROENTGEN VIDEOMETRY

Figure 6

Simultaneous wall-thickness changes in a 47-year-old man with total occlusion of left circumflex coronary artery and ECG evidence of previous infero-posterior myocardial infarction. Postero-lateral wall shows paradoxical systolic thinning.

anterior myocardial infarction. Values for wall thickness in the apical regions of his left ventricle were constant and showed no systolic or diastolic variation.

Dyskinesia

Three patients had regional left ventricular wall thinning during systole (fig. 6). Systolic wall thinning is expressed as a negative value in table 1. Both patients with left ventricular apical wall thinning had complete occlusion of the left anterior descending coronary artery and electrocardiographic evidence of an old anterior scar. The patient with systolic thinning of the postero-lateral wall had a complete occlusion of his left circumflex and right coronary arteries and electrocardiographic evidence of an infero-posterior myocardial infarction.

Discussion

Mitchell et al.\textsuperscript{19} suggested that changes in wall thickness were strongly influenced by the Frank-Starling mechanism and changes in the contractile state. With end-diastolic ventricular volume and wall thickness held constant, the systolic increase in wall thickness was 42% during a control period and 62% after infusion of norepinephrine. The same authors also found that the extent of wall thickening when using direct measurement (25 to 45%) was considerably less than that found by angiographic techniques (80 to 120%). This latter result agreed with the percentage increase in wall thickness found in human subjects by other investigators,\textsuperscript{20} and the discrepancy between the two methods was attributed to the behavior of the trabeculae carnæ during systole. Gault et al.\textsuperscript{21} related changes in wall thickness to left ventricular disease and, using a direct measurement technique, found a percentage increase in wall thickness of 32% in patients without and 23% in patients with left ventricular disease. Eber et al.,\textsuperscript{11} in a study of two patients with coronary artery disease, observed that regions of normal and diseased myocardium in the same ventricle could be differentiated by analysis of changes in the left ventricular wall thickness.

Our study utilized rates of wall thickening rather than the percentage increase in wall thickness for the purpose of evaluating regional left ventricular wall dynamics. As evidenced in figure 3, a more distinct differentiation between patients without and with left ventricular disease was achieved with the use of this criterion. The parameters that we used also correlated well with the anatomic location of significant coronary artery lesions and with the ventricular regions having abnormal dynamics. In all cases, lesions of the left anterior descending artery corresponded to anterior wall dynamics abnormalities, and lesions of the left circumflex corresponded to postero-lateral wall dynamics abnormalities. However, it is important to emphasize that the patients in whom abnormalities of left ventricular contraction were quantitated represent a selected group. No implication can be made from the current data that all patients with a given degree of narrowing in a coronary artery will have detectable changes in rate of wall thickening in the area supplied by the diseased coronary artery. Studies are planned to evaluate the presence or absence of such abnormalities in regional left ventricular contraction in relation to severity of coronary artery narrowing in an unselected consecutive series of patients. This study also provided an objective means of differentiation among three types of regional wall dynamics abnormalities: hypokinesia, akinesia, and dyskinesia. These data also correlated well with electrocardiographic findings, akinesia and dyskinesia being found only in patients with prior myocardial infarction. In patients with regional hypokinesia, reduction in regional wall dynamics had a tendency to be most severe in those who had electrocardiographic evidence of a previous myocardial infarction.

Values of end-diastolic ventricular wall thickness and percentage increase in wall thickness for our group of normal patients were similar to those observed by previous investigators.\textsuperscript{11, 19, 20, 22} In certain patients (cases 20, 21, 23, and 26, table 1) with regional wall dynamics abnormalities, peak $dTw/dt$ in the regions supplied by a coronary artery with more than 75% stenosis was higher than in the normal group and suggests a compensatory increased function of these regions.
The increased rate of wall thickening seen during postectopic beats is consistent with the Frank-Starling mechanism and extrasystolic augmentation of contractility. It also illustrates the advantages of a capability to make analyses through several selected cardiac cycles. The possibility that an extrasystole during the left ventricular injection may provide an important intervention for evaluating left ventricular function is under investigation, as is the evaluation of other positive or negative inotropic influences on rates of wall thickening.

A method is described for the quantitation and objective evaluation of regional wall dynamics. This method of study has several potential applications, particularly in the study of patients with coronary artery disease. The relationship between abnormal patterns of contraction (as depicted by the ventriculogram) and the wall dynamics is not always clear or amenable to quantitation on visual inspection because of the rapidity of the events, the relatively small dimensional changes involved, and the concurrent movement of the section of wall under study. At present, this measurement can be interpreted as an empirical index of myocardial function as far as quantitative determination is concerned, but the measurement is considered to be a direct indication of the regional localization of myocardial dysfunction.

Acknowledgment

We gratefully acknowledge the support and assistance of Mr. Ralph E. Sturm and Dr. Earl H. Wood in the development of the roentgen videometry system used in this study. Skilled computer programming and patient data analysis were performed by Mr. Donald L. Cravath, and assistance in the conduct of these studies was given by Messrs. Gerald M. Alborn, Richard Christopherson, James C. Fellows, and Ralph G. Goodrich, in addition to the members of the technical staff of the Cardiac Laboratory.

References

13. Ritman EL, Sturm RE, Wood EH: Biplane roentgen videometry system for dynamic (60/sec) studies of the shape and size of circular structures, particularly the left ventricle. Am J Cardiol 32: 180, 1973
Quantitative Determination of Regional Left Ventricular Wall Dynamics by Roentgen Videometry
JEAN G. DUMESNIL, ERIK L. RITMAN, ROBERT L. FRYE, GERALD T. GAU, BARRY D. RUTHERFORD and GEORGE D. DAVIS

Circulation. 1974;50:700-708
doi: 10.1161/01.CIR.50.4.700
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1974 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/50/4/700