The Effect of Acute Digitalization on the Hemodynamic Response to Exercise in Coronary Artery Disease

By John O. Parker, M.D., Roxroy O. West, M.D., J. Rodney Ledwich, M.B., and Salvatore Di Giori, M.D.

SUMMARY

Hemodynamic observations are made at rest and during exercise before and following the administration of 0.5 mg of ouabain in patients with coronary artery disease and in normal subjects. Patients with coronary artery disease and exertional angina showed evidence of reversible cardiac failure during exercise. The patients with coronary artery disease who did not have angina and the normal subjects had normal hemodynamic responses to exercise. Acute digitalization did not prevent angina in any of the symptomatic patients, although there was hemodynamic evidence of improved ventricular performance. In the patients with coronary disease without angina minor hemodynamic changes at rest and during exercise were suggestive of improved left ventricular function.

Additional Indexing Words:
Digitalis glycosides Ventricular failure Myocardial ischemia
Left ventricular end-diastolic pressure Ventricular function

HEMODYNAMIC STUDIES during acute coronary insufficiency occurring either spontaneously1, 2 or precipitated by exercise,1, 3-9 isoproterenol,8 or atrial pacing10-13 have shown depression of left ventricular function. The elevated left ventricular end-diastolic pressure found during exertional angina3, 6-9 has been considered to be due to altered left ventricular compliance,9 but a more likely explanation is reversible left ventricular failure.12

The occurrence of hemodynamic changes suggestive of left ventricular failure during exercise in patients with coronary artery disease has led to limited investigation of the effect of digitalis on the hemodynamic response to exercise and on exercise tolerance. Malmborg4 found that exertional angina was prevented in nine of 17 patients by acute digitilization with lanatoside C. Smith and co-workers14 found no increase in exercise tolerance as measured on a treadmill after the intravenous administration of lanatoside C. The present study reports the hemodynamic findings at rest and during exercise in patients with coronary artery disease with and without exertional angina before and after the intravenous administration of ouabain.

Methods

Hemodynamic investigations with subsequent selective cine-coronary arteriography and left ventriculography were performed on 30 patients with known or suspected coronary artery disease who were free of cardiomegaly, arrhythmias, and clinical evidence of cardiac failure. None of the patients were receiving digitalis or diuretics at the time of study. Fourteen patients had systolic pressures greater than 140 mm Hg, but only three exceeded 160 mm Hg, and only two had diastolic pressures above 90 mm Hg. None had been treated for arterial hypertension or exhibited hypertensive retinopathy greater than...
grade I. The patients were brought to the laboratory the day prior to the study so that they would be familiar with the surroundings and the nature of the procedure. Resting ventilation was recorded, and an 8-min period of supine leg exercise on a bicycle ergometer was carried out. This allowed us to observe the patient’s response and to estimate the degree of stress that would be required to produce angina.

The patients were studied the following day while in the fasting state without premedication. Under local anesthesia the brachial artery and an accompanying vein were isolated. A double lumen, no. 9 Courmand catheter was passed into the right heart and advanced so that the tip lay in the pulmonary artery and the proximal lumen in the right ventricle. A no. 8 Sones catheter was introduced into the left ventricle from the right brachial artery, and a Courmand needle was inserted into the left brachial artery. Lead II of the electrocardiogram and pressures from the pulmonary artery, right ventricle, brachial artery, and left ventricle were recorded at 5-min intervals during a 20-min control period, and the cardiac output was measured in triplicate during the final 3 min by the dye-dilution technic using indocyanine green. The patients then exercised for 8 min at the predetermined work load. The electrocardiogram and pressures were recorded after 1, 3, 5, and 8 min of exercise and the cardiac output determination repeated during the final 3 min. Oxygen consumption was measured with each cardiac output determination. Hemodynamic observations were repeated after a 30-min rest period, and then the patients were given 0.5 mg of ouabain* diluted in 5 ml of normal saline through the pulmonary artery catheter over a 30-sec period. Thirty minutes after the drug administration, hemodynamic measurements were obtained again at rest, and the patient exercised for a second period of 8 min at the same work load as that during the initial exercise period with hemodynamic observations being recorded as during the first study period.

Pressures were measured with a P23Db Statham strain-gauge from a zero reference level 5 cm below the angle of Louis and recorded on a photographic recorder.† Pressures were measured over at least two respiratory cycles and the mean pressures in the brachial and pulmonary arteries were obtained electronically. The left ventricular stroke-work index (LVSWI) in g-m/m² was calculated using the formula:

\[
LVSWI = \frac{SI \times (BA_m - LVEDP) \times 13.6}{1,000}
\]

where \( SI \) = stroke index in ml/m², \( BA_m \) = mean brachial arterial pressure in mm Hg, and \( LVEDP = \) left ventricular end-diastolic pressure in mm Hg. The systolic ejection rate index was calculated of the stroke index, by dividing and the systolic ejection period obtained from high speed brachial artery pressure tracings. A modified tension-time index was calculated as the product of peak left ventricular systolic pressure and heart rate. This modification is based on the fact that peak systolic pressure is a more important determinant of myocardial oxygen consumption than is the total area under the left ventricular systolic pressure curve.\(^{15}\) Expired air was collected in a Tissot spirometer at the time of cardiac output determinations and analyzed for carbon dioxide and oxygen by the micro-Scholander technic.

Following the completion of these hemodynamic studies, selective cine-coronary arteriography and left ventriculography were carried out without complications in all patients. Eight patients who were found to have normal coronary arteriograms and left ventriculograms and no hemodynamic or electrocardiographic evidence of heart disease were placed in the normal group. The remaining 22 patients had major obstructive lesions in one or more coronary vessels. The left ventriculograms showed minor impairment of contractility in some patients, but none showed enlargement of the left ventricle or evidence of a ventricular aneurysm.

**Results**

Twelve of the 22 patients with coronary artery disease developed angina pectoris during the period of exercise. This developed within the first 2 or 3 min of exercise and persisted, usually with increasing severity, until exercise was terminated. In no case was it necessary to discontinue exercise prematurely. For purposes of analysis, three groups will be considered: (a) angina group—12 patients with coronary artery disease who experienced angina with exercise; (b) non-angina group—10 patients with coronary artery disease who did not experience angina with exercise; (c) normal group—eight patients free of cardiac disease. During exercise after ouabain all patients in the angina group again experienced chest pain while the non-angina group and the normal subjects remained free of symptoms. The hemodynamic data for these three

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*Supplied as Ouabine Arnaud (Laboratoire Nationale) Lyster Chemicals Ltd., Toronto, Ontario.
†DR8 Recorder, Electronics for Medicine, Inc., White Plains, New York.
groups of patients at rest (Rest I), during the final 3 min of the first exercise period (Exercise I), after 30 min of recovery (Recovery), 30 min after the administration of ouabain (Rest II), and during the final 3 min of the second exercise period (Exercise II) are summarized in Table 1.

**Left Ventricular End-Diastolic Pressure (Fig. 1)**

The average left ventricular end-diastolic pressure in the three groups was normal at rest, although two patients of the angina group and one of the non-angina group had left ventricular end-diastolic pressure above the upper limit of normal (12 mm Hg). During exertional angina the left ventricular end-diastolic pressure rose from 11.1 mm Hg to 34.6 mm Hg ($P < 0.001$). In the non-angina group and the normal group the increase in left ventricular end-diastolic pressure during exercise was not significant, although three patients in the non-angina group showed abnormal exercise values. At rest after the administration of ouabain, the left ventricular end-diastolic pressure in the angina group decreased from 10.3 to 7.8 mm Hg ($P < 0.001$), but there was essentially no change in the other two groups. In the angina group during the second exercise period, the average left ventricular end-diastolic pressure rose to 30.1 mm Hg ($P < 0.001$). This level, although still grossly elevated, was significantly lower than that in these patients during the first exercise period ($P < 0.001$). In the non-angina group and in the normal subjects, the left ventricular end-diastolic pressure was similar during the two exercise periods.

**Mean Pulmonary Arterial Pressure (Fig. 2)**

The average mean pulmonary arterial pressure was normal in the three groups at rest and rose significantly during exercise in each group ($P < 0.001$), the increase being most marked in the angina group where the value during exercise was 41.6 mm Hg. At rest following the administration of ouabain there was a slight decline in the mean pulmonary arterial pressure in all three groups. During the second exercise period the average pressure again rose significantly in each group ($P < 0.001$), and the greatest increase occurred in the angina group where the average pressure was 35.0 mm Hg. The levels observed during the second exercise period were significantly lower than during the initial exercise period in both the angina and non-angina groups ($P < 0.001$) but not in the normal subjects.

**Right Ventricular End-Diastolic Pressure (Fig. 3)**

The average right ventricular end-diastolic pressure was normal at rest in all groups and became abnormal during exercise only in the angina group where it rose from 3.7 to 10.3 mm Hg ($P < 0.001$). At rest following the
Table 1

Summary of Hemodynamic Values at Rest and Exercise Before and After the Administration of Ouabain

<table>
<thead>
<tr>
<th></th>
<th>BA&lt;sub&gt;m&lt;/sub&gt; (mm Hg)</th>
<th>LVEDP (mm Hg)</th>
<th>PA&lt;sub&gt;m&lt;/sub&gt; (mm Hg)</th>
<th>RVEDP (mm Hg)</th>
<th>CI (L/min/m&lt;sup&gt;2&lt;/sup&gt;)</th>
<th>HR (beats/min)</th>
<th>SI (ml)</th>
<th>VO&lt;sub&gt;2&lt;/sub&gt; (ml/min/m&lt;sup&gt;2&lt;/sup&gt;)</th>
<th>SERI (ml/sec/m&lt;sup&gt;2&lt;/sup&gt;)</th>
<th>LVSWI (g-m/m&lt;sup&gt;2&lt;/sup&gt;)</th>
<th>TTI (units)</th>
<th>dp/dt (mm Hg/sec)</th>
</tr>
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<tbody>
<tr>
<td><strong>Angina Group</strong></td>
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</tr>
<tr>
<td>Rest I</td>
<td>106 ± 10</td>
<td>11.1 ± 4.6</td>
<td>14.3 ± 2.8</td>
<td>3.7 ± 1.8</td>
<td>3.06 ± 0.32</td>
<td>77 ± 10</td>
<td>40 ± 6.0</td>
<td>136 ± 11</td>
<td>118 ± 15.5</td>
<td>51.2 ± 7.8</td>
<td>1159 ± 140</td>
<td>1640 ± 458</td>
</tr>
<tr>
<td>Exercise I</td>
<td>129 ± 14</td>
<td>34.6 ± 10.1</td>
<td>41.6 ± 7.9</td>
<td>10.3 ± 3.8</td>
<td>4.56 ± 0.88</td>
<td>125 ± 19</td>
<td>37 ± 6.8</td>
<td>525 ± 95</td>
<td>122 ± 21.1</td>
<td>47.1 ± 14.0</td>
<td>2230 ± 413</td>
<td>2565 ± 721</td>
</tr>
<tr>
<td>Recovery</td>
<td>104 ± 12</td>
<td>10.3 ± 6.5</td>
<td>13.0 ± 3.5</td>
<td>3.3 ± 1.4</td>
<td>2.90 ± 0.28</td>
<td>79 ± 12</td>
<td>37 ± 4.9</td>
<td>140 ± 8</td>
<td>113 ± 16.2</td>
<td>47.2 ± 8.7</td>
<td>1165 ± 146</td>
<td>1601 ± 447</td>
</tr>
<tr>
<td>Rest II</td>
<td>104 ± 11</td>
<td>7.8 ± 4.4</td>
<td>11.8 ± 2.9</td>
<td>3.2 ± 1.6</td>
<td>2.96 ± 0.45</td>
<td>77 ± 11</td>
<td>39 ± 6.2</td>
<td>140 ± 13</td>
<td>122 ± 14.9</td>
<td>51.0 ± 9.3</td>
<td>1139 ± 171</td>
<td>1798 ± 468</td>
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<tr>
<td>Exercise II</td>
<td>133 ± 16</td>
<td>30.1 ± 9.8</td>
<td>35.0 ± 8.4</td>
<td>8.0 ± 4.2</td>
<td>4.84 ± 0.93</td>
<td>123 ± 21</td>
<td>40 ± 6.7</td>
<td>518 ± 96</td>
<td>129 ± 18.3</td>
<td>55.6 ± 12.0</td>
<td>2313 ± 395</td>
<td>2527 ± 588</td>
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<tr>
<td><strong>Non-angina Group</strong></td>
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<tr>
<td>Rest I</td>
<td>95 ± 12</td>
<td>6.8 ± 3.8</td>
<td>13.0 ± 4.4</td>
<td>3.4 ± 2.3</td>
<td>3.11 ± 0.60</td>
<td>81 ± 9</td>
<td>39 ± 5.9</td>
<td>140 ± 14</td>
<td>116 ± 19.9</td>
<td>46.2 ± 7.0</td>
<td>1102 ± 209</td>
<td>1443 ± 490</td>
</tr>
<tr>
<td>Exercise I</td>
<td>116 ± 11</td>
<td>10.5 ± 7.9</td>
<td>24.1 ± 5.9</td>
<td>5.7 ± 3.5</td>
<td>5.81 ± 1.22</td>
<td>132 ± 21</td>
<td>44 ± 7.1</td>
<td>577 ± 62</td>
<td>145 ± 24.4</td>
<td>62.5 ± 8.8</td>
<td>2276 ± 499</td>
<td>3089 ± 964</td>
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<tr>
<td>Recovery</td>
<td>91 ± 16</td>
<td>5.6 ± 4.9</td>
<td>7.9 ± 3.8</td>
<td>2.8 ± 1.9</td>
<td>3.07 ± 0.51</td>
<td>83 ± 9</td>
<td>37 ± 6.1</td>
<td>144 ± 18</td>
<td>115 ± 15.5</td>
<td>43.1 ± 9.1</td>
<td>1065 ± 195</td>
<td>1396 ± 338</td>
</tr>
<tr>
<td>Rest II</td>
<td>92 ± 15</td>
<td>5.7 ± 4.0</td>
<td>7.2 ± 3.6</td>
<td>2.4 ± 2.0</td>
<td>3.19 ± 0.58</td>
<td>81 ± 13</td>
<td>40 ± 5.0</td>
<td>144 ± 22</td>
<td>125 ± 11.0</td>
<td>47.1 ± 9.8</td>
<td>1059 ± 214</td>
<td>1604 ± 442</td>
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<tr>
<td>Exercise II</td>
<td>112 ± 9</td>
<td>8.6 ± 6.3</td>
<td>14.7 ± 4.5</td>
<td>4.3 ± 2.6</td>
<td>6.04 ± 0.89</td>
<td>129 ± 21</td>
<td>48 ± 8.5</td>
<td>508 ± 71</td>
<td>147 ± 18.1</td>
<td>66.6 ± 12.6</td>
<td>2149 ± 421</td>
<td>3076 ± 894</td>
</tr>
<tr>
<td><strong>Normal Group</strong></td>
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</tr>
<tr>
<td>Rest I</td>
<td>88 ± 8</td>
<td>8.5 ± 2.3</td>
<td>13.0 ± 3.1</td>
<td>3.4 ± 1.2</td>
<td>3.64 ± 0.79</td>
<td>80 ± 13</td>
<td>46 ± 9.0</td>
<td>145 ± 8</td>
<td>137 ± 28.9</td>
<td>48.9 ± 9.9</td>
<td>1001 ± 183</td>
<td>1409 ± 285</td>
</tr>
<tr>
<td>Exercise I</td>
<td>98 ± 10</td>
<td>7.8 ± 3.7</td>
<td>20.5 ± 4.8</td>
<td>4.6 ± 2.0</td>
<td>6.36 ± 1.48</td>
<td>128 ± 13</td>
<td>51 ± 14.0</td>
<td>551 ± 61</td>
<td>168 ± 40.1</td>
<td>61.5 ± 12.8</td>
<td>1896 ± 346</td>
<td>2417 ± 540</td>
</tr>
<tr>
<td>Recovery</td>
<td>87 ± 9</td>
<td>7.8 ± 2.6</td>
<td>13.8 ± 3.7</td>
<td>3.6 ± 1.7</td>
<td>3.82 ± 0.75</td>
<td>85 ± 14</td>
<td>45 ± 7.7</td>
<td>150 ± 15</td>
<td>134 ± 19.6</td>
<td>48.3 ± 8.5</td>
<td>1040 ± 147</td>
<td>1389 ± 229</td>
</tr>
<tr>
<td>Rest II</td>
<td>90 ± 7</td>
<td>7.3 ± 2.4</td>
<td>13.4 ± 3.2</td>
<td>3.5 ± 1.5</td>
<td>3.95 ± 0.80</td>
<td>79 ± 12</td>
<td>51 ± 9.6</td>
<td>152 ± 18</td>
<td>151 ± 25.4</td>
<td>57.0 ± 10.0</td>
<td>1030 ± 143</td>
<td>1495 ± 200</td>
</tr>
<tr>
<td>Exercise II</td>
<td>97 ± 9</td>
<td>8.0 ± 3.7</td>
<td>20.5 ± 5.6</td>
<td>4.1 ± 2.0</td>
<td>6.81 ± 1.26</td>
<td>124 ± 14</td>
<td>55 ± 12.0</td>
<td>535 ± 103</td>
<td>184 ± 28.7</td>
<td>66.0 ± 10.6</td>
<td>1844 ± 394</td>
<td>2426 ± 378</td>
</tr>
</tbody>
</table>

Abbreviations: BA<sub>m</sub> = mean brachial arterial pressure; LVEDP = left ventricular end-diastolic pressure; PA<sub>m</sub> = mean pulmonary arterial pressure; RVEDP = right ventricular end-diastolic pressure; CI = cardiac index; HR = heart rate; SI = stroke index; VO<sub>2</sub> = oxygen consumption per min; SERI = systolic ejection rate index; LVSWI = left ventricular stroke work index; TTI = modified tension-time index (Ba<sub>s</sub> X HR X 10<sup>-1</sup>); dp/dt = first derivative of left ventricular systolic pressure curve.
administration of ouabain, there was essentially no change in right ventricular filling pressure. During the second exercise period the right ventricular end-diastolic pressure remained normal in the non-angina and normal groups, but it rose from 3.2 to 8.0 mm Hg in the angina group (P < 0.001), a level significantly lower than that during the first exercise period (P = 0.02).

Mean Brachial Arterial Pressure (Fig. 4)

There was essentially no change in the mean brachial arterial pressure in the three groups at rest following the administration of ouabain. The response to exercise was similar before and after the administration of this drug in each of the three groups.

Cardiac Index (Fig. 5)

The cardiac index was similar in each of these three groups at rest and rose by 32.9%, 86.8%, and 74.8% in the angina, non-angina and normal groups respectively. There was essentially no change in the resting cardiac index following the administration of ouabain. The cardiac index during the second exercise period was higher than during the initial exercise period in all three groups, but the difference was statistically significant only in the normal subjects (P = 0.01).

Heart Rate (Fig. 6)

The average heart rates at rest were similar and rose to between 125 and 132 in all groups during exercise. Digitalization did not affect the resting heart rate or the response of the heart rate to exercise.

Oxygen Consumption

The oxygen consumption at rest and the increase during exercise were similar in all three
Mean brachial arterial pressure (B-Am). In the three groups during both study periods the administration of ouabain had no effect on the resting mean brachial arterial pressure and the response to exercise following it was similar during both exercise periods in the three groups of patients.

Systolic Ejection Rate (Fig. 7)
The systolic ejection rate during the initial exercise period increased 23% in the normal subjects and 25% in the non-angina group, but only 3% in the patients with angina. Following the administration of ouabain there was a small but significant increase in the systolic ejection rate at rest in all three groups. During the second exercise period the systolic

The increase in cardiac index (CI) during exercise was lowest in the angina group. There was no significant change in cardiac index at rest following the administration of ouabain. During the second exercise period the cardiac output was higher in each group than in the initial study, but this was significant only in the normal subjects.

The heart rates in the three groups at rest and exercise, before and after administration of ouabain, were similar.
HEMODYNAMIC RESPONSE TO EXERCISE

During the initial exercise period the systolic ejection rate index (SERI) increased in the normal and non-angina groups, but there was little change in the patients with angina. The resting systolic ejection rate index increased significantly in each group following ouabain and SERI was higher during the second exercise period than during the first period in the normal subjects, but not in the patients with coronary artery disease.

ejection rate increased 18% in the non-angina group, 21% in the normal subjects, but only 7% in the patients with angina. There was no significant change in the systolic ejection rates between the two exercise periods in the angina and non-angina groups, but in the normal subjects the systolic ejection rate was significantly greater after ouabain ($P = 0.025$).

Left Ventricular Stroke Work Index (Fig. 8)

During the initial exercise period the left ventricular stroke work index (LVSWI) increased during the initial exercise period in the normal and non-angina group, but fell in the angina group. The resting LVSWI increased significantly in all three groups following ouabain. During the second exercise period there was a significant increase in left ventricular stroke work. The levels attained in the normal and non-angina groups were similar to those in the initial study period.

left ventricular stroke work index at rest increased significantly in all three groups. During the second exercise period the left ventricular stroke work index rose in the non-angina and normal groups to levels comparable to those in the initial exercise period. In the angina group, in contrast to the initial exercise period, the left ventricular stroke work index increased during exercise after ouabain and was significantly higher than during the first period of exercise ($P < 0.001$).

dp/dt

The dp/dt during the initial rest period was higher in the angina subjects than in the normal or non-angina group. Following the administration of ouabain there was an increase in resting dp/dt in each of the three

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groups, but this was significant in only the angina group. During the second exercise period the values for dp/dt were similar to the initial exercise study for each group.

**Tension-Time Index**

The modified tension-time index was not altered at rest or during exercise in any of the three groups following the administration of ouabain.

**Discussion**

The present study was undertaken to examine the effects of cardiac glycosides in coronary artery disease, where there is, by present standards, no clinical indication for its use. Four areas were investigated: (1) the effectiveness of cardiac glycosides in preventing exertional angina; (2) their effect on the performance of the left ventricle at rest; (3) their effect on the hemodynamic response to exercise in patients with coronary artery disease who do not experience angina; and (4) their effect on the performance of the ischemic left ventricle during exertional angina.

The present investigation and that of Smith and co-workers\(^4\) show that the administration of cardiac glycosides did not prevent angina. Malmborg,\(^4\) however, found that exertional angina was prevented in 53% of patients by the prior administration of 1.2 mg of lanatoside C, possibly because he applied less severe stress, the heart rates during exercise being 114 prior to, and 108 after, digitalization, whereas the corresponding values in our subjects were 125 and 123. The most probable explanation for a reduction in the frequency of exertional angina would be decreased myocardial oxygen consumption during the post-digitalization exercise period. Cardiac glycosides have generally been considered to have no effect on myocardial oxygen consumption,\(^16-19\) although recent studies in dogs have shown that the cardiac glycosides, like other inotropic agents, increase myocardial oxygen consumption by a direct action.\(^20\) This effect would be counteracted by the reduction in left ventricular volume secondary to the inotropic effect of the drug which would decrease myocardial oxygen consumption through a reduction in wall tension.\(^21-23\) In our patients and those of Malmborg, left ventricular end-diastolic pressure was significantly lower following digitalization in the angina group suggesting a decrease in end-diastolic volume. Nitroglycerin acting probably by a similar mechanism of reduction in left ventricular volume will protect against similar stress in most patients studied\(^1, 6, 7, 24, 25\) and does not have the arrhythmogenic properties of digitalis.

The present study and that of Malmborg suggest that left ventricular function is improved at rest following digitalization in patients with coronary artery disease, even though the left ventricular end-diastolic pressure is within normal limits. In our series left ventricular end-diastolic pressure fell from 10.3 to 7.8 mm Hg in the angina group following digitalization, and Malmborg observed a decrease of 3.0 mm Hg in pulmonary capillary pressure. During exercise in the non-angina group, left ventricular end-diastolic pressure was lower following digitalization, again suggesting the beneficial effect of the drug, although this did not occur in the normal subjects. The possibility seems real that mild deficiency in left ventricular function is present in patients with coronary artery disease without angina or evidence of failure. The finding of a paradoxically split second sound\(^26\) in ambulatory asymptomatic patients after myocardial infarction suggests that left ventricular ejection rate is abnormal in these patients, even though there are no other indications of abnormal ventricular function. The mechanism of this depressed function could be myocardial fibrosis or possibly the anaerobic myocardial metabolism, which is surprisingly common in patients at rest without angina.\(^27, 28\)

Sarnoff and associates\(^19\) have shown that digitalization improves the performance of the heart during hypoxia. Although ouabain did not prevent the development of exertional angina in any of our patients, hemodynamic changes noted suggested improved ventricular performance. The left ventricular end-diastolic pressure during exercise after ouabain was significantly lower than during the first exercise.
period, and there was a rise in left ventricular stroke work in contrast to the decrease seen during the initial study. These changes, however, were small, but showed that the ischemic ventricle after digitalization is still operating on a very depressed ventricular function curve (fig. 9).

This study was not designed to determine a threshold for angina, and thus minor improvements in effort tolerance might not be detected in the present investigation. There is, however, no evidence from this study to suggest that in the absence of heart failure digitalis is of significant clinical or hemodynamic benefit in exertional angina.

References
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