Analysis of Left Ventricular Function
by Atrial Pacing

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SUMMARY
With the technique of right atrial pacing, left ventricular function was assessed in
21 normal subjects and in 13 patients with elevated left ventricular filling pressures.
Since cardiac output does not change significantly with atrial pacing, the stroke vol-
ume decreases as an inverse function of the pacing rate. Stroke volume can thus be
varied over a wide range, and by simultaneous measurement of left ventricular end-
diastolic pressure, pacing ventricular function curves can be obtained. The calculated
average slope for the ventricular function curve relating stroke volume index to left
ventricular end-diastolic pressure was steeper in the normal subjects than in the group
with elevated left ventricular end-diastolic pressure, but considerable overlap occurred
between the groups. However, in individual patients the pacing ventricular function
curve appears useful in assessment of the effect of interventions that augment or
depress ventricular performance.

Additional Indexing Words:
Hemodynamics
Angina
Ouabain

An assessment of adequacy of ventric-
ular function in man by the Starling
relationship is limited to one value relating
filling pressure to strength of contraction
unless either cardiac performance or filling
pressure is altered in some manner. In some
instances, this relationship has been examined
over a range of values by interventions such as
exercise, change in blood volume, restriction
of venous inflow, or drug administration in
which stroke volume or stroke work are altered.
However, a number of factors directly affecting myocardial contractility are
simultaneously affected by such interventions.

The introduction of atrial pacing has present-
ed an unusual opportunity for evaluation
of cardiac performance during almost experi-
mentally controlled conditions. Cardiac output
is not altered and aortic pressure, an impor-
tant determinant of myocardial performance,
is essentially unchanged during atrial pac-
ing. At the same time, stroke volume is
reduced progressively as an inverse function
of heart rate, and is accompanied by reduc-
tions in left ventricular end-diastolic pres-
sure. Precise alterations in stroke volume
can be obtained by changes in the pacemaker
rate, and a number of observations relating
filling pressure and stroke volume may be
quickly obtained without change in cardiac
output and aortic pressure or in humoral,
neural, and metabolic states. These considera-
tions led us to evaluate during pacing the
Starling mechanism in normal patients, pa-
tients with elevated left ventricular end-
diastolic pressures, and in patients after
interventions known to depress or augment
myocardial contractility, including myocardial

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ischemia and the administration of digitalis, propranolol, and isoproterenol.

Methods

Hemodynamic investigations were carried out in 34 patients. Twenty-one patients in whom coronary artery disease was suspected clinically but who subsequently showed no hemodynamic or angiographic evidence of cardiac disease were considered as normal subjects, group 1. Eleven patients with coronary artery disease and two patients with primary myocardial disease who were not in overt clinical failure at the time of study but in whom the left ventricular end-diastolic pressure at rest was greater than 12 mm Hg were considered as group 2. These investigations were clinically indicated in all patients, and informed consent was obtained. The patients were studied while they were in the fasting state without premedication. Under local anesthesia the brachial artery and two veins were isolated in the right antecubital fossa. A no. 9 double lumen or a no. 7 or 8 single lumen Cournand catheter was placed so the tip lay in the pulmonary artery. Pacing was achieved by either a no. 6 bipolar pacemaker catheter positioned against the lateral wall of the right atrium or a no. 8 Goodale-Lubin catheter, modified by the incorporation of bipolar electrodes 1.5 and 3.0 cm from the tip, placed in the mid-position of the coronary sinus. A no. 8 Sones catheter was introduced into the left ventricle from the right brachial artery, and the left brachial artery was cannulated with a short Teflon catheter by the Seldinger technique.

Lead II of the electrocardiogram and pressures from the pulmonary artery, brachial artery, and left ventricle were recorded during a 10-min control period. The cardiac output was measured in duplicate by the dye-dilution technique with indocyanine green during the final 2 min of the control period. In 16 normal subjects and three patients with elevated left ventricular filling pressures a 6- to 8-min period of pacing* at a constant rate for each patient, ranging from 110 to 160 beats/min, was then carried out, during which time the electrocardiogram and pressures were recorded continuously. The cardiac output was determined between the fifth and seventh minute of pacing.

In five patients of group 1 and nine of group 2 the heart rate was increased in steps of 10 to 15 beats/min and maintained at each new rate for 2 min. In these studies, pressure measurements were obtained at each rate and the cardiac output measured at two, three, or four pacing rates.

Interventions

Multiple point analyses were carried out in one normal subject and five patients with elevated left ventricular filling pressures before and after interventions for alteration of left ventricular contractility. In three of the latter group this was carried out 30 min after the intravenous administration of 0.75 mg of ouabain and in the normal subject, 12 min after the intravenous administration of propranolol (0.15 mg/kg) and 15 min later during an isoproterenol infusion (4 μg/min). Ventricular function was analyzed prior to and during angina pectoris in the other two patients of this group. We designed the control study to examine the effect of multiple rates without precipitating angina. This was accomplished by pacing for 2 min at each rate and allowing periods of sinus rhythm for at least 2 min between pacing periods. Ten minutes later, angina was precipitated by pacing at a rate of 140 to 150 beats/min for several minutes. Thereafter the rate was abruptly reduced and maintained at this reduced rate for 1 to 2 min during hemodynamic measurements. The rate was then restored to the maximal rate and maintained for at least 2 min prior to subsequent rate reduction. In this manner the effect of several different rates was examined in the presence of continuing angina. The cardiac outputs were measured before and after the interventions while in sinus rhythm and during at least one pacing rate near the maximum.

Direct left atrial pressures were obtained in six patients (subjects of a separate study) by the transseptal technique along with retrograde left ventricular catheterization. Simultaneous pressures obtained from the left atrium and the left ventricle at multiple pacing rates in one such patient are reported here.

Pressures were measured with P23Db Statham strain gauges from a zero reference level 5 cm below the angle of Louis and were recorded on a photographic recorder (Electronics for Medicine DR12 recorder, White Plains, New York). Pressures were measured over at least two respiratory cycles, and the mean pressures in the brachial and pulmonary arteries were obtained electronically. Recording speed was normally 25 mm/sec, but for determination of ventricular end-diastolic pressure a high sensitivity was employed with a speed of 100 mm/sec.

The precise point where left ventricular end-diastolic pressure was measured corresponded with the nadir of the atrial kick in the left ventricular pressure tracing. In the tracings where the atrial kick could not be clearly defined, the left ventricular end-diastolic pressure was measured at a point corresponding with the peak of the R wave of the electrocardiogram and/or

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ATRIAL PACING

\[ \text{LVSWI} = \frac{\text{SI} \times (\text{BAm} - \text{LVEDP}) \times 13.6}{1000} \]

where LVSWI = left ventricular stroke-work index in g-m/m², SI = stroke index in ml/m², BAm = brachial artery mean pressure in mm Hg, and LVEDP = left ventricular end-diastolic pressure in mm Hg. If the factor of left ventricular end-diastolic pressure is neglected, stroke work and stroke volume changes are parallel in the presence of a constant aortic mean pressure, and the graphical representation of stroke work or stroke volume vs. left ventricular end-diastolic pressure is similar. This correction for left ventricular end-diastolic pressure in the calculation of stroke work is minimal up to 12 mm Hg (fig. 1), so that stroke volume is an accurate representation of true stroke work over this range.

Calculations

Graphical representation of the Starling relationship is presented throughout this paper as the relationship between stroke volume and filling pressure. This expression was used instead of the more commonly employed stroke work¹, 3, 4, 6, 10, 11, 13, 14 since aortic pressure was not changed significantly during pacing. Left ventricular stroke work was calculated for each point according to the standard formula:

\[ \text{LVSWI} = \frac{\text{SI} \times (\text{BAm} - \text{LVEDP}) \times 13.6}{1000} \]
of left ventricular end-diastolic pressures in this constant aortic pressure study.

This correction for left ventricular end-diastolic pressure becomes increasingly important at higher filling pressures, tending to depress the ventricular function curve at this area (fig. 1). The concept of equating energy of contraction with increment of pressure achieved by the ventricle at a given flow has been questioned, and evidence given that the subtraction of filling pressure is responsible for the descending limb of the Starling curve. While hydraulically correct, this subtraction may not be meaningful in terms of ventricular function. Moreover, Monroe et al. have shown that energy of contraction is undiminished even at left ventricular filling pressures up to 100 mm Hg. Also, stroke work can be considerably increased in the absence of changes in left ventricular end-diastolic pressure due to an increase in after-load alone. With these considerations in mind, it appeared more reasonable to relate actual parameters measured (filling pressure and stroke volume) rather than calculated values.

### Results

**Cardiac Index, Stroke-Volume Index, and Arterial Pressure During Rate Change**

Control cardiac index was 2.99 liters/min/m² in group 1 and 2.60 liters/min/m² in group 2, and at maximal pacing rates there was no change in cardiac index from control values (table 1). Cardiac index was averaged at three different pacing rates in five normal subjects and in the nine patients of group 2 with multiple rate points (fig. 2). There was no change in either group at rates ranging from 57 to 166 beats/min.

Control brachial arterial mean pressure was 98.5 mm Hg in group 2 and 92.2 mm Hg in the normal subjects. At maximal rate of pacing these values became 103 and 95.4 mm Hg, respectively. No change was apparent at intermediate rates in the multiple point group (fig. 2). Stroke-volume index fell progressively in both groups as the rate was increased (fig. 3).

### Left Ventricular End-Diastolic Pressure During Rate Change

Left ventricular end-diastolic pressure fell in all patients studied when the rate was increased. When examined at multiple rates (table 2 and fig. 4) left ventricular end-diastolic pressure in the normal subjects fell in a linear fashion, approaching zero at the higher rates. In the group with elevated left ventricular filling pressures a more rapid but an approximately linear fall also occurred, although left ventricular end-diastolic pressure did not reach the low levels seen in the normal subjects. At a rate of 130 beats/min left ventricular end-diastolic pressure in this group ranged from 6 to 16 mm Hg, whereas in the

### Table 1

**Hemodynamic Data for the Groups During Normal Sinus Rhythm and at Maximum Pacing Rate**

<table>
<thead>
<tr>
<th>Status</th>
<th>HR (beats/min)</th>
<th>LVEDP (mm Hg)</th>
<th>SVI (ml/m²)</th>
<th>CI (liters/min/m²)</th>
<th>BAm (mm Hg)</th>
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</thead>
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<tr>
<td><strong>Group 1 (n = 21)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>NSR</td>
<td>75.2 (11.9)</td>
<td>8.7 (2.3)</td>
<td>40.2 (6.3)</td>
<td>2.99 (0.52)</td>
<td>92.2 (10.8)</td>
</tr>
<tr>
<td>Pacing</td>
<td>140.9 (11.3)</td>
<td>2.0 (1.7)</td>
<td>22.8 (8.1)</td>
<td>3.18 (0.70)</td>
<td>95.4 (10.9)</td>
</tr>
<tr>
<td><strong>Group 2 (n = 13)</strong></td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>NSR</td>
<td>78.5 (17.8)</td>
<td>21.3 (5.5)</td>
<td>34.2 (9.2)</td>
<td>2.60 (0.52)</td>
<td>98.5 (22.5)</td>
</tr>
<tr>
<td>Pacing</td>
<td>139.7 (17.9)</td>
<td>7.6 (3.7)</td>
<td>19.1 (6.5)</td>
<td>2.60 (0.72)</td>
<td>103.0 (23.3)</td>
</tr>
</tbody>
</table>

Abbreviations: HR = heart rate; LVEDP = left ventricular end-diastolic pressure; SVI = stroke-volume index; CI = cardiac index; BAm = brachial artery mean pressure; NSR = normal sinus rhythm.

Values in parentheses represent the standard deviation from the mean.

Group 1 = normal patients; Group 2 = patients with elevated LVEDP.
Table 2

Hemodynamic Data During Sinus Rhythm and at Multiple Heart Rates

<table>
<thead>
<tr>
<th>Patient Diagnosis</th>
<th>Status</th>
<th>HR (beats/min)</th>
<th>LVEDP (mm Hg)</th>
<th>SVI (ml/m²)</th>
<th>CI (liters/min/m²)</th>
<th>BAm (mm Hg)</th>
</tr>
</thead>
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<td></td>
</tr>
<tr>
<td>1</td>
<td>NSR</td>
<td>72</td>
<td>43.0</td>
<td>3.12</td>
<td>98</td>
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</tr>
<tr>
<td>P₁</td>
<td>99</td>
<td>6</td>
<td>31.0</td>
<td>3.11</td>
<td>106</td>
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<tr>
<td>P₂</td>
<td>130</td>
<td>5</td>
<td>25.0</td>
<td>3.27</td>
<td>130</td>
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<tr>
<td>P₃</td>
<td>160</td>
<td>2</td>
<td>19.0</td>
<td>2.98</td>
<td>118</td>
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</tr>
<tr>
<td>2</td>
<td>NSR</td>
<td>91</td>
<td>47.0</td>
<td>4.23</td>
<td>107</td>
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</tr>
<tr>
<td>P₁</td>
<td>117</td>
<td>2</td>
<td>36.1</td>
<td>4.23*</td>
<td>104</td>
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<tr>
<td>P₂</td>
<td>138</td>
<td>0</td>
<td>30.6</td>
<td>4.23*</td>
<td>105</td>
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<tr>
<td>P₃</td>
<td>153</td>
<td>0</td>
<td>28.0</td>
<td>4.26</td>
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<td>3</td>
<td>NSR</td>
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<tr>
<td>P₁</td>
<td>92</td>
<td>5</td>
<td>50.0</td>
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</tr>
<tr>
<td>P₂</td>
<td>100</td>
<td>2</td>
<td>45.6</td>
<td>4.56*</td>
<td>90</td>
<td></td>
</tr>
<tr>
<td>P₃</td>
<td>110</td>
<td>1</td>
<td>41.4</td>
<td>4.56*</td>
<td>91</td>
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</tr>
<tr>
<td>4</td>
<td>NSR</td>
<td>57</td>
<td>36.0</td>
<td>2.05</td>
<td>90</td>
<td></td>
</tr>
<tr>
<td>P₁</td>
<td>70</td>
<td>5</td>
<td>28.6</td>
<td>2.00</td>
<td>95</td>
<td></td>
</tr>
<tr>
<td>P₂</td>
<td>102</td>
<td>2</td>
<td>22.8</td>
<td>2.33</td>
<td>95</td>
<td></td>
</tr>
<tr>
<td>P₃</td>
<td>120</td>
<td>0</td>
<td>19.6</td>
<td>2.35</td>
<td>90</td>
<td></td>
</tr>
</tbody>
</table>

Group 1

|                  |        |                |               |             |                   |             |
|                  |        |                |               |             |                   |             |
| 1                | NSR    | 76             | 47.0          | 3.55        | 110               |             |
| CAD              |        |                |               |             |                   |             |
| P₁               | 98     | 12             | 36.2          | 3.55*       | 116               |             |
| P₂               | 145    | 5              | 25.0          | 3.58        | 108               |             |
| P₃               | 157    | 2              | 22.8          | 3.58*       | 110               |             |
| 2                | NSR    | 90             | 27.3          | 2.46        | 80                |             |
| PMD              |        |                |               |             |                   |             |
| P₁               | 105    | 18             | 20.7          | 2.18        | 80                |             |
| P₂               | 125    | 15             | 17.4          | 2.18*       | 85                |             |
| P₃               | 150    | 13             | 13.2          | 1.98        | 80                |             |
| 3                | NSR    | 96             | 23.0          | 2.23        | 80                |             |
| CAD              |        |                |               |             |                   |             |
| P₁               | 114    | 14             | 19.5          | 2.23*       | 85                |             |
| P₂               | 140    | 12             | 16.9          | 2.37        | 85                |             |
| P₃               | 150    | 11             | 14.0          | 2.06        | 85                |             |
| 4                | NSR    | 112            | 21.0          | 2.36        | 73                |             |
| CAD              |        |                |               |             |                   |             |
| P₁               | 125    | 13             | 18.8          | 2.36*       | 75                |             |
| P₂               | 150    | 11             | 14.6          | 2.19        | 72                |             |
| P₃               | 166    | 10             | 12.7          | 2.11        | 75                |             |
| 5                | NSR    | 66             | 31.0          | 2.07        | 97                |             |
| CAD              |        |                |               |             |                   |             |
| P₁               | 90     | 17             | 27.0          | 2.45        | 98                |             |
| P₂               | 103    | 13             | 23.7          | 2.45*       | 90                |             |
| P₃               | 135    | 6              | 11.0          | 1.46        | 105               |             |

Abbreviations: P₁ = pacing 1; P₂ = pacing 2; P₃ = pacing 3; CAD = coronary artery disease; PMD = primary myocardial disease; others = same as in table 1.

Groups = same as in table 1.

Control values of patients 5 of the normal group and 6, 7, 8, and 10 of the failure group also included in the analysis of multiple heart rates are shown in table 3.

*Value presumed same as determined at the immediate previous heart rate.

normal subjects it ranged from 1 to 6 mm Hg. A flattening of the left ventricular end-diastolic pressure vs. rate relationship occurred at approximately 12 mm Hg in four patients of group 2 at rates above 130 beats/min.
Stroke-volume index (SVI) fell progressively in both groups as the pacing rate was increased. Groups = same as in figure 2.

Relation of Stroke-Volume Index and Left Ventricular End-Diastolic Pressure

Evaluation at several rates (fig. 5) shows that the relation between stroke-volume index and filling pressure is linear during the wide variations in both parameters resulting from pacing. This is seen in both groups, and the slope of this line was steeper for the normal group.

Since this multiple rate analysis showed a linear relationship between left ventricular end-diastolic pressure and stroke-volume index, an additional group of patients, 16 normal subjects and three patients of group 2, were included for analysis who were studied at only two rates, a control and a pacing rate averaging 141 beats/min. Figure 6 illustrates all 21 normal subjects on the left, with two extreme points from each and a connecting line. Two extreme points from each patient of group 2 are presented on the right at the same scale. A broad line representing the calculated average slope is drawn for each group. The slope for group 1 was 3.148 (±1.792 s.d.) and for group 2, 1.141 (±0.543 s.d.) (P < 0.001). It is apparent that there is considerable overlap between the two groups.

Interventions

Angina was produced in two patients and the ventricular function curve was obtained prior to and during angina (table 3). Cardiac output was not significantly changed from control during pacing-induced angina. The ventricular function curves obtained in one such patient are shown in figure 7, and it is evident that ventricular function is depressed during myocardial ischemia so that, in order to deliver a stroke-volume index of 24 ml/m², a left ventricular end-diastolic pressure of 9 mm Hg was required in the control state and

![Figure 3](image1)

![Figure 4](image2)
In one normal subject the effect of intravenous propranolol was studied and also the effect of a subsequent infusion of isoproterenol (table 3). The cardiac output fell after propranolol administration, and the ventricular function curve is shown in figure 9. It is apparent that the ventricular function is depressed by this drug. At a stroke-volume index of 26 ml/m² the left ventricular end-diastolic pressure rose from 5 to 13 mm Hg. Isoproterenol infusion reversed this depression of left ventricular function.

Figure 10 illustrates the changes in left ventricular end-diastolic pressure and directly obtained left atrial pressure at multiple pacing rates in one patient.

Discussion

It is evident that the manipulation of stroke volume through changes in heart rate during atrial pacing permits an extended examination of the relationship between stroke volume or stroke work and ventricular filling pressure. As heart rate can be varied greatly, this relationship may be observed over a range of stroke volumes and filling pressures. The finding that this is a linear relationship simplifies examination by this method, as it is possible for the function of any ventricle to be described by determination of cardiac output and left ventricular end-diastolic pressure during sinus rhythm and at a single pacing rate. The question may be raised as to why the pacing ventricular function curve differs from that customarily seen, which exhibits a sharply ascending limb at low filling pressures followed by a plateau developing at approximately 15 to 20 mm Hg. One possibility is that this linearity results from the progressive increases in rate at the lower filling pressures with their accompanying increase in the force generated. Evidence that this linear relationship may be due instead to the constant aortic pressure is seen in the isolated heart studies of Sarnoff and Mitchell. They found a linear relationship between stroke work and filling pressure when stroke volume was raised while aortic pressure was kept constant. When stroke volume was increased

**Figure 5**

The relationship between stroke-volume index (SVI) and left ventricular end-diastolic pressure (LVEDP) in patients studied at multiple pacing rates. For any change in SVI there is a greater decrease in LVEDP in group I than in group II.

20 mm Hg during myocardial ischemia. Similar depression was apparent in the other patient during pacing-induced angina. Although a second curve was not obtained after disappearance of angina, the left ventricular end-diastolic pressure returned to its control values after the disappearance of angina following cessation of pacing, indicating a return to the initial ventricular function curve.

In three patients of the group with elevated left ventricular filling pressures the effect of acute digitalization was studied (table 3). The cardiac output was unchanged in two patients and decreased in the third following administration of ouabain, but ventricular function was improved in all three. The ventricular function curves from one patient whose output did not change is shown in figure 8, and it is evident that ventricular function is improved following the administration of ouabain. At a stroke-volume index of 40 ml/m² the left ventricular end-diastolic pressure falls from 24 to 14 mm Hg after digitalization.

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### Table 3

**Hemodynamic Data Before and Following Interventions at Multiple Heart Rates**

<table>
<thead>
<tr>
<th>Patient Diagnosis</th>
<th>Status</th>
<th>Oubain intervention</th>
<th>Angina intervention</th>
<th>Propranolol and isoproterenol intervention</th>
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</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>HR (beats/min)</td>
<td>LVEDP (mm Hg)</td>
<td>SVI (ml/m²)</td>
</tr>
<tr>
<td>6</td>
<td>NSR</td>
<td>52 51 24 16</td>
<td>40.0 43.0</td>
<td>2.08 2.18</td>
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<td></td>
<td>PMD</td>
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<tr>
<td>7</td>
<td>CAD</td>
<td>80 74 17 10</td>
<td>38.8 34.0 3.11 2.52</td>
<td>105 100</td>
</tr>
<tr>
<td></td>
<td></td>
<td>140 135 6 5</td>
<td>23.5 24.7 3.29 3.29</td>
<td>105 100</td>
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<tr>
<td>8</td>
<td>NSR</td>
<td>100 84 20 17</td>
<td>26.0 31.0 2.58 2.57</td>
<td>70 73</td>
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<tr>
<td>9</td>
<td>NSR</td>
<td>87 - 13 - 28.0 - 2.47 -</td>
<td>112 -</td>
<td>CAD</td>
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<tr>
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<td>150 -</td>
<td>CAD</td>
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</tbody>
</table>

**Abbreviations:** P1 = pacing 1; P2 = pacing 2; P3 = pacing 3; P4 = pacing 4; P5 = pacing 5; C = control; I = following intervention; others = same as in Table 2.

*Value presumed same as determined at the immediate previous heart rate.

†Values during isoproterenol intervention.

and aortic pressure allowed to increase simultaneously, the typical Starling-type curve was obtained. In view of the finding that aortic pressure, and hence stroke work, may be considerably elevated without significant alterations in end-diastolic pressure or end-diastolic volume, it would appear that the best evaluation of the Frank-Starling relationship is obtained at a constant after-load, which is fortuitously present during the pacing ventricular function curve described here.

Confirmation that these ventricular function curves reflect the state of myocardial contractility is seen after the administration of cardiac glycosides, where a shift in the ventricular function curve occurs upwards and to the left. The depressant effect of propranolol on ventricular performance and its reversal with isoproterenol likewise supports the validity of

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The relationship between stroke-volume index (SVI) and left ventricular end-diastolic pressure (LVEDP) in all patients. The value during sinus rhythm and at the maximal pacing rate are joined, and the broad line depicts the average of the individual slopes. The slope in group I (3.148) is steeper than that in group 2 (1.141) (P < 0.001).

This technique. The demonstration that the ventricular function curve is transiently depressed during myocardial ischemia is also clear. This reversible cardiac failure during myocardial ischemia was reported by us previously on the basis of two points; one obtained during pacing-induced angina and the other in the immediate post-pacing period. The present study demonstrates this depression more conclusively.

The present study shows that analysis of left ventricular end-diastolic pressure in terms of left ventricular function is not meaningful during pacing unless the heart rate is also considered. Extension of this concept to patients with sinus rhythm at different heart rates appears logical. Thus during bradycardia one would expect a higher left ventricular end-diastolic pressure with normal ventricular function and lower left ventricular end-diastolic pressure during tachycardia. A high left ventricular end-diastolic pressure may be due to an elevated position on a normal Starling curve, depression of ventricular performance, or reduced ventricular compliance. Because of the nature of the patients studied, reduced compliance may have been a frequent feature, and the elevated left ventricular end-diastolic pressure due in part to reduced compliance and in part to impaired contractility. With a stiff ventricle it is conceivable that greater changes in filling pressures would occur as stroke volume is reduced and, thus, a more normal appearing ventricular function curve would result.

This method of evaluating contractility
obviously separated by the pacing ventricular function curve, this method appears to be of little value in predicting mild or incipient failure. In fact, measurement of the heart rate, left ventricular end-diastolic pressure, and cardiac output during sinus rhythm supplies as much information as the entire curve in separating the abnormal from normal hearts.

This may be related to the fact that with this technique we are able to explore only the region of the Starling curve below the control value. The primary usefulness of a pacing ventricular function curve appears to be in the evaluation of acute interventions. It would be applicable as a rapid and simple assessment of the effects of drugs on myocardial contractil-

Figure 7
Ventricular function curve in patient 10 with and without angina. Depression of ventricular performance is evident during myocardial ischemia.

Figure 8
Ventricular function curve in patient 6 demonstrating improvement in ventricular performance after administration of ouabain.

Figure 9
Ventricular function curve in patient 5 demonstrating depression of ventricular performance after propranolol administration, and subsequent improvement with isoproterenol administration.
Atrial pacing

It is shown in figure 10, where pressures were measured directly from the left atrium and left ventricle simultaneously while the rate was raised by pacing. The two pressures are similar at a control rate of 70 beats/min and at a rate of 115 beats/min, but beyond this the left atrial pressure rises sharply while left ventricular end-diastolic pressure falls. At the rate of 155 beats/min, when left ventricular end-diastolic pressure has fallen to zero, the left atrial pressure has risen to its highest value of 14 mm Hg from a control value of 8 mm Hg. This rise in left atrial pressure was due to contraction of the atrium while the atrio-ventricular valve was closed, thus raising mean atrial pressure. A tracing from this patient showed a superimposition of the A and V waves at the higher rates, as reported by Leighton and associates. A similar situation undoubtedly obtains for the right atrium and right ventricular end-diastolic pressure at higher pacing rates.

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Figure 10

Left atrial mean pressure (LAm) and left ventricular end-diastolic pressure (LVEDP) are similar at lower pacing rates, but with rapid rates LAm pressure rises as LVEDP continues to fall.
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