Left Heart Hemodynamics During Angina Pectoris Induced by Atrial Pacing

By JOSEPH W. LINHART, M.D., FRANK J. HILDNER, M.D., S. SERGE BAROLD, M.B., JOHN W. LISTER, M.D., AND PHILIP SAMET, M.D.

SUMMARY

In order to determine the associated hemodynamic events, angina pectoris was induced by an atrial pacing technic. In all patients developing angina pectoris, ischemic changes were noted electrocardiographically. The hemodynamic findings were variable: in some patients no changes were noted; in others, significant increases in cardiac output, left ventricular end-diastolic pressures (LVEDP), and femoral arterial and pulmonary arterial pressures occurred. These changes suggest that, initially, ischemia produces a decrease in myocardial compliance and this may also be associated with enhanced sympathetic nervous system activity. Myocardial failure may subsequently ensue, however. The administration of nitroglycerin, during angina at a fixed rapid heart rate, resulted in a reduction in LVEDP and cardiac work. Chest pain was always relieved following these hemodynamic changes, indicating a dependence upon these changes for the effectiveness of nitroglycerin.

Additional Indexing Words:
Coronary artery disease
Left ventricular end-diastolic pressure
Nitroglycerin
Sympathetic nervous system
Myocardial compliance

ALTHOUGH angina pectoris was accurately described 200 years ago, understanding of the coincident hemodynamic events is still limited.1 Various hemodynamic changes have been described by different investigators, but there has been no general agreement as to precise alterations. In part, this may be due to the evaluation of different population groups and the use of varying methods to induce the chest pain.2-16 However, even the intravascular events associated with spontaneously developing angina appear not to be consistent.5-7, 15, 17

Angina occurring clinically is usually produced by exertion; however, the hemodynamic changes induced by exercise and the underlying state of the myocardium may obscure the effects of the ischemia itself. Therefore, numerous investigators have adopted the use of an atrial pacing technic to increase myocardial oxygen consumption, independent of any effects on total body work load. It has been shown that increasing the heart rate by right atrial pacing in patients with coronary artery disease can provoke anginal pain, with associated electrocardiographic changes and altered hemodynamics. However, the hemodynamic changes with the induced angina have not been consistent.4, 7, 13, 18-21 In this study, right atrial pacing was used to induce angina pectoris in patients with a known amount of coronary artery disease (CAD) (proven by coronary arteriography) and to compare the hemodynamic changes of angina pectoris to those induced by atrial pacing in normal patients and others with CAD not developing angina pectoris. It was anticipated that atrial pacing would permit this evaluation under safe, controlled conditions, obvi-

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ating the problems of reaching a steady state that one has with exercise-induced angina pectoris. Additionally, the changes induced by sublingual nitroglycerin were examined after angina pectoris was induced.

Methods
Twenty-six patients, 21 male and five female, ranging in age from 29 to 69 years (mean 51 years), were studied in the postabsorptive state under light pentobarbital sedation. Each patient was informed regarding the nature and technic of the investigation. In each instance, coronary arteriography was indicated either for the evaluation of atypical chest pain (10 patients) or in patients with angina pectoris as part of a preoperative study. Therefore, those with true angina were subsequently shown to have moderate to severe coronary artery disease with occlusions of greater than 50% of a vessel diameter in two or three coronary artery branches. Of the 16 patients with coronary artery disease (CAD), 14 were considered to have angina by the usual criteria and in two the chest symptoms were atypical enough to cause doubt in the minds of some examiners. A mild to moderate degree of cardiomegaly was present in eight of the 16 with CAD, and six patients had a history of previous myocardial infarction.

Under local anesthesia with lidocaine, an indwelling needle was placed in the left femoral artery (LFA), and a cutoff was performed in the right antecubital area. With standard technics, one catheter was placed in the pulmonary artery (PA) through an antecubital vein and another in the left ventricle (LV) retrograde from the right brachial artery. A bipolar electrode catheter was then positioned in the right atrium. Control measurements and the cardiac output were determined as previously described, with dye curves performed with PA injections and LFA sampling. The electrocardiogram (lead II only) was continuously monitored.

These measurements were repeated sequentially as the heart rate was gradually increased by right atrial pacing. The pacing rate was increased in increments of 10 beats per minute, and the hemodynamic parameters were recorded after every two or three increases in rate. A 3-to-4 minute period of stability was allowed for each point, except when angina pectoris was precipitated. Angina pectoris was considered to have occurred after the heart rate was increased and chest pain developed similar to that occurring clinically and having the usual characteristics. The highest pacing rate was determined individually for each patient and depended upon his response. In those developing angina pectoris with an increase in heart rate, this rate was the limiting one. In those not developing chest pain, the highest rate used was that which permitted 1:1 atrioventricular conduction (without any second-degree block) or the highest rate not associated with any decrease in arterial blood pressure.

In three patients in whom angina pectoris developed, the hemodynamics were recorded promptly and then the pacing was discontinued. In six other patients with angina, the pacing rate was maintained at the rate inducing angina and the patient was given 0.6 mg of nitroglycerin sublingually. The various hemodynamic measurements were repeated, and the electrocardiogram was monitored during and after the response to this drug.

After atrial pacing, during normal sinus rhythm, a left ventricular cineangiogram was performed, followed by selective coronary arteriography. These films were then evaluated and graded to determine the presence and magnitude of any coronary artery disease. In general, significant coronary artery disease was considered to be present when there was at least a 50% occlusive lesion in one major coronary artery with evidence of lesser lesions in other vessels. The technics used, the evaluation of the films, and the grading systems have been previously described.

Results
General Observations
All the patients were in normal sinus rhythm, and no persisting rhythm disturbances were induced by atrial pacing. Except for the induction of angina in some patients, the pacing studies were uneventful.

On the basis of the angiographic and hemodynamic studies, the patients could be divided into three groups. The first group consisted of 10 patients who were normal in all respects. Sixteen patients had significant CAD, nine had angina pectoris during pacing (group 2), and seven did not (group 3). When these groups were compared as to the hemodynamic effects of pacing, it was seen that those with CAD and no angina, and those considered normal, behaved in a similar fashion (table 1).

Heart rate was increased in those with CAD and no induced angina from 70±3 to 119±7 beats per minute, and in the normal subjects from 72±3 to 132±4 beats per minute. There is no difference in the pacing rates between the two groups (0.2 > P > 0.1). In both
ANGINA PECTORIS INDUCED BY ATRIAL PACING

Table 1

Hemodynamic Response to Atrial Pacing in Normal Patients and Those with Coronary Artery Disease Not Developing Angina Pectoris

<table>
<thead>
<tr>
<th>Coronary artery disease</th>
<th>HR</th>
<th>LVEDP</th>
<th>FA</th>
<th>PA</th>
<th>CO</th>
<th>SV</th>
</tr>
</thead>
<tbody>
<tr>
<td>(no induced angina)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>70 ± 3</td>
<td>13 ± 3</td>
<td>130 ± 12</td>
<td>18 ± 1</td>
<td>4.9 ± 0.3</td>
<td>70 ± 6</td>
</tr>
<tr>
<td>Pacing</td>
<td>119 ± 7</td>
<td>4 ± 1</td>
<td>120 ± 3</td>
<td>17 ± 2</td>
<td>5.1 ± 0.4</td>
<td>39 ± 4</td>
</tr>
<tr>
<td>Normal subjects</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>72 ± 3</td>
<td>8 ± 1</td>
<td>136 ± 8</td>
<td>23 ± 2</td>
<td>4.3 ± 0.4</td>
<td>66 ± 4</td>
</tr>
<tr>
<td>Pacing</td>
<td>132 ± 4</td>
<td>2 ± 1</td>
<td>142 ± 12</td>
<td>19 ± 1</td>
<td>4.5 ± 0.4</td>
<td>45 ± 5</td>
</tr>
</tbody>
</table>

Pressures are in millimeters of mercury; LVEDP = left ventricular end-diastolic pressure; FA = femoral artery systolic pressure; PA = pulmonary artery systolic pressure; CO = cardiac output in liters per minute; SV = stroke volume in milliliters.

groups, there was a significant decrease in LVEDP (CAD, \( P = 0.01 \); normals, \( P = 0.001 \)) and in stroke volume (\( P = 0.001 \) for both), but no difference between the responses when the CAD patients were compared to the normal \( (P = 0.2) \). No significant changes occurred during pacing, in femoral artery pressures within the same groups \( (P = 0.2) \) or between the two groups \( (0.1 > P > 0.05) \). The cardiac output values also did not change during atrial pacing \( (0.3 > P > 0.2) \) within and between the two groups. Although the control pulmonary artery systolic pressure was slightly higher in the normal subjects \( (0.05 > P > 0.01) \), there were no significant differences in their lack of change with atrial pacing \( (CAD, P = 0.6; \) normal, \( P = 0.1) \).

It is apparent that with atrial pacing, there is no difference in the hemodynamic responses of these two groups. Since these groups are hemodynamically comparable, in this study, changes in left ventricular end-diastolic pressure (LVEDP) for the combined groups (17 patients) are shown in figure 1. As the average rate was increased from 71 to 128 beats per minute, there was a progressive fall in LVEDP \( \text{control} 10 ± 2 \text{ mm Hg; pacing} 3 ± 1 \text{ mm Hg; } P = 0.01) \). No significant ischemic electrocardiographic changes occurred in either of these two groups of patients during atrial pacing.

The following results are limited to the nine subjects in whom angina developed during atrial pacing.

Figure 1

Changes in left ventricular end-diastolic pressure (LVEDP) as the heart rate is increased by right atrial pacing in those patients not developing chest pain. The first point is the unpaced control level, and subsequent points are during atrial pacing.

 Electrocardiographic Observations

Chest pain occurred at an average rate of 114 beats per minute with a range of 93 to 138. In each instance, there was an associated ischemic depression of the ST segment, which was noted 20 to 30 seconds prior to the onset of the chest pain (fig. 2). With the relief of pain afforded by either nitroglycerin or the elimination of atrial pacing, the elec-
trocardiogram normalized prior to the subsidence of pain.

**Left Ventricular End-Diastolic Pressure (LVEDP)**

Figure 3 demonstrates the changes in LVEDP in the nine patients in whom angina pectoris developed during pacing. In five there was a definite increase in LVEDP during angina pectoris. This increase occurred prior to the onset of chest pain, and it was possible to predict the development of pain in any individual patient by noting this change. Except for one patient, the initial response to atrial pacing in these patients was normal, that is, a decrease in LVEDP. In the four patients in whom LVEDP did not rise with the onset of chest pain, it fell in a normal fashion, despite the previously described electrocardiographic changes.

**Systemic Arterial Pressure**

There was no consistent change noted in arterial pressure in this group of patients (fig. 4). The most dramatic change, from 118 to 154 mm Hg systolic pressure, was associated with the largest increase (8 to 33 mm Hg) in LVEDP and occurred coincident with this latter elevation. However, increases in LVEDP occurred in the absence of any systemic pressure changes.
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Pulmonary arterial pressure responses during pacing before and after the development of angina.

Figure 6
The changes in cardiac output with pacing, with and without associated anginal pain.

Pulmonary Artery Pressure
The pulmonary artery pressure (fig. 5) also did not change consistently. Prominent increases in two patients were associated with

The changes in stroke work and LVEDP after the administration of sublingual nitroglycerin (NTG). The heart rate is kept constant during angina at an average rate of 118 per minute by atrial pacing. The chest pain is no longer present after NTG.

the largest rises in LVEDP, suggesting a passive response in the pulmonary artery bed.

Cardiac Output
Figure 6 shows that the cardiac output increased during anginal pain in five of nine patients. This rise could not be correlated directly with an increase in arterial pressure or LVEDP. In the others, no change occurred, as is usual during atrial pacing.

Effect of Nitroglycerin During Angina Pectoris at a Constant Heart Rate
Angina pectoris occurred in six patients at an average paced heart rate of 118±8 beats per minute. With the rate kept constant by atrial pacing, there was a fall in arterial pressure, cardiac output, and stroke volume and no change in systemic vascular resistance after the administration of sublingual nitroglycerin (table 2). Figure 7 demonstrates the consistent reductions in stroke work and LVEDP after nitroglycerin at the same heart rate that
had induced angina. These hemodynamic changes reached their maximum at 2 to 3 minutes after nitroglycerin administration, at which time the angina also was relieved. The pressures (systemic arterial and LVEDP) always decreased prior to the relief of angina pectoris, and, in fact, it was possible to predict this relief by noting the pressure change.

**Discussion**

It has been demonstrated that increases in heart rate may uncover inadequacies in the coronary reserve of patients with coronary artery disease. Prominent depressions in the ST segment of the electrocardiogram may be produced, both with and without the development of angina pectoris, which generally occurs subsequent to the electrocardiographic change. This sequence is quite reproducible in any given patient and is related to the increase in myocardial oxygen consumption coincident with an increased heart rate.

In this study, the atrial pacing method was utilized in an attempt to produce angina in patients with coronary artery disease, in order to evaluate any associated hemodynamic changes. In normal subjects, a similar increase in heart rate engendered no changes in systemic or pulmonary arterial pressure, and the cardiac output remained constant. Stroke volume and LVEDP decreased. In this series, patients with coronary artery disease, who did not develop chest pain during acceleration of their heart rate, behaved hemodynamically like normal patients.

By comparison, if anginal chest pain develops, a patient may or may not have significant hemodynamic changes. Usually the initial response to atrial pacing was indistinguishable from normal. However, in five of nine patients, following an initial decline, the LVEDP rose. This was accompanied, in those with the largest EDP increases, by elevations in pulmonary artery and femoral artery pressures and occasionally by increases in cardiac output. In four patients, ST depression and chest pain occurred in the absence of significant hemodynamic changes.

It is apparent that the hemodynamic responses to the clinical syndrome of angina pectoris, that is, characteristic chest pain and electrocardiogram, will vary in different individuals. This occurs regardless of the method utilized to precipitate angina pectoris, as similar variations in response are evident during

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**Table 2**

*Intravascular Responses to Nitroglycerin in Patients with Angina Pectoris, with Heart Rate Maintained by Atrial Pacing*

<table>
<thead>
<tr>
<th>Pt</th>
<th>State</th>
<th>HR</th>
<th>LVEDP</th>
<th>FA</th>
<th>CO</th>
<th>SV</th>
<th>SW</th>
<th>SVR</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>A</td>
<td>93</td>
<td>12</td>
<td>102</td>
<td>4.4</td>
<td>48</td>
<td>66</td>
<td>1860</td>
</tr>
<tr>
<td>N</td>
<td>93</td>
<td>7</td>
<td>93</td>
<td>4.2</td>
<td>45</td>
<td>57</td>
<td>1770</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>A</td>
<td>124</td>
<td>2</td>
<td>112</td>
<td>4.3</td>
<td>35</td>
<td>53</td>
<td>2100</td>
</tr>
<tr>
<td>N</td>
<td>122</td>
<td>1</td>
<td>110</td>
<td>4.1</td>
<td>34</td>
<td>50</td>
<td>2160</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>A</td>
<td>130</td>
<td>7</td>
<td>123</td>
<td>5.1</td>
<td>40</td>
<td>66</td>
<td>1920</td>
</tr>
<tr>
<td>N</td>
<td>130</td>
<td>3</td>
<td>105</td>
<td>3.9</td>
<td>30</td>
<td>43</td>
<td>2150</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>A</td>
<td>130</td>
<td>18</td>
<td>107</td>
<td>6.3</td>
<td>48</td>
<td>69</td>
<td>1780</td>
</tr>
<tr>
<td>N</td>
<td>130</td>
<td>7</td>
<td>83</td>
<td>4.9</td>
<td>38</td>
<td>44</td>
<td>1360</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>A</td>
<td>138</td>
<td>13</td>
<td>122</td>
<td>4.4</td>
<td>32</td>
<td>53</td>
<td>2230</td>
</tr>
<tr>
<td>N</td>
<td>138</td>
<td>4</td>
<td>92</td>
<td>3.5</td>
<td>25</td>
<td>31</td>
<td>2110</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>A</td>
<td>93</td>
<td>33</td>
<td>103</td>
<td>4.5</td>
<td>48</td>
<td>67</td>
<td>1830</td>
</tr>
<tr>
<td>N</td>
<td>85</td>
<td>2</td>
<td>80</td>
<td>3.4</td>
<td>40</td>
<td>44</td>
<td>1870</td>
<td></td>
</tr>
</tbody>
</table>

Mean

| A  | 118 ± 8| 14 ± 4*| 112 ± 3*| 4.8 ± 0.3*| 42 ± 3*| 62 ± 3*| 1960 ± 70|
| N  | 117 ± 8| 4 ± 1* | 94 ± 5* | 4.0 ± 0.2*| 35 ± 3*| 45 ± 3*| 1900 ± 120|

* Significant differences.

Abbreviations: A = during angina pectoris; N = following nitroglycerin—chest pain no longer present; FA = mean femoral artery pressure in mm Hg; SW = stroke work in g-m; SVR = systemic vascular resistance in dynes sec cm⁻². Pressures are in millimeters of mercury; CO in liters per minute; SV in milliliters.
exercise or pacing-induced angina or when it occurs spontaneously.\textsuperscript{2–17} Pain is not an invariable accompaniment of a rise in LVEDP and arterial pressure in patients with coronary artery disease, as during exercise and after angiotensin infusions large increases in both may occur with no associated symptoms.\textsuperscript{2, 23, 26} It appears that the mechanisms producing the chest pain do not necessarily require changes in intravascular pressure for their effect.

The hemodynamic and clinical findings in angina pectoris have been explained on the basis of left ventricular failure.\textsuperscript{3} Indeed, when these patients are exercised, they may respond abnormally even in the absence of chest pain when compared to normal subjects.\textsuperscript{8, 11, 14, 26} Following the stress of an increase in afterload, which rarely induces pain, a significant depression in myocardial reserve has also been noted.\textsuperscript{23} However, the increase in LVEDP in this and other studies is not invariably associated with alterations commonly ascribed to heart failure.\textsuperscript{12} In fact, the cardiac output and mean systolic ejection rate may increase; even the left ventricular dp/dt has been elevated in some patients during angina.\textsuperscript{12} This implies not heart failure, but a change in ventricular compliance during myocardial ischemia.

Diastolic compliance is one of the fundamental determinants of end-diastolic pressure when one considers the pressure-volume relation of a particular ventricle under study. Normally, at any given compliance level, large changes in end-diastolic volume will be accompanied by only small changes in end-diastolic pressure.\textsuperscript{31} A decrease in myocardial compliance implies a large end-diastolic pressure change associated with relatively minor changes in end-diastolic volume. In this group of patients, four of the five with a rise in LVEDP had normal-sized or only slightly enlarged left ventricles, and one had moderate cardiomegaly. Most of them should have been able to accept significant changes in volume with no change, or only small changes, in LVEDP. Therefore, although we cannot be certain, since no volume studies are available on these patients, the hemodynamic alterations during angina suggest a decrease in myocardial compliance.

Some of these hemodynamic alterations are also suggestive of an increase in sympathetic nervous system activity.\textsuperscript{5, 9, 15, 17} and it has been shown that a definite increase in catecholamine excretion may be present during angina and follow a myocardial infarction.\textsuperscript{52} This does not mean that the sympathetic nervous system-like effects are necessary for the production of angina in all patients, but they may be important in certain situations. Certainly, emotional upsets, which may be associated with a catecholamine discharge, can precipitate angina pectoris, and a similar reflex mechanism may explain angina induced by cold weather. Intracardiac reflexes may also occasionally be operative in spontaneously developing angina and in exertional angina, since heart rate and blood pressure changes usually occur prior to the onset of chest pain. However, the chest pain itself may trigger the autonomic nervous system-like effect in other patients.

Certain patients with chest pain and electrocardiographic abnormalities have no evidence of any change compatible with altered myocardial compliance, left heart failure, or enhanced sympathetic nervous system activity. Since LVEDP changes are related to many factors\textsuperscript{31} these subjects may, of course, have the same basic mechanisms operative but fail to demonstrate gross hemodynamic abnormalities, except occasionally for an increase in cardiac output. Alternatively, the sympathetic nervous system, for some unknown reason, may have less influence on these patients.

A summary of the intravascular dynamics during myocardial ischemia may be stated as follows. The basic myocardial defect is a disparity between the myocardial oxygen needs and oxygen supply, and this defect can be produced by numerous mechanisms. The hemodynamic response to this inadequate coronary blood flow varies significantly from one patient to another. Changes suggesting altered myocardial compliance and enhanced sympathetic nervous system activity may be noted. However, no hemodynamic alterations may be produced at all.
We believe that the initial response to this ischemia is compatible with a decrease in myocardial compliance which, due to the many influences determining the level of LVEDP, may or may not be noted as a major hemodynamic change. Since myocardial function in patients with CAD may be quite abnormal even when chest pain is absent, it is to be anticipated that overt ischemia would make this function worse. Therefore, if the stress to which the myocardium is exposed during angina is excessive, myocardial failure might also ensue. These hemodynamic changes would then be superimposed upon those initially due to ischemia alone. Although initial hemodynamic changes might indicate no evidence of myocardial failure, as in this study, subsequently, hemodynamic and clinical evidence of poor myocardial function may be noted. The patients with changes only in compliance, not reflected by alterations in LVEDP, would demonstrate no consistent hemodynamic abnormalities. In this small group of patients, there was no difference in the extent of coronary artery disease, or in clinical characteristics between those who manifested an increase in LVEDP and those who did not.

The relief of chest pain, subsequent to the sublingual administration of nitroglycerin, was associated with distinct hemodynamic changes in the patients whose heart rate was maintained constant at their angina-precipitating heart rate. In each, there was a decrease in left ventricular end-diastolic pressure, cardiac output, stroke volume, systemic arterial pressure, and the calculated left ventricular stroke work. The changes in pressure preceded the alleviation of pain, and it was possible to predict the relief of pain by monitoring the intravascular pressures.

The mechanism of action of nitroglycerin was initially thought to be secondary to dilatation of the coronary vessels, with a consequent increase in coronary blood flow. Although this dilatation is evident angiographically, most studies do not indicate an increase in myocardial blood flow, either in normal subjects or in those with coronary artery disease. There may be some increase in flow to ischemic areas of the myocardium without any rise in total blood flow, however, and there is some recent evidence to suggest that blood flow through myocardial collaterals is enhanced by nitroglycerin. However, since no definite increase in myocardial oxygen supply has been demonstrated, attention has been directed to other possible modes of action. These investigations have shown that it is possible to explain the beneficial effects of nitroglycerin through its actions on the peripheral circulation, especially the venous system. Pooling of blood in the veins, as well as arteriolar dilatation, results in a decreased venous return, a reduction in end-systolic and end-diastolic ventricular dimensions, a reduction in cardiac output and stroke volume, and a lowering of arterial pressure. The heart rate increases as a compensatory mechanism, and no consistent changes have been noted in the peripheral vascular resistance. The over-all work of the heart is reduced. The net effect is, therefore, a reduc-

![Figure 8](https://circ.ahajournals.org/doi/10.1161/01.CIR.40.4.490)
tion in the external cardiac work and intramyocardial tension and thereby a decrease in myocardial oxygen needs, which can then be met by the compromised coronary circulation.4, 16, 33, 36-42

The findings in this study are consistent with this concept. Figure 8 represents the time course of the hemodynamic changes induced by atrial pacing, the induction of angina pectoris, and its subsequent alleviation by nitroglycerin in one of the patients. Angina was produced by augmenting myocardial work and thereby oxygen requirements through rapid atrial pacing. Despite a maintenance of the augmented heart rate, angina was consistently eliminated by a reduction in cardiac work through the actions of nitroglycerin, which was given at the time noted by the arrow in figure 8. It was only after these hemodynamic changes that the chest pain was relieved, indicative of a dependence upon these peripheral actions for the effectiveness of nitroglycerin.

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