Correlation of Mean Pulmonary Artery Wedge Pressure, Left Atrial Dimension, and PTF-V₁ in Patients with Acute Myocardial Infarction

JOAN ORLANDO, M.D., MICHAEL DEL VICARIO, M.D., WILBERT S. ARONOW, M.D., AND JOHN CASSIDY, M.D.

SUMMARY The mean pulmonary artery wedge pressure (PAWP), left atrial dimension (LAD) by echocardiography, and PTF-V₁ in the electrocardiogram were correlated with each other in 16 patients with acute myocardial infarction in the control period and after therapeutic intervention with either Dextran or furosemide and/or nitroprusside. No significant correlation was found between a normal control PAWP and the LAD. An increased control PAWP correlated well with an increased LAD (r = 0.98). No significant correlation was found between the LAD and the PAWP whether normal or elevated after therapeutic intervention. No significant correlation was found between the PAWP whether normal or elevated and the PTF-V₁. No significant correlation was found between the LAD and the PTF-V₁. We conclude in acute myocardial infarction 1) the PTF-V₁ is not useful in assessing PAWP before or after therapeutic intervention, 2) the LAD correlates poorly with a normal control PAWP but correlates well with an elevated control PAWP, and 3) the LAD cannot be used to assess PAWP after therapeutic intervention.

MORRIS AND ASSOCIATES found a positive correlation between abnormalities in the terminal portion of the P wave of the electrocardiogram in lead V₁ and evidence of pressure or volume overload in patients with valvular heart disease.1 Abnormalities in the terminal portion of the P wave in lead V₁ were also reported in separate studies in patients with pulmonary edema2 and in patients with acute myocardial infarction presenting with clinical and radiographic evidence of congestive heart failure.3,4 These electrocardiographic abnormalities tended to regress with clinical improvement, suggesting that an abnormal terminal negativity of the P wave in lead V₁ was the result of left atrial hypertension.3,4

In a more recent study of patients with acute myocardial infarction, a direct correlation was found between the mean left atrial (LAm) pressure, as estimated by the pulmonary artery end-diastolic pressure (PAEDP) or mean pulmonary artery wedge pressure (PAWP) and the P wave terminal force in V₁ (PTF-V₁) in the standard 12-lead electrocardiogram.5 In this study, an abnormal PTF-V₁, defined as greater than −0.03 mm-sec negativity, occurred in patients with initially elevated LAm pressure and regressed following reduction of the LAm pressure to normal.

In spite of these reports, it remains unclear whether the electrocardiogram is sufficiently sensitive to detect rapid fluctuations in left atrial pressure which may occur in the setting of an acute myocardial infarction and whether in patients with acute left atrial hypertension, left atrial dilatation plays a role in the genesis of the abnormalities in PTF-V₁.

This study was undertaken to answer these questions in a group of patients with acute myocardial infarction by comparing the LAm pressure estimated by the PAWP to the PTF-V₁ and to the size of the left atrium as measured by echocardiography during a control period and after interventions designed to acutely alter the LAm pressure.

Materials and Methods

Study subjects were 16 patients with acute myocardial infarction documented by clinical history, by electrocardiography, and by elevated serum enzymes. The mean age of these patients was 59.7 years. The patients had elevation of their serum creatine phosphokinase, serum glutamic oxaloacetic transaminase, and serum lactic dehydrogenase enzymes compatible with acute myocardial infarction. Fourteen of the 16 patients had electrocardiographic evidence of evolving transmural myocardial infarction. Two of the 16 patients had electrocardiographic evidence of evolving subendocardial myocardial infarction. These two patients also had a strongly positive technetium 99m stannous pyrophosphate myocardial scintigram. Eleven of the 16 patients were studied within one day, four of the 16 patients within two days, and one of the 16 patients within three days of infarction.

Criteria for selection of patients were the presence of a regular sinus rhythm, the absence of pulmonary disease, and the ability to record rapidly a technically satisfactory left atrial echocardiogram. All study patients were undergoing routine hemodynamic monitoring of their PAWP with a number 7 flow-directed Swan-Ganz catheter. Study patients were chosen sequentially. Twenty-five patients were screened initially. All patients in this study signed an informed research consent form.

The control PAWP was measured with a Statham model P23 Db catheter tip pressure transducer and recorded with an Electronics for Medicine VR-6 simultrace recorder with a direct writer. The zero reference level for pressure recording was 5 cm below the sternal angle. Mean pressures were obtained by electronic integration. The criteria for a satisfactory pulmonary artery wedge pressure were 1) a change from the typical pulmonary artery pressure waveform to the typical pulmonary artery wedge pressure waveform upon inflation of the Swan-Ganz balloon catheter and 2) a mean pressure step-up upon deflation of the Swan-Ganz balloon catheter. The pulmonary artery wedge pressure was characterized by distinct and v waveforms, with the v wave occurring after the t wave of the electrocardiogram. The PAWP and the pulmonary artery end-diastolic pressures were equal.

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A control 12-lead electrocardiogram was recorded at a paper speed of 25 mm/sec and a sensitivity of 1 mV/cm on a Hewlett-Packard model 1511B ECG machine. In addition, to facilitate P wave measurements, lead V1 was recorded at a paper speed of 50 mm/sec and a sensitivity of 2 mV/cm. P waves in V1 were measured by the method of Morris and associates using a magnifying lens. The PTF-V1 was obtained from the product of the depth of the terminal deflection and its duration in lead V1. PTF-V1 values were considered abnormal when the PTF-V1 was -0.04 mm-sec or more negative.

Echocardiography was performed in all patients with an Ekoline-20 ultrasound unit and a Honeywell strip chart recorder. All echocardiograms were recorded with the patients in a slight left lateral decubitus position with 10° to 20° elevation of the head. The transducer was placed in the third, fourth, or fifth left sternal interspace. On recognition of the characteristic mitral valve echoes, the transducer was angled medially, posteriorly, and superiorly to obtain a recording of the aortic root and left atrium. Measurements of left atrial dimension were made directly posterior to the posterior wall at ventricular end diastole between the external surface of the posterior aortic root and the internal surface of the left atrial wall. In our tracings, measurements of the left atrial dimension (LAD) at end diastole were more accurately defined than at end systole. The range of normal for LAD at end diastole is from 7 to 30 mm.

After the control measurements, the 11 patients with a PAWP of 10 mm Hg or less received a low molecular weight dextran (Dextran 40) intravenous infusion over a 10 min period until the PAWP was increased to at least twice the control value or until 500 cc had been infused. The PAWP was monitored at frequent intervals during the infusion, and was not allowed to exceed 20 mm Hg. The five subjects whose control PAWP exceeded 15 mm Hg were treated with intravenous furosemide and/or nitroprusside to reduce the PAWP to below 15 mm Hg.

Upon reaching the desired PAWP changes, the PAWP was recorded and a 12-lead ECG and an echocardiogram were then obtained in rapid sequence as described for the control period. Care was taken to record the left atrial echocardiogram from the same interspace with the same gain setting as for the control tracing. In all cases, the left atrium and aortic root echocardiograms were comparable in appearance to those obtained before the intervention.

### Results

Table 1 indicates the individual values and the means ± 1 SD in the control period and after Dextran infusion for the PAWP, the LAD by echocardiography, and the PTF-V1 in each of the 11 patients with acute myocardial infarction and a normal control PAWP.

Table 2 indicates the individual values and the means ± 1 SD in the control period and after intravenous furosemide and/or nitroprusside for the PAWP, the LAD by echocardiography, and the PTF-V1 in each of the five patients with acute myocardial infarction and an elevated control PAWP.

Table 3 indicates the mean values ± 1 SD in the control period for the PAWP, the LAD by echocardiography, and the PTF-V1 in the entire group of 16 patients with acute myocardial infarction.

For the entire group of 16 patients, the association between the control PAWP and the control LAD was \( r = 0.63 \); the association between the control PAWP and the control PTF-V1 was \( r = 0.24 \); the association between the control PTF-V1 and the control LAD was \( r = 0.21 \).

For the 11 patients with a normal control PAWP, the association between the control PAWP and the control LAD was \( r = 0.33 \); the association between the control PAWP and the control PTF-V1 was \( r = 0.40 \); the association between the control PTF-V1 and the control LAD was \( r = 0.39 \).

For the five patients with an elevated control PAWP, the correlation between the control PAWP and the control LAD was \( r = 0.98 \); the association between the control PAWP

### Table 1. Mean Pulmonary Artery Wedge Pressure, Left Atrial Dimension, and PTF-V1 in the Control Period and after Dextran in 11 Patients with Acute Myocardial Infarction and a Normal Control Mean Pulmonary Artery Wedge Pressure

<table>
<thead>
<tr>
<th>Pt</th>
<th>PAWP (mm Hg)</th>
<th>LAD (cm)</th>
<th>PTF-V1 (mm-sec)</th>
<th>PAWP (mm Hg)</th>
<th>LAD (cm)</th>
<th>PTF-V1 (mm-sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>6</td>
<td>2.5</td>
<td>-0.014</td>
<td>13</td>
<td>2.5</td>
<td>-0.021</td>
</tr>
<tr>
<td>2</td>
<td>9</td>
<td>1.9</td>
<td>-0.002</td>
<td>14</td>
<td>2.3</td>
<td>-0.005</td>
</tr>
<tr>
<td>3</td>
<td>6</td>
<td>2.5</td>
<td>-0.028</td>
<td>12</td>
<td>2.9</td>
<td>-0.045</td>
</tr>
<tr>
<td>4</td>
<td>6</td>
<td>3.2</td>
<td>-0.028</td>
<td>13</td>
<td>3.5</td>
<td>-0.020</td>
</tr>
<tr>
<td>5</td>
<td>4</td>
<td>2.8</td>
<td>-0.014</td>
<td>12</td>
<td>3.0</td>
<td>-0.020</td>
</tr>
<tr>
<td>6</td>
<td>5</td>
<td>2.1</td>
<td>-0.033</td>
<td>10</td>
<td>2.6</td>
<td>-0.054</td>
</tr>
<tr>
<td>7</td>
<td>8</td>
<td>2.6</td>
<td>-0.020</td>
<td>18</td>
<td>3.1</td>
<td>-0.028</td>
</tr>
<tr>
<td>8</td>
<td>10</td>
<td>1.9</td>
<td>-0.018</td>
<td>20</td>
<td>2.1</td>
<td>-0.030</td>
</tr>
<tr>
<td>9</td>
<td>6</td>
<td>3.0</td>
<td>-0.030</td>
<td>15</td>
<td>3.0</td>
<td>-0.041</td>
</tr>
<tr>
<td>10</td>
<td>5</td>
<td>1.7</td>
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<td>13</td>
<td>1.7</td>
<td>-0.015</td>
</tr>
<tr>
<td>11</td>
<td>6</td>
<td>3.5</td>
<td>-0.023</td>
<td>10</td>
<td>3.6</td>
<td>-0.009</td>
</tr>
<tr>
<td>Mean</td>
<td>6.5</td>
<td>2.52</td>
<td>-0.020</td>
<td>13.6</td>
<td>2.75</td>
<td>-0.026</td>
</tr>
<tr>
<td>± 1 SD</td>
<td>±1.8</td>
<td>±0.58</td>
<td>±0.008</td>
<td>±3.1</td>
<td>±0.58</td>
<td>±0.015</td>
</tr>
</tbody>
</table>

### Table 2. Mean Pulmonary Artery Wedge Pressure, Left Atrial Dimension, and PTF-V1 in the Control Period and after Furosemide and/or Nitroprusside in Five Patients with Acute Myocardial Infarction and Elevated Control Mean Pulmonary Artery Wedge Pressure

<table>
<thead>
<tr>
<th>Pt</th>
<th>PAWP (mm Hg)</th>
<th>LAD (cm)</th>
<th>PTF-V1 (mm-sec)</th>
<th>PAWP (mm Hg)</th>
<th>LAD (cm)</th>
<th>PTF-V1 (mm-sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>25</td>
<td>3.8</td>
<td>-0.078</td>
<td>8</td>
<td>3.5</td>
<td>-0.043</td>
</tr>
<tr>
<td>2</td>
<td>36</td>
<td>3.5</td>
<td>-0.057</td>
<td>14</td>
<td>3.3</td>
<td>-0.036</td>
</tr>
<tr>
<td>3</td>
<td>26</td>
<td>3.7</td>
<td>+0.034</td>
<td>6</td>
<td>3.1</td>
<td>+0.021</td>
</tr>
<tr>
<td>4</td>
<td>16</td>
<td>4.0</td>
<td>-0.024</td>
<td>5</td>
<td>2.5</td>
<td>-0.023</td>
</tr>
<tr>
<td>5</td>
<td>22</td>
<td>3.9</td>
<td>-0.025</td>
<td>13</td>
<td>3.2</td>
<td>-0.016</td>
</tr>
<tr>
<td>Mean</td>
<td>25.0</td>
<td>3.78</td>
<td>-0.030</td>
<td>9.2</td>
<td>3.12</td>
<td>-0.019</td>
</tr>
<tr>
<td>± 1 SD</td>
<td>±7.3</td>
<td>±0.19</td>
<td>±0.042</td>
<td>±4.1</td>
<td>±0.38</td>
<td>±0.024</td>
</tr>
</tbody>
</table>

### Table 3. Mean Control Pulmonary Artery Wedge Pressure, Left Atrial Dimension, and PTF-V1 in the Entire Group of 16 Patients with Acute Myocardial Infarction

<table>
<thead>
<tr>
<th>PAWP (mm Hg)</th>
<th>LAD (cm)</th>
<th>PTF-V1 (mm-sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>12.25</td>
<td>2.91</td>
</tr>
<tr>
<td>± 1 SD</td>
<td>±9.75</td>
<td>±0.77</td>
</tr>
<tr>
<td>±0.023</td>
<td>±0.023</td>
<td>±0.023</td>
</tr>
</tbody>
</table>
and the control PTF-Vr was \( r = 0.24 \); the association between the control PTF-Vr and the control LAD was \( r = 0.10 \).

After therapeutic intervention, no significant correlation was found between the PAWP or PAEDP and the LAD or the PTF-Vr. After therapeutic intervention, no significant correlation was found between the PTF-Vr and the LAD.

**Discussion**

The frequent determination of left ventricular filling pressure as estimated by the PAWP or PAEDP is a well-established method of evaluating left ventricular function in acute myocardial infarction. Noninvasive methods of predicting impending power failure have included bedside observations of the onset and disappearance of gallop rhythms, of pulmonary rales, and of radiologic alterations consistent with pulmonary congestion. These noninvasive methods are useful, but are relatively insensitive for assessing left ventricular function in the setting of the rapidly changing hemodynamics in acute myocardial infarction. Some investigators have reported that the P wave in the standard 12-lead ECG and, in particular, the atrial vector in lead V1, fluctuates with the severity of left ventricular failure and is a valuable adjunct to the hemodynamics in following left ventricular function in acute myocardial infarction. Chandraratna and Hodges found that the PTF-Vr in the standard 12-lead ECG correctly identified whether the LAm pressure was normal or abnormal at admission in 25 of 30 patients with acute myocardial infarction. Simultaneous measurements of PTF-Vr and LAm pressure on subsequent days resulted in discordant changes in only seven of the 56 measurements made. In another series of patients with acute myocardial infarction, Heikkinen and associates reported a close correlation between PAWP or PAEDP and its serial changes and the terminal forces of the P wave in lead V1, P terminal forces more negative than \(-0.03 \) mm-Hg were always seen when the mean pulmonary capillary wedge pressure exceeded 12 mm Hg.

On the other hand, Rubler and co-workers found no correlation between PAWP and PTF-Vr in a group of patients undergoing cardiac catheterization. Moreover, Kasser and Kennedy reported that the PTF-Vr correlated more significantly with left atrial volume than with left atrial pressure.

Left atrial dimension can be readily and accurately measured by echocardiography. To our knowledge, the relationship between a rapidly changing PAWP and LAD has not been previously investigated. Our intent was to determine whether measurements of LAD or of PTF-Vr could accurately predict changes in PAWP in patients with acute myocardial infarction. In addition, we attempted to evaluate the role of left atrial enlargement in the development of the echocardiographic abnormalities in PTF-Vr attributed to left atrial hypertension.

As shown in table 1, PTF-Vr was normal in the control period in all patients with a normal control PAWP and became abnormal after Dextran infusion in only three of 11 patients (27%). The LAD was abnormal in the control period in two of 11 patients (18%) and became abnormal after Dextran infusion in only one additional patient (9%). Hemodynamic changes after Dextran infusion other than the elevation of PAWP were unlikely to influence the observed results.

Table 2 shows that two of the five patients with an elevated control PAWP had an abnormal PTF-Vr. After furosemide and/or nitroprusside intervention, the PTF-Vr remained abnormal in one of these two patients. The control left atrial dimension was abnormal in all five patients with an elevated control PAWP and remained abnormal in four of these five patients after furosemide and/or nitroprusside intervention. Hemodynamic changes after furosemide and/or nitroprusside intervention other than the lowering of PAWP were unlikely to influence the observed results.

The alterations in PTF-Vr reported to occur with left atrial hypertension may result from a complex interplay of 1) myocardial pathology which interferes with intra-atrial conduction, 2) duration of left atrial hypertension, and 3) severity of left atrial distention. It seems reasonable, therefore, that transient alterations in PAWP may not be reflected in significant changes in the PTF-Vr or in the LAD.

In conclusion, our data indicate that in acute myocardial infarction, PTF-Vr is not useful in assessing PAWP before or after therapeutic interventions, and that the LAD correlated poorly with a normal control PAWP, but correlated well with an elevated control PAWP. However, the LAD cannot be used to quantitate the PAWP before intervention, and cannot be used to assess the PAWP after intervention.

**Acknowledgment**

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**References**


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