Relationship of Pulmonary Artery to Left Ventricular Diastolic Pressures in Acute Myocardial Infarction

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SUMMARY
We have measured pulmonary artery (PA) and left ventricular diastolic pressures (LVDP) in patients with acute myocardial infarction to establish the relationships of PA pressure to LVDP. Paired determinations for the various parameters showed (mean difference in mm Hg): left ventricular end-diastolic pressure (LVEDP)—LVDP pre-a + 7.9, P < 0.001; LVEDP—mean PA wedge + 6.0, P < 0.001; mean PA wedge—LVDP pre-a, + 0.8, P > 0.2; PA end-diastolic pressure (PAEDP)—mean PA wedge (in all patients) + 3.3, P < 0.001; PAEDP—mean PA wedge (patients with pulmonary vascular resistance ≤ 2 units) + 1.3, P < 0.1; LVEDP—PAEDP + 4.7, P < 0.001; and LVEDP—mean PA — 2.0, P < 0.02. The relationship of LVEDP to mean PA wedge was: LVEDP (y) = 1.12 mean PA wedge (x) + 4.69; Sy.x = 3.42; r = 0.92.

After acute myocardial infarction, PA pressures did not accurately reflect LVEDP because atrial contraction made a large contribution to ventricular filling pressure. In addition, PAEDPs were not the same as mean PA wedge pressures because of some increase of pulmonary vascular resistance in many patients. Thus, PA pressures only provided reliable information about the level of pulmonary venous pressure. LVDP pre-a correlated well with mean PA wedge pressure, and therefore measurement of LVDP (pre-a and EDP) yielded information not only about pulmonary edema, but also about LV performance.

Additional Indexing Words:
Left ventricular end-diastolic pressure
Pulmonary artery diastolic pressure
Left ventricular diastolic pressure pre-a wave
Pulmonary edema

Left ventricular performance
Left atrial pressure
Pulmonary artery wedge pressure

IT IS IMPORTANT to assess objectively the degree of left ventricular (LV) functional impairment, and to detect early progressive deterioration of LV function in patients with acute myocardial infarction. Left ventricular end-diastolic pressure (LVEDP) is an indicator of LV function. LVEDP equals mean left atrial pressure (MLAP),¹ and at end-diastole

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pressures in the pulmonary artery (PA), left atrium (LA), and LV are equal \(1^2-5\) in people with normal LV function and normal pulmonary vascular resistance. The availability of the Swan-Ganz catheter \(6\) makes it possible to record PA and PA wedge pressures easily in most acutely ill patients. If PA pressure provided an accurate and reliable estimate of LVEDP in such patients, then a parameter of LV performance could be monitored continuously. However, it has been shown that in patients with chronic LV disease, MLAP and PAEDP are not equal to LVEDP,\(3,4,7\) Therefore, the present study was undertaken to examine the relationships between PA, PA wedge, and LV diastolic pressures in patients with acute myocardial infarction.

**Methods**

Hemodynamic studies were carried out in patients who had recently sustained an acute myocardial infarction. The relationships of the values of pulmonary artery (PA) and pulmonary artery wedge (PA wedge) pressures to left ventricular diastolic pressures (LVEDP) in 64 patients, in whom simultaneous pressures at adequate paper speeds were available, have been analyzed in detail for this report.

Fifty-four patients were studied in the Coronary Care Unit. Fifty of these patients were studied within the first 3 days after acute myocardial infarction. Their mean age was 57 years (range 37–81 years). Thirty-seven patients were considered to have a transmural acute myocardial infarction. Studies were performed using previously described technics\(5\). Twelve of these patients were restudied a few weeks later in the cardiac catheterization laboratory.

Twenty-two patients were studied 3–12 weeks after acute myocardial infarction in the diagnostic cardiac catheterization laboratory. Their average age was 61.2 years (range 51–79 years). Sixteen patients had a transmural acute myocardial infarction. The technics used for the studies have been described in detail previously.\(9\)

All patients were in sinus rhythm. The pressures from the right and left sides of the heart were recorded simultaneously using two identical calibrated Statham P23Db strain gauges. The right heart pressures were recorded using Goodale Lubin or Swan-Ganz catheters,\(6\) and the left heart pressures were recorded using no. 5 Teflon or no. 7 NIH catheters. Left ventricular end-diastolic pressures were measured at the Z point,\(1\) identified as a change in the velocity of upstroke of the pressure tracing. It occurs after atrial contraction, at an average time of 0.05 sec after onset of the QRS of the simultaneously recorded electrocardiogram.\(10\) LVEDP pre-a wave was measured just prior to the increase of LVDP that occurs with atrial contraction. Pulmonary artery end-diastolic pressure (PAEDP) was measured at the lowest point of its tracing. Six to twelve consecutive beats were analyzed, and the mean value was used. Mean pulmonary artery (PA) and mean pulmonary artery wedge (mean PA wedge) pressures were obtained by electronic integration, except in a few patients where they were determined by planimetry. Pulmonary vascular resistance (PVR), i.e. pulmonary arterial resistance (Rpa), was calculated, in units, as: PA–mean PA wedge pressure, in mm Hg/cardiac output, in liters/min. For patients in whom PA wedge pressure was not recorded, LVDP pre-a wave was used instead because we have found no significant difference between LVDP pre-a and mean PA wedge pressures in the present group of patients (vide infra).

**Results**

Not all parameters were measured in each patient. Therefore, paired determinations for the different variables have been made (table 1). Figure 1 shows the relationship of LVEDP to LVDP pre-a. The LVEDP averaged 19.8 mm Hg (range 6–42), and the LVDP pre-a averaged 11.9 mm Hg (range 0–27). The difference, which is the atrial contribution to LVEDP, averaged 7.9 mm Hg. When LVEDP exceeded 12 mm Hg, atrial contraction contributed an average of 8.9 mm Hg (range 2–18) to EDP, and when the LVEDP was \(\leq 12\) mm Hg, atrial contraction contributed an average of 4.4 mm Hg (range 1–11) to LVEDP. Therefore, it can be seen that atrial contraction made a large contribution to LVEDP, particularly when LVEDP exceeded 12 mm Hg.

The mean PA wedge pressure was lower than the LVEDP in all instances (fig. 2). However, there was no significant difference between the mean PA wedge pressure and the LVDP pre-a (fig. 2, table 1). The relationship between mean PA wedge to LVEDP was expressed by the following regression equation: \(n = 23; y (LVEDP) = 1.12 x \) (mean PA wedge) \( + 4.69; Sy_x = 3.42; r = 0.9213.\) The a wave of the PA wedge pressure tracing

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Circulation, Volume XLVI, August 1972
Table 1

Data from Patients in whom Paired Values were Available

<table>
<thead>
<tr>
<th>Parameter</th>
<th>No. determinations</th>
<th>Mean (mm Hg)</th>
<th>SEM</th>
<th>Mean diff (mm Hg)</th>
<th>SEM</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 LVEDP</td>
<td>73</td>
<td>19.8</td>
<td>0.99</td>
<td>7.9</td>
<td>0.49</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LVDP pre-a</td>
<td>73</td>
<td>11.9</td>
<td>0.69</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 LVEDP</td>
<td>23</td>
<td>17.5</td>
<td>1.79</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean PA wedge</td>
<td>23</td>
<td>11.4</td>
<td>1.48</td>
<td>6.1</td>
<td>0.71</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>3 Mean PA wedge</td>
<td>23</td>
<td>11.4</td>
<td>1.22</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVDP pre-a</td>
<td>23</td>
<td>10.7</td>
<td>1.48</td>
<td>0.8</td>
<td>0.64</td>
<td>&gt;0.2</td>
</tr>
<tr>
<td>4 All patients (with normal or elevated PVR):</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVEDP</td>
<td>47</td>
<td>18.7</td>
<td>1.16</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PAEDP</td>
<td>47</td>
<td>14.7</td>
<td>0.91</td>
<td>4.3</td>
<td>0.77</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>5 PVR &gt; 2 units</td>
<td>21</td>
<td>18.7</td>
<td>1.89</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVEDP</td>
<td>21</td>
<td>16.5</td>
<td>1.39</td>
<td>2.2</td>
<td>1.68</td>
<td>&gt;0.1</td>
</tr>
<tr>
<td>PAEDP</td>
<td>21</td>
<td>12.8</td>
<td>1.02</td>
<td>6.2</td>
<td>0.80</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>6 PVR ≤ 2 units</td>
<td>27</td>
<td>19.0</td>
<td>1.36</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVEDP</td>
<td>27</td>
<td>12.8</td>
<td>1.02</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7 All patients (with normal or elevated PVR):</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PAEDP</td>
<td>24</td>
<td>15.1</td>
<td>1.32</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean PA wedge</td>
<td>24</td>
<td>11.1</td>
<td>1.42</td>
<td>4.0</td>
<td>0.77</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>8 PVR &gt; 2 units</td>
<td>9</td>
<td>16.8</td>
<td>1.60</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>PAEDP</td>
<td>9</td>
<td>10.1</td>
<td>4.04</td>
<td>6.7</td>
<td>0.83</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Mean PA wedge</td>
<td>9</td>
<td>10.1</td>
<td>4.04</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9 PVR ≤ 2 units</td>
<td>15</td>
<td>13.4</td>
<td>1.62</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PAEDP</td>
<td>15</td>
<td>12.1</td>
<td>2.04</td>
<td>1.3</td>
<td>0.74</td>
<td>&gt;0.1</td>
</tr>
<tr>
<td>Mean PA wedge</td>
<td>15</td>
<td>12.1</td>
<td>2.04</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10 LVEDP</td>
<td>47</td>
<td>18.7</td>
<td>1.16</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean PA</td>
<td>47</td>
<td>20.6</td>
<td>1.11</td>
<td>-1.9</td>
<td>0.75</td>
<td>&lt;0.02</td>
</tr>
</tbody>
</table>

Abbreviations: LVEDP = left ventricular end-diastolic pressure; LVDP = left ventricular diastolic pressure; PA = pulmonary artery; PAEDP = pulmonary artery end-diastolic pressure; PVR = pulmonary vascular resistance.

correlated well with the a wave of the LV diastolic pressure.

The PAEDP was significantly higher than mean PA wedge pressure (table 1, fig. 3). When patients with an elevated pulmonary vascular resistance (>2 units) were excluded, there was no significant difference between PAEDP and mean PA wedge pressures. Even when all patients were included, PAEDP > 15 mm Hg was associated with mean PA wedge pressure > 12 mm Hg in all but one patient, and only one patient with PAEDP ≤ 15 mm Hg had mean PA wedge > 12 mm Hg (i.e. at 13 mm Hg).

PAEDP was lower than an elevated LVEDP (table 1, fig. 4), and the mean of the values for PAEDP was between the averages of the values for mean PA wedge and LVEDP. The regression equation for these two parameters was y (LVEDP) = 0.93 - x (PAEDP) + 5.32; Sy.x = 5.46; r = 0.7314; n = 47. When patients with an elevated pulmonary vascular resistance were excluded, the difference between PAEDP and LVEDP was increased (table 1), and the relationship was y (LVEDP) = 1.07 x (PAEDP) + 5.25; Sy.x = 4.33; r = 0.808; n = 27. Of 19 patients with elevated PAEDP (>15 mm Hg) all had an elevated LVEDP. However, 17 patients with PAEDP ≤ 15 mm Hg also had an elevated LVEDP.

As can be expected, the PA pressure was higher than the LVDP pre-a and mean PA.
wedge pressure. PA was also significantly different from LVEDP (table 1). When patients with elevated PVR were excluded, PA still did not bear a constant relationship to LVEDP. When patients with normal and elevated PVR were considered, all 21 with elevated PA (>20 mm Hg) also had an elevated LVEDP. However, 15 patients with PA <20 mm Hg also had an elevated LVEDP.

Discussion

In the presence of a normal left ventricle (LV), and when there is no obstruction to flow between the pulmonary artery (PA) and LV, PA pressures bear a constant relationship to left atrial pressure (LAP) and LVDP, and at end-diastole, pressures in the PA, LA, and LV are equal. In the presence of normal LV diastolic pressures, LVEDP equals mean LAP (MLAP). In patients with LV dysfunction, an elevated LVEDP helps to maintain the force of ventricular contraction. Should the MLAP rise equiincrementally with increasing LVEDP, the patient is subject to the risk of developing pulmonary edema. Left atrial contraction is capable of elevating LVEDP without a proportionate increase of MLAP.
LV AND PA PRESSURES IN AMI

Figure 2
The relationships of mean pulmonary artery (PA) wedge pressure to left ventricular end-diastolic pressure (LVEDP) (left) and to LV diastolic pressure pre-a wave (right). Mean PA wedge pressure was consistently and significantly lower than LVEDP, and there was no significant difference between mean PA wedge pressure and LV diastolic pressure pre-a wave.

thus dissociating the pulmonary circulation from the consequences of an elevated LVEDP.7,11,12 This function of the LA has been called the “booster pump” action.12

Braunwald and co-workers1 have shown that in normal people the LA pressure prior to atrial contraction (which equals LVDP pre-a since there is no pressure gradient between

Figure 3
The relationship of PAEDP (vertical axis) to mean PA wedge pressure (horizontal axis) in all patients (left and right), and only in patients with pulmonary vascular resistance (PVR) ≤ 2 units (center). There is no significant difference between PAEDP and mean PA wedge pressure only in patients with PVR ≤ 2 units (center). All patients but one with PAEDP > 15 mm Hg had mean PA wedge > 12 mm Hg, and all but one patient with mean PA wedge > 12 mm Hg had PAEDP > 15 mm Hg (right).

Circulation, Volume XLVI, August 1972
LA and LV) averaged 7.1 mm Hg, and LVEDP averaged 8.7. Thus, the mean increase of LVDP as a result of atrial contraction was 1.6 mm Hg. In our patients the mean values for LVDP pre-a and LVEDP were 11.9 and 19.8 mm Hg, respectively. Thus atrial contraction contributed an average of 7.9 mm Hg to LVEDP, a fivefold increase in the contribution of atrial contraction to EDP (7.9 vs 1.6). The contribution of atrial contraction to LVEDP averaged 18.4% (1.6 of 8.7 mm Hg) in normal subjects, and in our patients averaged 39.9% (7.9 of 19.8 mm Hg). Because of this atrial contribution to ventricular filling, mean PA wedge pressures were lower than LVEDPs after acute myocardial infarction in most patients. Thus, the booster pump action of the LA is operative in patients with acute myocardial infarction, and pressures in the pulmonary circulation do not equal LVDP at end-diastole. The regression equation LVEDP = 1.12 mean PA wedge + 4.69, Sy.x = 3.42 expresses the relationship of LVEDP to mean PA wedge, and could be used to predict the LVEDP if the mean PA wedge pressure is known.

Patients with an elevated LVEDP after acute myocardial infarction had either normal or elevated PAEDP. PAEDP was higher than mean PA wedge pressure. However, if patients with PVR > 2 units are excluded, PAEDP was not significantly different from mean PA wedge pressure. Thus, the lack of identity between PAEDP and mean PA wedge pressure was due to minimal-to-moderate increases of PVR. Although Rutherford et al. have found an elevated PA (>20 mm Hg) to be a clinically reliable, objective measure of left ventricular failure, in our patients an elevated LVEDP was associated with either normal or elevated PA.

It could be questioned whether it is important to know the LVEDP in assessing LV performance in patients with acute myocardial infarction. The major determinants of the LV pressure-volume relationships are the initial ventricular volume, chamber geometry, wall thickness, and stiffness. Thus, an elevated LVEDP may be the result of
increased left ventricular end-diastolic volume (LVEDV) and, therefore, increased myocardial fiber length, changes in ventricular compliance, or both. It has been suggested that the increased LVEDP in patients with acute myocardial infarction could be related largely to changes in LV compliance and, therefore, LVEDP may not be an important parameter in assessing ventricular performance. The finding of a normal LVEDV in association with an elevated LVEDP in man and after experimental myocardial infarction would support such a hypothesis.

The diastolic volumes in the studies just quoted were measured only at end-diastole. However, other studies have shown: (1) Atrial contraction can increase LVDP, LVEDV, and LV myocardial length in the experimental animal with a normal LV. Stott and co-workers have measured LV volumes throughout diastole in patients with LV hypertrophy and in a group of control patients. It can be seen from their data that, when patients with LV hypertrophy and an elevated LVEDP are considered, atrial contraction contributed 47% to the LV stroke volume and 33% to LVEDV even though the LVEDV was in the normal range. In the control patients atrial contraction only contributed 26% to the LV stroke volume and 20% to LVEDV. Thus, the study of Stott et al. shows that in the presence of LV dysfunction the left ventricle was less compliant (normal LVEDV, elevated LVDP), and also that atrial contraction made a significant contribution to LVEDV and therefore to myocardial fiber length. It is interesting that the average atrial contribution to LVEDP and LVEDV was similar in controls (18.41 and 20%, respectively) and in patients with LV dysfunction (39.9 and 33%, respectively). (3) In patients with acute myocardial infarction ouabain has been shown to reduce the elevated LVEDP, suggesting that the abnormal LVEDP in these patients was associated with an increased LVEDV. Thus, it is likely that in patients with acute myocardial infarction the elevated LVEDP is probably the result of an increased LVEDV and of altered LV compliance, and that atrial contraction made a significant contribution to LVEDV. Therefore, we feel that LVEDP rather than PAEDP or mean PA wedge pressure is the parameter that should be used in the assessment of ventricular performance.

In acute myocardial infarction, atrial contraction made a large contribution to LVEDP. Therefore, LVEDP and MLAP were not equal and there was no equalization of pressure between PA and LV at end-diastole. In addition, in many patients there were differences between PAEDP and mean PA wedge pressure due to increases in pulmonary vascular resistance. If LV filling pressures are monitored in patients with acute myocardial infarction, the following data are important: (1) Mean PA wedge pressure, to assess the pulmonary venous pressure and thus the risks of patients developing pulmonary edema, and (2) LVEDP, to assess the performance characteristics of the LV. Measurement of PA and PA wedge pressures only involved catheterization of the right side of the heart but they did not provide a reliable estimate of LVEDP. PAEDP, if elevated, usually signified an elevated PA wedge pressure. Although determination of left ventricular diastolic pressures necessitated catheterization of the LV, LVEDP could be directly measured. Since LVDP pre-a correlated well with the mean PA wedge pressure, reliable information about pulmonary venous pressure was also obtained.

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References

3. FALICOV RE, RESNEKOV L: Relationship of the pulmonary artery end-diastolic pressure to the left ventricular end-diastolic and mean filling pressures in patients with and without left ventricular dysfunction. Circulation 42: 65, 1970


5. JENKINS BS, BRANDLEY RD, BRANTHWAITE MA: Evaluation of pulmonary arterial end-diastolic pressure as an indirect estimate of left atrial mean pressure. Circulation 42: 75, 1970


19. STOTT DK, MARFOLE DGF, BRISTOW JD, KLOSTER FE, GRISWOLD HE: The role of left atrial transport in aortic and mitral stenosis. Circulation 41: 1031, 1970


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