Relationship between Pulmonary Artery End-Diastolic Pressure and Left Ventricular Filling Pressure in Patients in Shock

By Melvin Scheinman, M.D., G. Thomas Evans, M.D., Alan Weiss, M.D., and Elliot Rapaport, M.D.

SUMMARY
Bedside catheterization permitted 56 simultaneous measurements of left ventricular end-diastolic pressure (LVEDP) and pulmonary artery end-diastolic pressure (PAEDP) in 25 patients in shock. Measurements were made over a wide range of heart rates (60–160 beats/min), arterial oxygen tension (P_{O2}; 33–562 mm Hg), stroke volumes (9–105 ml), and systolic arterial pressures (65–250 mm Hg), and before and during various therapeutic interventions. There was good correlation between PAEDP and LVEDP (r = +0.85) and PAEDP and left ventricular pre-a pressure (r = +0.82). In 16 patients breathing room air, mean arterial P_{O2} was 58 ± 15 mm Hg and rose significantly to 282 ± 16 mm Hg after breathing oxygen (P < 0.01), but there was no significant change in the correlation between PAEDP and LVEDP before (r = +0.80) or during oxygen breathing (r = +0.80). Similarly, in seven patients treated with inotropic agents there was excellent correlation between PAEDP and LVEDP before (r = +0.96) and during inotropic therapy (r = +0.88). A PAEDP in excess of 15 mm Hg nearly always reflected increased LVEDP while a PAEDP less than 10 mm Hg was always associated with normal LVEDP. Thus in our patients with shock PAEDP represented a good estimate of LVEDP during a variety of therapeutic interventions and therefore represents a useful tool in guiding the therapy of patients in shock.

Additional Indexing Words:
Pulmonary artery pressures Myocardial infarction Left ventricular pressures
Hypoxia Inotropic agents Shock

The left ventricular end-diastolic pressure (LVEDP) constitutes an important index of left ventricular function and is accurately reflected by the pulmonary artery end-diastolic pressure (PAEDP) in subjects with normal ventricular function. However, uncertainty exists concerning use of the PAEDP as a valid index of LVEDP in patients with shock. Our study was designed to analyze the effects of change in arterial oxygen tension (P_{O2}), heart rate, stroke volume, and systemic pressure, induced by various therapeutic agents, on the relationship between PAEDP and LVEDP in these patients.

Materials and Methods
Twenty-five patients in shock were studied. The cause of shock was acute myocardial infarction in 12 patients, sepsis in five, postcardiac arrest in three, hypovolemia in three, drug overdose in one, and cerebrovascular accident in one. Shock was defined on the basis of decreases in systemic pressure as well as by signs of inadequate peripheral blood flow. Most patients showed intravenous systolic pressures below 100 mm Hg, clammy skin, and depressed sensorium. All patients had a urine flow less than 25 ml/min in association with one or more of the latter findings. Patients in shock whose arterial blood pressures were maintained by infusions of catecholamines were also included in the present study, while patients with severe chronic bronchopulmonary or valvular heart disease were excluded.

All hemodynamic measurements were performed without fluoroscopy at the bedside in the Coronary Care Unit. The pulmonary artery was catheterized either by a small nylon catheter inserted through a 17-gauge needle in a medial antecubital vein (eight patients) or by a Swan-Ganz balloon catheter (17 patients). Left ventricular catheterization was achieved with a no. 7 Judkins right coronary catheter (Cordis Corporation), introduced percutaneously by the Seldinger technic into either femoral artery and advanced in retrograde fashion across the aortic valve nonfluoroscopically. In three patients, left ventricular catheterization could not

From the Cardiopulmonary Laboratory and Medical Services, San Francisco General Hospital, and the Department of Medicine and the Cardiovascular Research Institute, University of California, San Francisco.

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Address for reprints: Melvin Scheinman, M.D., Regional Medical Programs, San Francisco General Hospital, 1001 Potrero Avenue, San Francisco, California 94110.

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be achieved via the femoral approach but was accomplished by percutaneous right axillary artery catheterization. The majority of subjects had occasional premature ventricular beats as the arterial catheter was manipulated into the left ventricle. No sustained arrhythmia was encountered during either right or left heart catheterization. The left ventricular catheter was removed in less than 30 min in all but two subjects.

Simultaneous pulmonary artery and left ventricular pressure pulses were recorded by means of Statham P23Db strain gauges with identically calibrated sensitivities on an Electronics-for-Medicine DR8 recorder. The PAEDP was measured at the lowest point of the pulmonary artery pressure tracing. The LVEDP was measured at a point immediately preceding the rapid upstroke of the left ventricular pressure and was identified 0.04–0.06 sec after onset of the QRS complex in all patients.9 "Pre-a" left ventricular pressure was also determined by measurement of left ventricular pressure immediately prior to the atrial contraction wave.

Oxygen consumption was determined with a basal metabolator as described previously from our laboratory10), arterial and mixed venous oxygen contents were measured by the Van Slyke method11 from blood withdrawn from catheters in the left ventricle and pulmonary artery, and cardiac output was determined by the direct Fick method. Arterial pH, arterial oxygen, and carbon dioxide (\(F_{\text{CO}_2}\)) tensions were measured by electrodes and read from a Beckman-160 meter.

In 15 patients, simultaneous measurements of pulmonary artery and left ventricular pressures were obtained while the subjects were breathing room air and repeated after 15 min of breathing a high-oxygen mixture which was delivered via nasal prongs or endotracheal tube. In seven patients heart rate, cardiac output, pulmonary artery and left ventricular pressures were measured before and after administration of various inotropic agents. The agents were infused in increasing amounts depending on clinical response (i.e., increases in systemic pressure, urine flow, improved sensorium, cardiac output), the limiting factors being an undue increase in heart rate or increased ventricular irritability. Four patients were treated with norepinephrine (4–12 \(\mu\)g/min), three with isoproterenol (3–14 \(\mu\)g/min) and one patient in the latter group received 5 mg of glucagon (table 3). Two patients were studied before and after intravenous administration of 80 mg of ethacrynic acid while another subject was studied before and 10 min after administration of a 250-mI volume challenge. The interventions tested were fairly uniformly distributed among the different causes of shock (tables 1, 2, and 3).

The standard Student's t test was used for statistical analyses of paired observations with differences at the 5% level considered significant.

### Results

Over a 14-month period 56 studies were carried out in 25 patients. The pertinent clinical and hemodynamic data are presented in tables 1–3. For the group as a whole, there was no statistically
LVEDP AND PAEDP WITH SHOCK

Table 2
Clinical, Hemodynamic, and Arterial Blood-Gas Measurements in Patients Breathing Room Air and a High Oxygen Concentration

<table>
<thead>
<tr>
<th>Pt (no.)</th>
<th>Diagnosis</th>
<th>LV pre-a (mm Hg)</th>
<th>LVEDP (mm Hg)</th>
<th>PAEDP (mm Hg)</th>
<th>PO2 (mm Hg)</th>
<th>pH units</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Room air</td>
<td>Room air</td>
<td>Room air</td>
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<tr>
<td></td>
<td></td>
<td>+ O2</td>
<td>+ O2</td>
<td>+ O2</td>
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<tr>
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<td>9</td>
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<td>21</td>
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<td>6</td>
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<td>17</td>
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<td>18</td>
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<td>5</td>
<td>11</td>
<td>8</td>
<td>17</td>
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<td>Hypovolemic shock</td>
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<td>5</td>
<td>4</td>
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<td>Postcardiac arrest</td>
<td>6</td>
<td>3</td>
<td>9</td>
<td>5</td>
<td>7</td>
</tr>
<tr>
<td>15</td>
<td>Postcardiac arrest</td>
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<td>4</td>
<td>4</td>
<td>10</td>
<td>14</td>
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<tr>
<td>16</td>
<td>Cerebrovascular accident</td>
<td>3</td>
<td>8</td>
<td>6</td>
<td>12</td>
<td>10</td>
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<tr>
<td>Mean</td>
<td></td>
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<td>11.87</td>
<td>13.25</td>
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<td>10.46</td>
<td>9.01</td>
<td>9.64</td>
<td>9.08</td>
<td>7.9</td>
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</table>

Abbreviations: LV = left ventricular; EDP = end-diastolic pressure; PAEDP = pulmonary artery end-diastolic pressure; PO2 = arterial oxygen tension.

A significant difference between mean PAEDP (±SD) (18.1 ± 9.4 mm Hg) and mean LVEDP (16.8 ± 10.8 mm Hg) or between mean PAEDP and mean left ventricular pre-a pressure (13.8 ± 8.9 mm Hg). There was a significant correlation between PAEDP and LVEDP with a correlation coefficient (r = +0.85) (P < 0.01) (fig. 1A). Similarly, in 46 studies in which left ventricular pre-a waves were recorded, there was a statistically significant correlation between PAEDP and left ventricular pre-a pressure with r = +0.82 (P < 0.01) (fig. 1B). After various interventions, changes in PAEDP correlated significantly (P < 0.01) with a simultaneous change in LVEDP in 27 studies (r = +0.81) (fig. 2A) or with changes in left ventricular pre-a pressure in 22 studies (r = +0.85) (fig. 2B).

In 28 of the 56 studies PAEDP exceeded LVEDP (range 3–9 mm Hg) while LVEDP exceeded PAEDP in 16 studies (range 3–12 mm Hg). In 12 studies the difference between PAEDP and LVEDP was 2 mm or less. The PAEDP exceeded 12 mm Hg while simultaneous LVEDP was less than 12 mm Hg in nine of 56 studies (16%), whereas LVEDP exceeded 12 mm Hg, while simultaneous PAEDP was less than 12 mm Hg in only two of 56 studies (3%). With one exception elevation of PAEDP

Table 3
Hemodynamic Findings in Patients in Shock Who were Treated with Volume Infusions or Diuretics

<table>
<thead>
<tr>
<th>Pt (no.)</th>
<th>Diagnosis</th>
<th>Treatment</th>
<th>LV pre-a (mm Hg)</th>
<th>LVEDP (mm Hg)</th>
<th>PAEDP (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>24</td>
<td>Acute MI</td>
<td>Control</td>
<td>30</td>
<td>43</td>
<td>43</td>
</tr>
<tr>
<td>25</td>
<td>Acute MI</td>
<td>Postethacrynic acid</td>
<td>17</td>
<td>20</td>
<td>33</td>
</tr>
<tr>
<td>26</td>
<td>Cerebrovascular accident</td>
<td>Control</td>
<td>11</td>
<td>14</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td></td>
<td>After 200 cc D5W</td>
<td>11</td>
<td>14</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Postethacrynic acid</td>
<td>14</td>
<td>18</td>
<td>25</td>
</tr>
</tbody>
</table>

Abbreviations: LV = left ventricular; EDP = end-diastolic pressure; PAEDP = pulmonary artery end-diastolic pressure MI = myocardial infarction.

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A good correlation is seen between all comparisons of simultaneously recorded pulmonary end-diastolic pressure (PAEDP) and left ventricular end-diastolic pressure (LVEDP) (left) or between PAEDP and left ventricular pre-a pressure (right).

Figure 1

A good correlation is seen between all comparisons of simultaneously recorded pulmonary end-diastolic pressure (PAEDP) and left ventricular end-diastolic pressure (LVEDP) (left) or between PAEDP and left ventricular pre-a pressure (right).

above 15 mm Hg was associated with an LVEDP greater than 12 mm Hg (patient 12, table 2) while a PAEDP of less than 10 mm Hg was always associated with an LVEDP less than 12 mm Hg.

Heart Rate

For the group as a whole, heart rate varied from 60 to 160 beats/min. There was a better correlation between LVEDP and PAEDP in 25 studies with heart rates equal to or above 100 beats/min (r = +0.80) (fig. 3). However, the PAEDP was not consistently higher than LVEDP in patients with tachycardia.

Hypoxia

In 16 patients, arterial blood Po2, pH, and Pco2 as well as PAEDP and LVEDP were obtained both while patients breathed room air and after at least 15 min of breathing a high inspired oxygen mixture (table 2). Mean arterial Po2 while breathing room air was 58 ± 15.8 mm Hg and rose significantly to 282 ± 164 while breathing oxygen (P < 0.01); there was no significant change in mean arterial Pco2 or pH after administration of oxygen. There was no
statistically significant change in the correlation of mean PAEDP with mean LVEDP both before and during oxygen breathing.

Inotropic Agents

In seven subjects measurements of heart rate, stroke volume, as well as left ventricular (table 1) and pulmonary artery pressures were recorded before and during inotropic therapy. Control mean heart rate averaged 85 ± 13 beats/min and stroke volume 29 ± 6 ml, and rose to 92 ± 24 beats/min and 34 ± 9 ml, respectively, after inotropic therapy, but these changes were not statistically significant. Control left ventricular systolic pressure averaged 88 ± 28 mm Hg and rose significantly to 124 ± 26 mm Hg during inotropic therapy (P < 0.01). There was a significant correlation between PAEDP and LVEDP in the control state (r = +0.92) as well as during administration of inotropic agents (r = +0.86). Change in LVEDP was associated with comparable change in PAEDP after administration of inotropic agents (table 1 and fig. 4). In one patient (no. 22), however, administration of levaterenol bitartrate resulted in large increases in arterial systolic pressure, with an inordinate rise in LVEDP compared with PAEDP.

Changes in Preload

The effects of acute changes in preload using either diuretic agents or volume infusion were assessed in three patients (table 3). Although PAEDP and LVEDP showed similar directional changes after treatment the paucity of data in this group precludes valid statistical evaluation.

Complications

No serious complications occurred during right heart catheterization except for occasional premature ventricular contractions as the catheter traversed the right ventricle. However, two significant complications were encountered after left heart

Figure 3

No significant relationship is noted between heart rate and the gradient between PAEDP and LVEDP.

Figure 4

Comparable rises in PAEDP and LVEDP are seen as afterload was increased after infusion of adrenalin.
catheterization. In one patient (no. 11) with severe liver disease, persistent arterial bleeding with formation of a large femoral hematoma occurred after the study. This complication was thought to be related to grossly depressed procoagulant factors resulting from the severe hepatic disease. One other patient (no. 17) showed loss of arterial pulses in the right upper extremity after right axillary catheterization. No deaths, serious arrhythmias, infections, or clinically manifest systemic emboli resulted from these studies.

Discussion

Assessment of left ventricular filling pressure has been shown to be a useful guide for both treatment and establishing prognosis for critically ill cardiac patients. Continuous left ventricular catheterization is contraindicated because of the unacceptably high incidence of arterial thrombosis and systemic thromboemboli. Pulmonary artery pressures are, however, safely and readily amenable to continuous monitoring, and in patients with normal left ventricular function PAEDP is an accurate reflection of LVEDP, but the validity of using the PAEDP as a measure of left ventricular filling pressure in patients with shock is still a matter of some debate. Previous studies of patients without shock showed excellent correlation between PAEDP and left ventricular pre-a pressure, and LVEDP was consistently higher than PAEDP in those patients with left ventricular dysfunction. Moreover, Bouchard et al. found that increases in systemic pressure induced by administration of methoxamine produced a consistently larger increase in LVEDP than PAEDP; atrial pacing in their studies also resulted in a consistent disparity in pressures with PAEDP exceeding LVEDP at heart rates greater than 124 beats/min.

It must be appreciated that previous studies comparing PAEDP and LVEDP were performed for the most part in patients with left ventricular dysfunction due to chronic myocardial and/or valvular disease. The present studies are unique in that we studied simultaneous measurements of PAEDP and LVEDP in patients with shock. Conceivably, patients with chronic cardiac disease show greater diminution in left ventricular compliance compared with the patients in our study and might therefore be expected to show a greater accentuation of left ventricular pressure resulting from atrial contraction. Large left ventricular "a" waves were not observed in the present study and while they are commonly seen in patients with chronic cardiac disease, these waves are inconsistently reflected in the pulmonary arterial pressure trace. This finding would explain the better correlation of PAEDP with left ventricular pre-a pressure obtained in previous studies. Moreover, in our previous studies we found excellent correlation between PAEDP and LVEDP in acute experiments in dogs in which the marked elevations in LVEDP were induced by increases in either preload (volume infusions) or afterload (constriction of the aorta). Our present study, therefore, suggests that patients in shock may not show similar changes in left ventricular compliance that occurs in patients with chronic myocardial failure or conceivably the shock state results in a shift of ventricular function to a different point on a nonlinear pressure-volume curve. Alternatively, the change in left ventricular pre-a pressure in patients with shock may reflect a diminished force of left atrial contraction and/or critical diminution of atrial stroke volume rather than changes in left ventricular compliance. The small or absent left ventricular "a" waves are most likely due to left ventricular compliance changes and/or diminished strength of left atrial contraction.

Although none of the patients in the present study was treated with methoxamine, we found excellent correlation between PAEDP and LVEDP after institution of a variety of commonly used inotropic agents that significantly elevated systolic arterial pressure. Similarly, although the correlation between PAEDP and LVEDP was poorer for patients with tachycardia, we found no consistent increase in PAEDP with respect to LVEDP in this group. It should be noted that in the present study relatively few observations (seven of 56 studies) were made in patients with heart rates in excess of 124 beats/min (fig. 3).

This study, by design, excluded patients expected to have marked increases in pulmonary vascular resistance. However, many of our patients had mild-to-moderate obstructive pulmonary disease and/or pneumonitis. Even patients with severe hypoxemia had good correlation between PAEDP and LVEDP. It should be emphasized that the vast majority of patients studied before and during oxygen breathing (14 of 16) had an arterial pH that was either normal or alkalotic due either to pretreatment with alkali or to compensatory respiratory alkalosis. These findings suggest that the levels of hypoxemia commonly seen in severely ill...
patients (without accompanying severe acidosis) exert little pulmonary vasoconstrictive effects\textsuperscript{25, 26} and, therefore, for practical purposes do not interfere with the use of PAEDP as a measure of LVEDP.

Certain limitations in the use of PAEDP as a reflection of LVEDP in patients with shock are apparent. For example, in 16% of the studies PAEDP suggested left ventricular failure in the face of a normal LVEDP, and in an additional 3% of studies PAEDP failed to reflect apparent left ventricular decompensation. These false-positive and false-negative reflections of left ventricular failure almost always occurred with PAEDPs in the borderline area between 10 and 15 mm Hg. A PAEDP greater than 15 mm Hg, however, nearly always reflected an abnormal elevation of LVEDP, while a PAEDP less than 10 mm Hg was always associated with normal left ventricular filling pressures. These findings represent useful guides when using the PAEDP as an index of LVEDP in patients with shock.

Certain other limitations in the use of PAEDP should be noted. Patients with severe pulmonary parenchymal disease or mitral stenosis would be expected to show a diastolic gradient in pressure across the pulmonary vascular bed. An additional source of error in the use of PAEDP as a reflection of LVEDP is the variable position of the pulmonary artery catheter. Catheterization of posterior pulmonary branches (in the supine patient) would be expected to result in slightly higher pressures as compared with pressures in anterior vessels. Furthermore, insufficient data in our studies preclude definitive judgment of the effects of severe acidosis or marked tachycardia on the relationship between PAEDP and LVEDP in patients in shock. Finally, in patients treated with mechanical respirators peak inspiration results in a greater increase in pulmonary artery pressures than in left ventricular pressure (fig. 5) presumably because the thicker left ventricular wall is less affected by changes in intrathoracic pressures. Therefore, PAEDP in these patients should be measured either during end expiration or while the ventilator is turned off.

In summary, we found that PAEDP is an excellent general reflection of LVEDP in patients with shock during a wide range of heart rates, cardiac outputs, arterial Pa\textsubscript{O\(_2\)}, and systemic pressures. More significantly, this relationship held for acute changes in LVEDP induced by various therapeutic interventions commonly used in the

\textit{Figure 5}

\textit{Increased intrathoracic pressure with positive-pressure breathing (IPPB) distorts the pulmonary artery trace yielding a diastolic gradient in pressure that is abolished during end-expiratory cycles or when the respirator is turned off.}

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management of critically ill patients. Thus, PAEDP represents a reasonably good reflection of LVEDP and therefore constitutes a useful clinical tool in the management of patients in shock.

References

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MELVIN SCHEINMAN, G. THOMAS EVANS, ALAN WEISS and ELLIOT RAPAPORT

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