Relationship of Pulmonary Artery-Wedge Pressure to Left Atrial Pressure in Man

By Peter C. Luchsinger, M.D., Harry W. Seipp, Jr., and Dali J. Patel, M.D., Ph.D.

The accuracy of the pulmonary artery-wedge pressure as a reflection of the left atrial pressure in man has been studied with conflicting results. Most studies were carried out during the resting state, and no detailed evaluation has been made during various experimental conditions. Yet the pulmonary artery-wedge pressure has been, and still is, extensively used in place of the left atrial pressure to calculate the pulmonary vascular resistance during the control state, as well as drug studies and other experimental conditions. Therefore, it seems important to study the relationship between the pulmonary artery-wedge and the left atrial pressures, particularly during various experimental conditions, in order to evaluate the validity of pulmonary vascular resistance calculations using the pulmonary artery-wedge pressure in human subjects. The present study was undertaken for this purpose. With the transseptal left heart catheterization, it was possible to monitor the left atrial pressure continuously. This pressure was compared to the pulmonary artery-wedge pressure during the control state, during norepinephrine administration, and during positive and negative intra-alveolar pressures.

Methods

In 11 patients with suspected mitral valve disease or no clinically detectable heart disease, right heart catheterization was performed in the usual manner in the supine position. A no. 9 double lumen Courand catheter was used throughout. The distal opening measured the right, mid, or lower lung-wedge pressure, while the proximal opening was lying in the right pulmonary artery. Left atrial pressures were recorded through a needle introduced through the right saphenous vein passing through the interatrial septum. A pulmonary artery-wedge pressure was accepted as satisfactory when the contour of the pressure wave resembled in magnitude and shape the left atrial pressure contour and/or when the aspirated blood had an oxygen tension of 80 mm Hg or above, as measured by the Clark Electrode in the adaptation by Severinghaus. The pressures were recorded on an Electronics for Medicine multichannel recorder using Statham P23ID gauges as transducers. All three gauges (pulmonary artery, wedge, and left atrium) were connected to one bottle with a water level 10 cm. above the spine as reference zero pressure. Mean pressures were obtained electronically. Cardiac output was determined by injecting Cardio-green into the pulmonary artery or superior vena cava and sampling at the brachial artery using a Colson Densitometer and a Texas Recti-Writer for recording.

The patients were studied during a control state, during norepinephrine infusion which raised the brachial artery pressure by 20 to 40 per cent, and during positive and negative intra-alveolar pressures which were read on a water manometer. Variations in alveolar pressures were created by having the patient blow against or apply suction to a "U" tube water manometer. A small leak was created in the tube connecting the manometer to the mouth so that pressures could be maintained only if the glottis remained open. Since the pressure to produce the very small air flow was negligible in relation to the total pressure, the oral pressures could be interpreted as intra-alveolar pressure. Then mean pressures during the fifth to tenth heart beat after onset of the positive or negative intra-alveolar pressures were taken for comparisons.

The pulmonary vascular resistance was calculated in two ways: first using the mean wedge pressure ($R_w$) and then using the mean left atrial pressure ($R_{LA}$), according to the following equations:

$$R_w = \frac{(PA) - (PA_{wedge}) \text{ mm. Hg}}{\text{cardiac output L/min.}}$$

$$R_{LA} = \frac{(PA) - (LA) \text{ mm. Hg}}{\text{cardiac output L/min.}}$$

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Equation 2 is correct by definition, whereas equation 1 is often used to estimate the pulmonary vascular resistance in human studies and has been referred to as "pulmonary arteriolar resistance."

**Results**

The results are tabulated in table 1. During the control phase, three of the 11 patients studied had a slightly elevated pulmonary artery pressure. In all but one patient, there was a pressure drop between the wedged position and the left atrium; accordingly, the calculated pulmonary vascular resistance using the left atrial pressure was higher than that using the pulmonary artery-wedge pressure. The failure of patient W.B.G. to show the same relationships must be due to a technical error not recognized at the time of the study.

The relationship between pulmonary artery-wedge and left atrial pressures during control, norepinephrine infusion, and positive and negative intra-alveolar pressure is shown in figure 2. It is apparent that this relationship is a linear one (regression coefficient $b = 0.86 \pm 0.04$) over the pressure range studied, irrespective of the maneuvers employed to alter the pulmonary hemodynamics.

**Discussion**

The differences between wedge pressure and left atrial pressure were determined in intact human subjects. Great care was taken that all pressures were measured in regard to the same reference point, eliminating any errors which might explain a left atrial pressure higher than the mean pulmonary artery-wedge pressures found in other studies. Norepinephrine was given to alter the pulmonary hemodynamics.

Although the pulmonary artery-wedge pressure is 35 per cent higher than the left atrial pressure, this difference is very consistent under all experimental conditions of this study, as evidenced by the correlation coefficient ($r = 0.95$). It is thus possible to correct a resistance value calculated by use of the pulmonary artery-wedge pressure. Also, the value of the pulmonary vascular resistance calculated in previous studies using the pulmonary artery-wedge pressure could be corrected, if necessary, by using the regression line shown in figure 2, which would permit estimation of the left atrial pressure for a given pulmonary artery-wedge pressure.

A few comments on the difference found between the pulmonary artery pressure and the left atrial pressure would seem appropriate: (1) Since all pressures in our study were measured from the same reference point, any error due to variation in reference point is eliminated. It should be pointed out that such an error could well explain findings wherein the left atrial pressure was higher in some instances than the pulmonary artery-wedge pressure. (2) It has been suggested that the pulmonary artery-wedge pressure is really the pulmonary venous pressure just
### TABLE 1

**Hemodynamic Data During Combined Left and Right Heart Catheterization**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Sex</th>
<th>Weight (Kg.)</th>
<th>Body surface (M²)</th>
<th>Control</th>
<th>BA (mm. Hg)</th>
<th>FA (mm. Hg)</th>
<th>FA-wedge (mm. Hg)</th>
<th>LA (mm. Hg)</th>
<th>Q (L/min.)</th>
<th>PA—FA-wedge (L/min.)</th>
<th>PA—LA (L/min.)</th>
<th>CO (ml/min/m²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>W.B.G.</td>
<td>41</td>
<td>M</td>
<td>67.5</td>
<td>1.86</td>
<td>Control</td>
<td>37.5</td>
<td>7.9</td>
<td>8.7</td>
<td>9.0</td>
<td>4.32</td>
<td>2.66</td>
<td>2.49</td>
<td>9.45</td>
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<tr>
<td>W.B.G.</td>
<td>69</td>
<td>M</td>
<td>69.5</td>
<td>1.86</td>
<td>Control</td>
<td>28.5</td>
<td>17.4</td>
<td>19.1</td>
<td>17.4</td>
<td>3.76</td>
<td>1.73</td>
<td>2.5</td>
<td>11.15</td>
</tr>
<tr>
<td>V.U.</td>
<td>33</td>
<td>M</td>
<td>77.5</td>
<td>2.06</td>
<td>Control</td>
<td>16.6</td>
<td>13.0</td>
<td>12.6</td>
<td>12.6</td>
<td>5.57</td>
<td>0.65</td>
<td>0.72</td>
<td>14.35</td>
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<tr>
<td>V.U.</td>
<td>40</td>
<td>M</td>
<td>59.4</td>
<td>1.72</td>
<td>Control</td>
<td>14.7</td>
<td>8.9</td>
<td>3.6</td>
<td>11.7</td>
<td>6.09</td>
<td>1.02</td>
<td>1.29</td>
<td>16</td>
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<tr>
<td>V.F.M.</td>
<td>40</td>
<td>M</td>
<td>63.5</td>
<td>1.8</td>
<td>Control</td>
<td>15.5</td>
<td>7.1</td>
<td>5.7</td>
<td>11.1</td>
<td>6.11</td>
<td>1.58</td>
<td>1.60</td>
<td>25</td>
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<tr>
<td>P.L.S.</td>
<td>53</td>
<td>M</td>
<td>71.5</td>
<td>1.83</td>
<td>Control</td>
<td>14.4</td>
<td>5.6</td>
<td>4.3</td>
<td>10.8</td>
<td>5.46</td>
<td>1.79</td>
<td>2.62</td>
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</tr>
<tr>
<td>T.W.S.</td>
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<td>M</td>
<td>62.5</td>
<td>1.7</td>
<td>Control</td>
<td>11.7</td>
<td>7.6</td>
<td>5.1</td>
<td>11.3</td>
<td>6.42</td>
<td>0.64</td>
<td>1.03</td>
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<tr>
<td>O.W.</td>
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<td>M</td>
<td>59.5</td>
<td>1.75</td>
<td>Control</td>
<td>12.9</td>
<td>8.9</td>
<td>5.6</td>
<td>12.5</td>
<td>6.64</td>
<td>0.71</td>
<td>1.11</td>
<td>23</td>
</tr>
<tr>
<td>I.V.R.</td>
<td>47</td>
<td>M</td>
<td>63.5</td>
<td>1.74</td>
<td>Control</td>
<td>23.0</td>
<td>14.5</td>
<td>12.8</td>
<td>13.0</td>
<td>5.11</td>
<td>1.66</td>
<td>1.99</td>
<td>32</td>
</tr>
<tr>
<td>J.M.M.</td>
<td>40</td>
<td>M</td>
<td>55.0</td>
<td>1.64</td>
<td>Control</td>
<td>19.0</td>
<td>14.8</td>
<td>10.1</td>
<td>12.9</td>
<td>5.91</td>
<td>0.71</td>
<td>1.51</td>
<td>20</td>
</tr>
</tbody>
</table>

**Legend:**
- **BA** = Mean brachial artery pressure.
- **PA** = Mean pulmonary artery pressure.
- **PA-wedge** = Mean pulmonary artery-wedge pressure.
- **LA** = Mean left atrial pressure.
- **Q** = Pulmonary blood flow.
- **B.S.** = Body surface.
- **Q = Pulmonary blood flow.**
distal to the pulmonary capillaries,¹¹ and the drop in pressure from the pulmonary artery-wedge position to the left atrium is due to the pulmonary venous resistance. This explanation, in general, appears to be correct. However, it should be pointed out that the exact site of pressure measured by the catheter in the wedged position is not known and would depend on vascular anastomosis in the pulmonary vascular bed.

Summary

The pulmonary artery-wedge and left atrial pressures were recorded simultaneously in 11 patients during a control state, during nor-epinephrine infusion, as well as during positive and negative intra-alveolar pressures. A good correlation \( r = 0.95 \) found between the two pressures permits the use of corrected pulmonary artery-wedge pressure in calculation of pulmonary vascular resistance under control and test conditions.

References

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