Inaccuracy of Wedge Pressure as an Index of Pulmonary Capillary Pressure

By Jerome P. Murphy, M.D.

The wedge and left atrial pressures were compared in 12 subjects with congenital and acquired heart disease; all experiments were carried out with closed chest under basal conditions. A poor correlation between the wedge and left atrial pressures was noted in over half the patients. The wedge pressure is especially inaccurate in mitral stenosis with marked elevations in left atrial pressure.

In 1906 MacCullum and his associates observed in dogs that pressure changes in the pulmonary venous system and the left atrium were transmitted to the pulmonary capillary bed and the end arteries of the lung. Subsequent investigators confirmed these findings and further demonstrated that the pulmonary venous and arterial capillary pressures were the same.¹ ² Direct measurements of the venous and arterial sides of the capillary bed in subjects with atrial septal defects showed that changes in venous pressure were quantitatively transmitted to the end arteries.³ These results strongly supported the concept that the pulmonary capillary bed offered little or no resistance to the retrograde transmission of the pulmonary venous pressure to the pulmonary end arteries.⁴ ⁵ Further investigations showed the pulmonary capillary pressure to be slightly higher but otherwise identical to the left atrial pressure.⁶ With the advent and widespread use of right heart catheterization a technic was evolved for measuring the pulmonary capillary pressure by means of the "wedge" method. This technic entails manipulating the cardiac catheter into a small pulmonary end artery so that all transmitted right ventricular pressures are damped completely. Whatever pressure is then recorded represents the pulmonary capillary pressure and transmitted pulsations from the left heart. A basic criterion for insuring a complete wedge is the sampling of fully oxygenated blood when the catheter is in place.

Although certain theoretical objections have been raised to considering the wedge pressure and the pulmonary capillary pressures to be identical, there has been rather widespread acceptance of the basic concept that the wedge pressure is an accurate index of the pulmonary capillary pressure and hence of the pulmonary venous and left atrial pressures. Numerous studies requiring knowledge of the pulmonary capillary pressure as a factor have been carried out with the wedge pressure substituted for the pulmonary capillary pressure. The best known example of this is the hydraulic formula of Gorlin for calculating the mitral valve area. In its clinical application this formula utilizes accepted principles of hydraulics for the calculation of orifice size. When applied to the mitral area, the left atrial pressure is estimated by substituting the indirect wedge pressure.

Gorlin's formula

\[
MVA = \frac{MVF}{31 \sqrt{PC} - 5}
\]

\(MVA\) represents mitral valve area in cm.²
\(MVF\) represents mitral valve flow in ml. per second or cardiac output in ml. per second
\(PC\) = pulmonary capillary pressure in mm. Hg (obtained by "wedge")
\(5\) = left ventricular diastolic pressure in mm. Hg, assumed
\(31 = \sqrt[2]{\pi} \times 0.7 \times 1960\)

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Recently catheterization of the left heart has evolved as a direct means of measuring the hemodynamic events in the chambers of the left heart. This technic provides a simple method for assaying the nature of the anatomic abnormalities of the diseased mitral and aortic valves. It also permits comparison of data obtained from catheterization of both sides of the heart and especially for the comparison of the wedge and left atrial pressures.

Connolly, Kirklin, and Wood obtained simultaneous wedge and left atrial pressures during mitral valvotomy in 5 patients. Although they noted a close correlation between the left atrial and wedge pressures, it is generally accepted that data obtained with the open chest and positive pressure anesthesia are often inaccurate and not reproducible. In 1953 Björk published the findings from the first group of patients studied by simultaneous catheterization of both the right and left hearts. Of the 9 patients studied 7 had complete data for comparison, including wedge and left atrial pressures. In 5 of these patients a close correlation between the wedge and left atrial pressures was noted. However, in 2 patients a significant difference between the wedge and left atrial pressure was seen. Similar disparities have been noted by other investigators. In a consideration of the relationships between the wedge and left atrial pressures, it would seem that any observed differences between these pressures cannot be regarded as representing real differences between the pulmonary capillary and left atrial pressures, particularly if the latter pressure is higher; in this event there could be no flow from right to left. The mean left atrial pressures must be lower than the pulmonary capillary pressure if unidirectional pulmonary flow is to occur. It follows, therefore, that any observed difference between the wedge and left atrial pressures represents a basic fault in measuring the left atrial pressure, or the wedge pressure is not a quantitative reflection of the pulmonary capillary pressure.

In an effort to resolve this problem a study was undertaken to compare the wedge and left atrial pressures in a group of patients from whom the necessary data has been obtained. Accordingly, the findings in a group of 32 patients subjected to left heart catheterization were reviewed. Twelve of these patients had complete right heart studies including authentic wedge pressures. Two of this latter group had examinations before and after mitral valvotomy, giving a total of 14 sets of data.

**METHODS**

All the patients were adults. The indication for catheterization in 3 of these was interatrial septal defect, while the remaining 11 cases had mitral or aortic disease. All studies were carried out under basal conditions with local anesthesia. The left heart studies were patterned after the method of Björk, with direct needle puncture of the left heart from the right paravertebral approach with a 15-cm. no.-17 needle. The ventricular pressures were obtained by direct puncture of this chamber rather than by passing a catheter from a needle in the left atrium. The pulmonary artery pressures were also obtained by direct needle puncture. The right heart catheterizations were done by a standard technic with the patient in the supine position. The zero setting for the pressure head during the left heart studies was the left atrium as marked on the chest wall at fluoroscopy. In the right heart studies the zero setting was 10 cm. from the table. All pressures were recorded by strain gage pick-up with photographic oscillographic recording. The wedge pressure was considered present at that point in the pulmonary arterial tree beyond which a no. 4 catheter would not pass but permitted sampling of fully oxygenated blood. The mean values were obtained by electric integration of the oscillographic tracings.

**RESULTS**

In the 14 sets of data obtained from 12 patients (table 1), 4 patients had no valvular disease, and the left atrial pressures in this group were normal or slightly elevated. In the remaining 8 cases with mitral or aortic valvular lesions, the left atrial pressure was elevated in all but 1. The mean and average values were compared; the latter values were found to be slightly lower. In the entire group a close correlation existed between the wedge
TABLE 1.—Data from Twelve Patients Having Fourteen Combined Heart Catheterizations

<table>
<thead>
<tr>
<th>Patients</th>
<th>Pulmonary artery (mm. Hg)</th>
<th>Left ventricle (mm. Hg)</th>
<th>Left atrium CV wave/Diastole (mm. Hg)</th>
<th>Pulmonary capillary pressure (mm. Hg)</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>35/15</td>
<td>110/5</td>
<td>9/4</td>
<td>13/6</td>
<td>Interatrium septal defect</td>
</tr>
<tr>
<td>2</td>
<td>20/10</td>
<td>118/9</td>
<td>8/3</td>
<td>11/5</td>
<td>Interatrium septal defect</td>
</tr>
<tr>
<td>3</td>
<td>40/20</td>
<td>90/0</td>
<td>10/2</td>
<td>12/4</td>
<td>Interatrium septal defect</td>
</tr>
<tr>
<td>4</td>
<td>32/10</td>
<td>135/3</td>
<td>22/18</td>
<td>23/21</td>
<td>Mitral stenosis</td>
</tr>
<tr>
<td>5</td>
<td>18/15</td>
<td>140/6</td>
<td>15/9</td>
<td>15/9</td>
<td>Mitral stenosis</td>
</tr>
<tr>
<td>6</td>
<td>65/40</td>
<td>128/2</td>
<td>45/2</td>
<td>28/12</td>
<td>Mitral insufficiency</td>
</tr>
<tr>
<td>7</td>
<td>58/35</td>
<td>100/0</td>
<td>42/9</td>
<td>21/6</td>
<td>Mitral insufficiency</td>
</tr>
<tr>
<td>8</td>
<td>68/48</td>
<td>98/5</td>
<td>55/45</td>
<td>22/20</td>
<td>Mitral stenosis (pulmonary edema)</td>
</tr>
<tr>
<td>9</td>
<td>48/35</td>
<td>92/9</td>
<td>32/26</td>
<td>20/14</td>
<td>Mitral stenosis</td>
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<tr>
<td>10</td>
<td>45/32</td>
<td>105/8</td>
<td>28/22</td>
<td>19/9</td>
<td>Mitral stenosis</td>
</tr>
<tr>
<td>11</td>
<td>60/35</td>
<td>90/0</td>
<td>45/38</td>
<td>22/16</td>
<td>Mitral stenosis (pulmonary edema)</td>
</tr>
<tr>
<td>12</td>
<td>60/33</td>
<td>120/5</td>
<td>12/6</td>
<td>16/12</td>
<td>Case 11, postoperative</td>
</tr>
<tr>
<td>13</td>
<td>50/38</td>
<td>105/5</td>
<td>30/25</td>
<td>16/8</td>
<td>Mitral stenosis</td>
</tr>
<tr>
<td>14</td>
<td>52/34</td>
<td>130/0</td>
<td>8/5</td>
<td>10/6</td>
<td>Case 13, postoperative</td>
</tr>
</tbody>
</table>

Cases 1 to 5 have little or no wedge/left atrium difference. In this group there is only 1 case where the left atrial pressure was significantly elevated (over 15).

Cases 6 and 7 represent severe mitral regurgitation. The wedge pressure curve shows CV waves 21 and 17 mm. less than the CV waves in the left atrial pressure.

Cases 11 and 13 are preoperative and postoperative studies that show marked wedge/left atrial pressure differences preoperatively when the left atrial pressure was high but no difference or reversal of the ratio when the left atrial pressure drops to low ranges postoperatively.

Cases 8, 9, 10, and 11 are all "tight" mitral with high left atrial pressures. In each case there is a significant wedge/left atrial pressure difference (9 to 33 mm.).

Cases 8 and 11 had clinical pulmonary edema with left atrial pressures compatible with this finding (over 35 mm. Hg) while the wedge was below 25 mm. Hg. (It is assumed that the colloid osmotic pressure component of the pulmonary capillary pressure averages 25 mm. Hg. Pulmonary edema should occur when the hydrostatic pressure exceeds 25 to 35 mm. Hg.)

and the left atrial pressure in 6 cases while a significant difference was noted in 8. The group with a marked difference between wedge and left atrial pressures included all cases in which the left atrial pressure was markedly elevated (over 20 mm. Hg). Furthermore the greatest differences occurred in those patients with the highest left atrial pressures. Of special note in this regard was case 11 in which the preoperative atrial pressure difference was 21 mm. Hg. Postoperatively a reversal of the ratio was seen. The other cases with studies before and after surgery showed a similar though less marked reversal. In the 2 cases of severe mitral insufficiency, the mean left atrial pressure exceeded the wedge pressure by 14 mm. Hg and 12 mm. Hg and the wedge tracing failed to show the height of the giant CV waves by 17 mm. Hg and 21 mm. Hg. In the patients with interatrial septal defects, the pressure differences were slight and were due to slight increase in the wedge pressure. In cases 8 and 11, with clinical pulmonary edema, the left atrial pressure was in the range compatible with this clinical finding (in excess of 35 mm. Hg) while the wedge pressure was well below edema levels.

**DISCUSSION**

This study was undertaken to evaluate the wedge pressure as an index of the pulmonary capillary pressure. To this end the data from a group of 12 patients having 14 combined heart studies were analyzed. In approximately 50 per cent of the cases a significant difference between the wedge pressure and the left atrial pressure was noted. Since the left atrial pressure and the pulmonary capillary pressure are virtually identical, any indirect method for measuring this latter pres-
sure should give values similar to the more direct left atrial pressure. Failing this, one must assume that the wedge pressure is not an accurate quantitative index of the capillary pressure. The possibility that the left atrial pressures as obtained in this study were inaccurate was obviated by the use of the end-diastolic pressure in the ventricle as the 0 to 5 baseline. The most striking deficit in the wedge tracings was seen in those cases of mitral insufficiency in which the wedge contours mirrored the left atrial waves but failed to quantitate these wave values by significant margins. In patients studied while in pulmonary edema, it was noted that the left atrial values were in excess of the edema levels, while the wedge values were far below this line.

It is not the purpose of this study to explain or speculate on the inaccuracy of the wedge pressure but rather to demonstrate that such inaccuracy is present. Therefore it would seem logical to abandon an indirect technic subject to such a significant error in favor of the more exact method of left heart catheterization. The analysis of timed pressure curves from the left atrium allows for ready definition of the nature and domination of the valvular lesions present and provides accurate values for use in the hydrokinetic orifice formulas and other basic techniques for evaluation of pressure flow systems.

Summary

A comparison of the wedge and left atrial pressures was carried out in a group of 12 patients with congenital and acquired heart disease. In 5 patients the left atrial pressures were not elevated and the wedge pressures showed similar contours and corresponding values. In 7 patients with elevated left atrial pressures the wedge pressures did not reflect these elevations with quantitative differences ranging from 9 to 33 mm. Hg. Two patients with clinical pulmonary edema and mean left atrial pressures of 50 and 41 had mean wedge pressures of 20 and 16.

In this study the wedge pressures paralleled the left atrial pressures when the latter were within normal ranges. The wedge pressures failed to reflect the left atrial pressures by significant values when the left atrial pressures were elevated.

**Summario in Interlingua**

Un comparation del pression a cuneo e del pression sinistro-atrial esseva effectuate in un gruppo de 12 patientes con congenite e acquirite morbo cardiaci. In 5 patientes le pression sinistro-atrial non esseva elevate, e le pression a cuneo mostrava simile contornos e correspondente valores. In 7 patientes con elevate pression sinistro-atrial, le pression a cuneo non reflecteva iste elevationes. Le differentias quantitative variava inter 9 e 33 mm. Hg. Duo patientes con clinic edema pulmonar e valores medie del pression sinistro-atrial de 50 e 41 habeva valores medie del pression a cuneo de 20 e 16.

In iste studio le pressiones a cuneo correspondeva al pressiones sinistro-atrial in tanto que istos se trovava intra le limites normal. Le pressiones a cuneo non reflecteva le pressiones sinistro-atrial e differeva ab illos significative mente quando le pressiones sinistro-atrial esseva elevate.

**References**


One hundred consecutive patients with atrial septal defect (ASD) are presented with a review of the clinical features and complications. The anatomy of 3 types is described: atrioventricular (A-V) defects, fossa ovalis defects, superior caval defects. These may be present in combination. The now well-recognized clinical signs of ASD are reviewed—right ventricular type of cardiac impulse, systolic lift over the outflow tract of the right ventricle, pulmonary systolic thrill (20 per cent), pulmonary systolic murmur loudest in midsystole, an obviously split second sound. A diastolic murmur may be present (20 per cent). A short delayed tricuspid murmur is often heard (50 per cent). The A-V type may be suspected with (1) unusual cardiac enlargement in childhood, (2) physical underdevelopment, (3) pulmonary hypertension below the age of 20, (4) pansystolic murmur of mitral incompetence, (5) left ventricular hypertrophy in the electrocardiogram, (5) left-to-right shunt at the atrial and ventricular levels, (7) bacterial endocarditis, (8) complete heart block. Superior caval defects may show a localized dilatation of the superior vena cava. Catheterization shows arterialized blood in the superior vena cava. The complications of ASD are obstructive pulmonary hypertension (15 patients, 7 with reversed shunts), pulmonary stenosis (11), mitral stenosis (6), atrial fibrillation (10), anomalous pulmonary veins (10). Cardiac symptoms were mild to the age of 20, but a grossly enlarged heart was found in 20 per cent of the cases aged 20 to 40 years and 60 per cent of those over 40. Consequently, the authors believe that the proper time to close an uncomplicated fossa ovalis defect with a shunt of 2:1 or more is as soon as the diagnosis is established and preferably before the age of 20. Thirty of the 100 patients with ASD were rejected for surgery for 1 of the following reasons: (1) A-V defect, (2) reversed shunt with cyanosis (3) obstructive pulmonary hypertension, (4) systemic hypertension, (5) advanced age and poor condition. Closure of an ASD by open heart surgery under hypothermia was completed in 40 patients. There was only 1 fatality, following a second operation undertaken to resuture the defect. In another patient, an unsatisfactory result was due to deflection of the inferior vena cava into the left atrium. Among the remaining patients, the immediate results were satisfactory.

Kurland
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