Atrial fibrillation modifies the association between pulmonary artery wedge pressure and left ventricular end-diastolic pressure

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Aims

During right heart catheterization, pulmonary artery wedge pressure (PAWP) is often assumed to reflect left ventricular filling pressure. We sought to determine the impact of atrial fibrillation (AF) on the relationship between PAWP and left ventricular filling pressure, as measured by left ventricular end-diastolic pressure (LVEDP).

Methods and results

We performed simultaneous left and right heart catheterization in 123 patients (mean age 69 years, 28% with AF) referred for suspicion of pulmonary hypertension (PH). The correlation between PAWP and LVEDP was moderate ($R^2 = 0.42$). The relationship between PAWP and LVEDP was modified by heart rhythm ($P$ for interaction <0.01). In sinus rhythm, PAWP underestimated LVEDP (Bland–Altman mean difference: $-2.96$ mmHg, limits of agreement 6.6 to $-12.5$; $R^2 = 0.54$), whereas in AF, PAWP overestimated LVEDP (Bland–Altman mean difference: $4.76$ mmHg; limits of agreement: 12.2 to $-3.3$; $R^2 = 0.58$). These differences impacted the differentiation between pre- and post-capillary PH, dependent on the use of either PAWP or LVEDP. In AF, post-capillary PH based on PAWP would have been classified as pre-capillary PH in 35% of patients if based on LVEDP. The opposite is true for sinus rhythm where 31% of pre-capillary PH based on PAWP would have been classified as post-capillary PH if based on LVEDP.

Conclusion

The relationship between PAWP and LVEDP varies by heart rhythm, with PAWP being higher than LVEDP among AF patients and lower than LVEDP among patients in sinus rhythm. Rhythm status and influences on the PAWP–LVEDP relationship should be considered when distinguishing between pre-capillary and post-capillary PH.

Keywords

Pulmonary artery wedge pressure • Atrial fibrillation • Heart failure with preserved ejection fraction • Pulmonary hypertension

Introduction

During invasive haemodynamic cardiac characterization, pulmonary artery wedge pressure (PAWP) measured by right heart catheterization is often regarded as a surrogate for left ventricular filling pressure when the latter is not directly measured by simultaneous left heart catheterization. Such invasive haemodynamic data are of diagnostic and therapeutic importance. For the work-up of patients with pulmonary hypertension (PH), PAWP is used to differentiate pre-capillary from post-capillary PH and to calculate pulmonary vascular resistance. For instance, the presence of raised left ventricular end-diastolic pressure (LVEDP) in association with elevated mean pulmonary arterial pressure classifies the patient as having post-capillary PH, which has different therapeutic implications compared with patients with pre-capillary PH.1

Pulmonary artery wedge pressure is a reflection of left atrial (LA) pressure. However, the usefulness of PAWP as a surrogate for LVEDP has been questioned in recent studies, showing only a moderate or poor correlation between PAWP and LVEDP.2–5 The lack of a perfect correlation has been attributed to differences in

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methods of measuring PAWP, with end-expiratory measurements resulting in a better correlation. In addition, measuring techniques as well as patient characteristics are known to contribute to discrepancy between PAWP and LVEDP. We hypothesized that alterations in LA haemodynamics, as occur in atrial fibrillation (AF), may also impact the relationship between PAWP and LVEDP. Accordingly, we aimed to determine the impact of AF on the relationship between PAWP and LVEDP among patients undergoing simultaneous left and right heart catheterization, and to investigate the effects of AF on the accuracy of PAWP measurement in reflecting left ventricular filling pressure using LVEDP.

**Methods**

**Study patients**

All patients (n = 137) who underwent simultaneous left and right heart catheterization between 2011 and 2014, without mitral valve stenosis or severe mitral valve regurgitation, were retrospectively analysed. Indications for simultaneous left and right heart catheterization were: suspicion or evidence of PH with or without coronary artery disease and heart failure. All pressure measurements were performed for clinical indications in our PH referral centre at the University Medical Center Groningen. In total, 14 patients were excluded due to incomplete data, resulting in 123 patients for study analyses. Patient characteristics were obtained from our centres’ digital medical records. Echocardiographic parameters at the time of (n = 87) or up to one year before (n = 34) invasive pressure measurements were analysed from our digital echocardiographic database. Echocardiographic data were not obtained in two patients. Patient history of these patients did not mention any mitral valve disease.

Atrial fibrillation was defined as having a history of AF (including type of AF) and having AF during invasive pressure measurement recordings. Pulmonary hypertension classification groups were defined according to the current European Society of Cardiology (ESC) guidelines.

**Haemodynamic measurements**

Haemodynamic measurements were performed with the patient in fasting state, in supine position. The transducer was adjusted to reflect the mid-thorax line. A 7F thermodilution balloon-tipped catheter (standard catheter for PAWP measurements) was inserted percutaneously, under local anaesthesia, into the right femoral vein and floated under fluoroscopy to the right atrium. The catheter was then advanced into the right ventricle, and positioned in the pulmonary artery and wedge position. Saturation, heart rate and blood pressure were measured. A change from the typical pulmonary artery waveform to the typical wedge pulmonary pressure waveform on inflation of the balloon catheter and typical fluoroscopic position of the catheter was considered for a satisfactory wedge pulmonary pressure—characterized by distinct “a” and “v” waveforms.

For left heart catheterization, an end-hole catheter (multipurpose) was introduced into the right femoral artery via a 6F sheath and advanced into the aorta and left ventricle to measure the aortic pressure and left ventricular systolic and end-diastolic pressure. Cardiac output (Fick equation), pulmonary arteriolar resistance and total systemic vascular resistance index were calculated.

**Simultaneous pressure recordings**

All haemodynamic measurements were taken before any contrast infusion into the coronary arteries or cardiac chambers was performed. When both (pulmonary artery and left ventricle) pressure catheters were simultaneously in place, two recordings were made with each recording simultaneously measuring both PAWP and LVEDP (Figure 1). The first recording was made during normal inspiration and expiration measuring mean PAWP and LVEDP, using automated pressure measurements. For further analysis, the second recording was taken during end-expiration. All recordings were retrospectively and blindly analysed by one single lab technician (T.E.V.) using Sensis Acquisition System (Siemens, The Hague, The Netherlands). For mean PAWP, automated analysis incorporated the contribution of both the A and V wave. In AF, the A wave is lacking. Therefore, A wave analysis was manually disabled by the technician. For these patients, the V wave has more influence on the mean PAWP value. This is the reason why we excluded patients with severe mitral valve regurgitation, since this can result in a disproportionally high V wave. For LVEDP measurement, the Z-point (80–120 ms behind QRS complex) was used.

For pressure analysis at end-expiration, the value was based on mean PAWP in a manually selected time period at end-expiration (regardless of patient’s rhythm), in accordance with current PH guidelines. Briefly, the pressures at end-expiration were measured by the algorithm of the system after narrowing the measuring window until the system gives the pressures at end-expiration (3–5 beats). To validate the tracing method, we randomly selected 15 patients for blind review by a second observer. Inter-observer agreement was high ($R^2 = 0.89$), validating the tracing method.

**Statistical analyses**

Pearson’s correlation coefficient was used to determine the correlation between PAWP and LVEDP. Interaction was tested to
Table 1: Patient characteristics

<table>
<thead>
<tr>
<th></th>
<th>All patients (n = 123)</th>
<th>Sinus rhythm (n = 89)</th>
<th>AF (n = 34)</th>
<th>P-value</th>
</tr>
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<tbody>
<tr>
<td>Female sex</td>
<td>80 (65)</td>
<td>59 (66)</td>
<td>14 (41)</td>
<td>0.440</td>
</tr>
<tr>
<td>Age (years)</td>
<td>69 ± 11</td>
<td>67 ± 11</td>
<td>74 ± 9</td>
<td>0.001</td>
</tr>
<tr>
<td>BSA (m²)</td>
<td>1.9 ± 0.2</td>
<td>1.9 ± 0.2</td>
<td>1.9 ± 0.3</td>
<td>0.989</td>
</tr>
<tr>
<td>PH classification</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No PH</td>
<td>35 (29)</td>
<td>33 (37)</td>
<td>2 (6)</td>
<td>0.001</td>
</tr>
<tr>
<td>PAH</td>
<td>14 (11)</td>
<td>12 (13)</td>
<td>2 (6)</td>
<td>0.371</td>
</tr>
<tr>
<td>Left heart disease</td>
<td>70 (56)</td>
<td>40 (45)</td>
<td>30 (88)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Chronic hypoxia</td>
<td>4 (3)</td>
<td>4 (5)</td>
<td>0 (0)</td>
<td></td>
</tr>
<tr>
<td>NYHA functional class III/IV</td>
<td>48 (39)/0</td>
<td>28 (32)/0</td>
<td>20 (59)/0</td>
<td>0.005</td>
</tr>
<tr>
<td>Haemodynamic measurements</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PAWP (mmHg)</td>
<td>15 ± 7</td>
<td>12 ± 6</td>
<td>20 ± 6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LVEDP (mmHg)</td>
<td>15 ± 6</td>
<td>15 ± 7</td>
<td>16 ± 4</td>
<td>0.785</td>
</tr>
<tr>
<td>sPAP (mmHg)</td>
<td>47 ± 20</td>
<td>45 ± 22</td>
<td>42 ± 13</td>
<td>0.041</td>
</tr>
<tr>
<td>dPAP (mmHg)</td>
<td>18 ± 8</td>
<td>17 ± 8</td>
<td>20 ± 7</td>
<td>0.025</td>
</tr>
<tr>
<td>mPAP (mmHg)</td>
<td>30 ± 11</td>
<td>28 ± 13</td>
<td>33 ± 8</td>
<td>0.029</td>
</tr>
<tr>
<td>PVR (dyn/s/cm²)</td>
<td>215 ± 214</td>
<td>205 ± 237</td>
<td>243 ± 141</td>
<td>0.386</td>
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<tr>
<td>sAo (mmHg)</td>
<td>143 ± 26</td>
<td>1439 ± 27</td>
<td>138 ± 23</td>
<td>0.170</td>
</tr>
<tr>
<td>dAo (mmHg)</td>
<td>69 ± 12</td>
<td>69 ± 12</td>
<td>69 ± 13</td>
<td>0.932</td>
</tr>
<tr>
<td>Heart rate (b.p.m.)</td>
<td>72 ± 12</td>
<td>71 ± 12</td>
<td>74 ± 14</td>
<td>0.091</td>
</tr>
<tr>
<td>CO (L/min)</td>
<td>5.8 ± 1.7</td>
<td>6.1 ± 1.8</td>
<td>5.3 ± 1.3</td>
<td>0.007</td>
</tr>
<tr>
<td>Co-morbidities</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Hypertension</td>
<td>84 (67)</td>
<td>58 (65)</td>
<td>25 (73)</td>
<td>0.376</td>
</tr>
<tr>
<td>Diabetes</td>
<td>31 (25)</td>
<td>22 (25)</td>
<td>9 (26)</td>
<td>0.841</td>
</tr>
<tr>
<td>COPD</td>
<td>24 (19)</td>
<td>16 (18)</td>
<td>8 (24)</td>
<td>0.487</td>
</tr>
<tr>
<td>AF</td>
<td>34 (28)</td>
<td>–</td>
<td>34 (100)</td>
<td></td>
</tr>
<tr>
<td>Paroxysmal/persistent/permanent</td>
<td>7</td>
<td>3/25</td>
<td>–</td>
<td>7</td>
</tr>
<tr>
<td>Antiarrhythmic agents</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Beta-blocker</td>
<td>86 (70)</td>
<td>58 (65)</td>
<td>28 (82)</td>
<td>0.087</td>
</tr>
<tr>
<td>ACE-inhibitor/ARB</td>
<td>55 (44)</td>
<td>32 (58)</td>
<td>23 (41)</td>
<td>0.002</td>
</tr>
<tr>
<td>Diuretic</td>
<td>59 (48)</td>
<td>36 (40)</td>
<td>23 (68)</td>
<td>0.007</td>
</tr>
<tr>
<td>Calcium channel blocker</td>
<td>9 (7)</td>
<td>0</td>
<td>7 (21)</td>
<td></td>
</tr>
<tr>
<td>Digoxin</td>
<td>9 (7)</td>
<td>5 (6)</td>
<td>4 (12)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Data are expressed as mean ± standard deviation, or numbers and proportion of patients (%).

ACE, angiotensin-converting enzyme; AF, atrial fibrillation; ARB, angiotensin receptor blocker; BSA, body surface area; CI, cardiac index; CO, cardiac output; COPD, chronic obstructive pulmonary disease; dAo, invasively measured diastolic pressure in the aorta; dPAP, diastolic pulmonary artery pressure; LVEDP, left ventricular end-diastolic pressure; mPAP, mean pulmonary artery pressure; NYHA, New York Heart Association; PAH, pulmonary arterial hypertension; PAWP, pulmonary artery wedge pressure; PH, pulmonary hypertension; PVR, pulmonary vascular resistance; sAo, invasively measured systolic blood pressure in the aorta; sPAP, systolic pulmonary artery pressure.

determine the individual subgroup effect. For subgroup analyses based on heart rhythm, differences between groups were determined by Bland–Altman plots and sampled t-tests. To determine the consequence of PAWP use in differentiating between pre- and post-capillary PH, both PAWP and LVEDP were dichotomized as ≤15 mmHg or >15 mmHg; cross tabs were made and significance was tested using McNemar’s test. A P-value of <0.05 was considered significant. All statistical analyses were performed using SPSS software version 22 (SPSS Inc., Chicago, IL, USA).

Results

Overall, 123 patients were analysed (Table 1). Mean age was 69 years, 65% were female. Seventy patients (56%) had PH due to left heart disease (PH-LHD), 14 patients (11%) had pulmonary arterial hypertension (PAH), 4 patients (3%) had PH due to hypoxic lung disease, and 35 patients (29%) had no PH. Of the 70 patients with PH-LHD, 69 patients had symptomatic heart failure with preserved ejection fraction (HFrEF) (left ventricular ejection fraction ≥45% and New York Heart Association functional class II–IV). One patient had heart failure with reduced ejection fraction (HFrEF; left ventricular ejection fraction 39%). The non-PH group consisted of the following patients: 18 HFrEF patients, 2 HFrEF patients, 7 patients without heart failure, 5 tetralogy of Fallot patients, 2 patients with chronic obstructive pulmonary disease, and one patient with atrial septal defect.

A total of 34 patients (28%) had AF at the time of invasive pressure measurements. Of these, 71% had permanent AF. Hypertension (67%), diabetes (25%), and chronic obstructive pulmonary disease (19%) were the other most frequent occurring co-morbidities.

Echocardiographic parameters are reported in the Supplementary material online, Table S1.
Figure 2 Pulmonary artery wedge pressure (PAWP) vs. left ventricular end-diastolic pressure (LVEDP). Correlation and mean values of PAWP and LVEDP divided by heart rhythm during pressure measurements, using automated pressure measurements.

Figure 3 Agreement between pulmonary artery wedge pressure (PAWP) and left ventricular end-diastolic pressure (LVEDP). Bland–Altman plots of PAWP and LVEDP for sinus rhythm (blue) and atrial fibrillation (red). The middle line indicates the mean difference between PAWP and LVEDP (–2.96 and 4.76 mmHg for sinus rhythm and atrial fibrillation, respectively). Automated pressure measurements were used. Dotted lines indicate upper and lower limits of agreement (6.6 to –12.5 for sinus rhythm; 12.2 to –3.3 for atrial fibrillation).

Pulmonary artery wedge pressure vs. left ventricular end-diastolic pressure

Using automated pressure measurements, no difference was found between PAWP and LVEDP when the group was analysed as a whole [Bland–Altman mean difference: −0.8 mmHg; limits of agreement: 10.5 to −12.1; 95% confidence interval (CI) 0.2 to −1.9]. The correlation between PAWP and LVEDP was moderate \((R^2 = 0.42)\). Using automated pressure measurements, the relationship between PAWP and LVEDP was modified by heart rhythm \((P\text{ for interaction }< 0.01)\). In sinus rhythm, PAWP underestimated LVEDP (Bland–Altman mean difference: −2.96 mmHg; limits of agreement: 6.6 to −12.5; 95% CI −1.9 to −3.9; \(R^2 = 0.54\)). In AF, PAWP overestimated LVEDP (Bland–Altman mean difference: 4.76 mmHg; limits of agreement: 12.2 to −3.3; 95% CI 6.2 to 3.3; \(R^2 = 0.58\)) (Figures 2 and 3). PH-LHD was seen more frequently in AF patients (Table 1); however, interaction analysis did not
show a significant interaction (P = 0.146). Sinus rhythm and AF patients showed similar left ventricular ejection fraction. Diastolic dysfunction was present in both groups. Patients with AF showed a slightly reduced right ventricular function (see Supplementary material online, Table S1).

By dichotomizing both PAWP and LVEDP at the distinction level of pre- and post-capillary PH (<15 mmHg or >15 mmHg), sensitivity and specificity between the two parameters could be determined (Tables 2 and 3). In AF, post-capillary PH based on PAWP would actually have been misclassified as pre-capillary PH in 35% of patients if based on LVEDP alone (McNemar’s P = 0.02). The opposite is true for sinus rhythm where 31% of pre-capillary PH based on PAWP would have been classified as post-capillary PH if based on LVEDP (McNemar’s P < 0.001) (Table 3).

### Atrial fibrillation modifies wedge pressure measurement

The lack of an adequate correlation has also been attributed to differences in method of measuring PAWP, with end-expiratory measurements resulting in a better correlation. These data are, however, in contrast with another study of mainly pre-capillary PH, which showed that for measurements of <15 mmHg, PAWP was an acceptable surrogate for LVEDP. The lack of an adequate correlation has also been attributed to differences in methods of measuring PAWP, with end-expiratory measurements resulting in a better correlation.

### Discussion

In the present study, we investigated the effects of heart rhythm on the correlation between PAWP and LVEDP during simultaneous left and right pressure measurements. In AF, PAWP overestimated LVEDP, whereas in sinus rhythm, PAWP underestimated LVEDP. These findings have important implications for the detection and interpretation of raised left-sided filling pressures using right heart catheterization as well as for classification of PH.

There are prior reports with inconsistent data on differences between PAWP and LVEDP measurements. Halpern and Taichman previously described that PAWP systematically underestimates LVEDP. A poor correlation between PAWP and LVEDP was also reported in a cohort of veterans. These data are, however, in contrast with another study of mainly pre-capillary PH, which showed that for measurements of <15 mmHg, PAWP was an acceptable surrogate for LVEDP. The lack of an adequate correlation has also been attributed to differences in methods of measuring PAWP, with end-expiratory measurements resulting in a better correlation.

However, none of the previous studies reported heart rhythm during invasive pressure measurements or investigated the possible effect of arrhythmia on these measurements. We therefore extend previous data and provide an explanation for the poor correlations found in previous studies by showing a profound impact of AF on the difference between PAWP and LVEDP during simultaneous invasive measurements. In sinus rhythm, PAWP underestimated LVEDP, whereas in AF, PAWP overestimated LVEDP. Our results suggest that the presence or absence of AF should be taken into account when evaluating PAWP as a surrogate for LVEDP. In addition, these data underline the important haemodynamic effects of an abnormal heart rhythm.

### Clinical implications

Our results may carry implications for the invasive diagnostic strategy in PH. As stated in the current ESC guidelines, PAWP is a reflection of LA pressure. In daily practice, PAWP is often also regarded as a surrogate for LVEDP, which it is not always the case as some factors can influence the correlation between PAWP and LVEDP.
Figure 4 Pulmonary artery wedge pressure (PAWP) and left ventricular end-diastolic pressure (LVEDP) in patients with pulmonary hypertension due to left heart disease. Correlation (a) and mean values (b) of PAWP and LVEDP divided by heart rhythm during automated pressure measurements. (c) Bland–Altman plots for sinus rhythm (blue; mean difference: −2.8 mmHg, limits of agreement: 9.2 to −14.8) and atrial fibrillation (red, mean difference: 5.1 mmHg; limits of agreement: 15.5 to −3.9).

Figure 5 Pulmonary artery wedge pressure (PAWP) vs. left ventricular end-diastolic pressure (LVEDP) measured during end-expiration. Mean values (a) and Bland–Altman plots (b) of PAWP and LVEDP in sinus rhythm, measured during end-expiration. Values are divided by heart rhythm during pressure measurements (for sinus rhythm, mean difference: −1.5 mmHg, limits of agreement: 8.5 to −11.5; for atrial fibrillation, mean difference: 5.5 mmHg, limits of agreement: 15.1 to −4.1).
including mitral valve dysfunction, LA function, or pulmonary disease. LVEDP measurement is recommended in certain clinical settings during PH work-up (e.g. assessment of post-capillary PH, or if PAWP is thought to be inaccurate). However, our results show for the first time that in AF patients, LVEDP does not reflect LA pressure, but was significantly lower in simultaneous measurements making the differentiation between pre- and post-capillary PH dependent on the use of either PAWP or LVEDP. In 35% of patients with AF, LVEDP was normal, whereas PAWP was elevated reflecting back transmission of elevated pressure from the left atrium to the pulmonary capillary system. Further studies that include fluid challenge or exercise during invasive pressure measurements could be helpful to further investigate the haemodynamic effects and the PAWP–LVEDP relationship in AF patients during PH work-up.

### Atrial fibrillation, left atrial remodelling and pulmonary venous pressure

Atrial fibrillation is a common condition in patients with PH-LHD as well as in pulmonary arterial hypertension, with a prevalence of up to 60%. Because of its high prevalence, incorrect interpretation of pressure measurements can have a major impact on diagnosis and treatment. The underlying mechanism of the discrepancy between PAWP and LVEDP in the setting of AF could be multifactorial. First, in AF, atrial contraction is disorganized resulting in loss of a true atrial systole. This loss in atrial kick results in a decreased LA ejection fraction and reduced atrioventricular coupling, as previously shown. Left atrial pressure increases and is uncoupled from LVEDP, resulting in a PAWP that is higher compared with the measured LVEDP. Second, reduced LA compliance in AF may further worsen PAWP–LVEDP agreement. In a recent study in patients with HFrEF, AF was associated with a lower LA ejection fraction and higher LA stiffness. A stiff, non-compliant left atrium, as seen in patients with permanent AF as in our study, will have higher LA pressures. Such an atrium is expected to transmit more backpressure to the pulmonary veins than a compliant left atrium, thus leading to increased pulmonary venous pressure (increased PAWP) that is disproportionately higher than LVEDP, as observed from our results. These factors combined may cause the discrepancy that we found between simultaneous measured PAWP and LVEDP during AF. In addition, owing to the lack of an A wave during pressure measurements, a large V wave (for instance due to severe mitral regurgitation or stenosis) could result in a disproportional high mean PAWP during AF. In our study, patients with mitral valve stenosis or significant mitral valve regurgitation were excluded. In a recent study including patients with mitral valve stenosis and LHD, large V waves were shown to affect negative diastolic pressure gradient measurements in patients with low pulmonary vascular resistance. Manually measuring a pre-V point resulted in a 1–2 mmHg higher pressure gradient, indicating the possible effects of the V wave. Unfortunately, heart rhythm was not described, making a comparison with our study difficult. Others have suggested that measurement of a post-V wave point (end of the V wave downslope) better reflects end-diastolic pressure.

Further studies may focus on whether measuring pre- or post-V point instead of mean PAWP would decrease the difference between PAWP and LVEDP in AF. Obviously, caution should be taken in manual pressure measurements since this may not reflect the full pressure the pulmonary vasculature is subjected to during the complete heart cycle.

Conclusion

This study shows the haemodynamic effects of AF on the relationship between invasively measured PAWP and LVEDP.
These findings may have important implications for (i) the detection of raised left heart filling pressures using right heart catheterization, and (ii) classification of PH. Specifically in AF, the question now rises if LVEDP alone is the appropriate measurement for differentiating pre-capillary from post-capillary PH.

Supplementary Information

Additional Supporting Information may be found in the online version of this article:

Table S1. Echocardiographic characteristics at the time of invasive pressure measurements.

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References


