The pulmonary capillary wedge pressure accurately reflects both normal and elevated left atrial pressure

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Background Pulmonary capillary wedge pressure (PCWP) is routinely used as an indirect measure of the left atrial pressure (LAP), although the accuracy of this estimate, especially under pathological hemodynamic conditions, remains controversial.

Objectives The aim of this prospective study was to investigate the reliability of PCWP for the evaluation of LAP under different hemodynamic conditions.

Methods Simultaneous left and right heart catheterization data of 117 patients with pure mitral stenosis, obtained before and immediately after percutaneous mitral comissurotomy, were analyzed.

Results A strong correlation and agreement between PCWP and LAP measurements was demonstrated (correlation coefficient = 0.97, mean bias ± CI, 0.3 ± −3.7 to 4.2 mm Hg). Comparison of measurements performed within a 5-minute interval and those performed simultaneously revealed that simultaneous pressure acquisition yielded better agreement between the 2 methods (bias ± CI, 1.82 ± 1.98 mm Hg). In contrast to previous observations, the discrepancy between the 2 measures did not increase with elevated PCWP. Multiple regression analysis failed to identify hemodynamic confounders of the discrepancy between the 2 pressures. The ability of PCWP to distinguish between normal and elevated LAP (cutoff set at 12 and 15 mm Hg, respectively), as tested by receiver operating characteristics analysis, demonstrated a remarkably high diagnostic accuracy (area under the curve: 0.989 and 0.996, respectively).

Conclusions Although the described limits of agreement may not allow the interchangeability of PCWP and LAP, especially at lower pressure ranges, our data support the clinical use of PCWP as a robust and accurate estimate of LAP. (Am Heart J 2014;167:876-83.)

In common clinical practice, pulmonary capillary wedge pressure (PCWP) is used as an equivalent of left atrial pressure (LAP). However, regarding the accuracy of this estimate, controversy has existed over the past 65 years.

Description of the first PCWP measurements in human dates back to 1949.[1] The accuracy of PCWP as a reflection of LAP under different conditions has subsequently been investigated, and numerous early studies performed in small groups of patients questioned the interchangeability of the 2 approaches.[2-11]

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The single large-scale study that investigated the agreement between PCWP and LAP, using direct LAP measurement through transseptal puncture, was published in 1973.[12] Waltson and coworkers reported the comparison of left and right heart catheterization data collected retrospectively for 13 years from a diverse population of 700 subjects, including healthy individuals as well as patients with ischemic heart disease and a variety of valvular lesions. Results from this study confirmed a good correlation and agreement between PCWP and LAP at reference ranges of mean PCWP (≤15 mm Hg). However, at higher wedge pressures, the prediction of LAP by PCWP was subject to considerable error. In fact, in case of wedge pressures greater than 15 mm Hg, the discrepancy between mean LAP and PCWP varied by roughly 15 mm Hg, increasing further at pressures greater than 20 mm Hg.

Both for the LA and PCWP measurements, the zero level was set at the midthoracic line. However, that the tip of the 2 catheters (wedge balloon and LA) could not by any means be certainly located at the same level due to obvious anatomical reasons, a minor discrepancy regarding the absolute pressure values cannot be excluded. Nonetheless, we believe that in this case, the error that might have been introduced would be random.

Considering the significant proportion of patients with elevated wedge pressures among those undergoing right heart catheterization, the reported observation renders the predictive ability of PCWP for LAP assessment, thus its diagnostic use, highly unreliable. Despite the demonstrated lack of concordance between the 2 measures at high pressures, PCWP is extensively used in everyday clinical practice in place of LAP for diagnostics as well as for hemodynamic monitoring.

On the background of the aforementioned, the present prospective study was designed to examine the agreement between PCWP and LAP and to elucidate possible physiological or methodological factors that may influence the concordance between the 2 measures.

Methods

Study population

One hundred seventeen consecutive patients with symptomatic mitral stenosis (MS) in sinus rhythm who underwent percutaneous transvenous mitral commissurotomy (PTMC) in the Sri Sathya Sai Institute between January and June 2012 were enrolled prospectively. Subjects were excluded from the study if they had significant aortic disease, more than mild (grade > 1) mitral regurgitation, associated ischemic heart disease, systemic hypertension, or diabetes mellitus. The study protocol conformed to the ethical guidelines of the 1975 Declaration of Helsinki and was approved by the Sri Sathya Sai Institution's ethics committee. All subjects provided written informed consent. Importantly, all measurements were performed in conscious patients without the use of anesthetics. Patient characteristics are summarized in Table.

Cardiac catheterization

Simultaneous right and left heart catheterization data were obtained during the PTMC procedure, before and after balloon inflation. Right heart catheterization was performed through femoral vein access using a 6F wedge catheter (Arrow Balloon Wedge-Pressure Catheters; Teleflex, Limerick, Ireland) connected to a pressure transducer (Philips 1290 Series, Andover, MA). Right atrial mean pressure, right ventricular systolic pressure (RVSP), pulmonary artery systolic and mean pressure, and PCWP were measured under fluoroscopic guidance. Concurrently, a 6F pigtail catheter was advanced through the aorta into the left ventricle (LV) to measure the LV end-diastolic and end-systolic pressures before and after balloon inflation. Interatrial septal puncture was performed with an 8F Mullins sheath, dilator, and a Brockenbrough needle. The LA pressure was measured directly through the Mullins sheath used during valvuloplasty. Two transducers were used, one as connected to the pigtail that was passed to the LV. The other dome measured the LA pressure. Both the transducers were zeroed before pressure measurements were commenced. In all cases, sequential LAP measurements were taken within 5 minutes after PCWP measurements. In 51 cases, in addition to the sequential pressure measurements, simultaneous, beat-to-beat, LAP and PCWP tracings were also recorded. All the various pressure tracings were recorded after careful calibration, during a period of 10 seconds, and subsequently stored in dedicated software (WITT Series III; Witt Biomedical Corp, Melbourne, FL) for offline analysis. The mean pressure values for all recordings were considered. The zero-pressure level was set at the midthoracic line for both transducers. No manifold was used during the pressure measurements.

Table. Demographic and echocardiographic characteristics of the study population

Parameter	Before PTCM	After PTCM
Age (y)	32 ± 9	
Female	84 (67%)	
BSA (m^2)	1.4 ± 0.2	
HR (beats/min)	75 ± 14	79 ± 15
SBP (mm Hg)	108.5 ± 10.3	
DBP (mm Hg)	70 ± 7.3	
EF (%)	64.3 ± 9.2	63.9 ± 9.3
EF < 55%	9 (7%)	
MVA (cm^2)	0.9 ± 0.2	
LVESP (mm Hg)	134.8 ± 17	131.4 ± 18
LVEDP (mm Hg)	12.6 ± 4	16.1 ± 4.9
RVSP (mm Hg)	62.5 ± 25	50.9 ± 18.6
Mean RAP (mm Hg)	6.1 ± 3.9	6 ± 3.9
Mean PCWP (mm Hg)	25.8 ± 7.3	19.2 ± 7.1
Mean LAP (mm Hg)	26.5 ± 7.1	18.5 ± 6.5
CO (L/min)	3.6 ± 1.1	4.5 ± 1.3
PVR (Wood Units)	4.4 ± 4.7	3.5 ± 3.9
SVR (Wood Units)	27.7 ± 94	22 ± 9.5

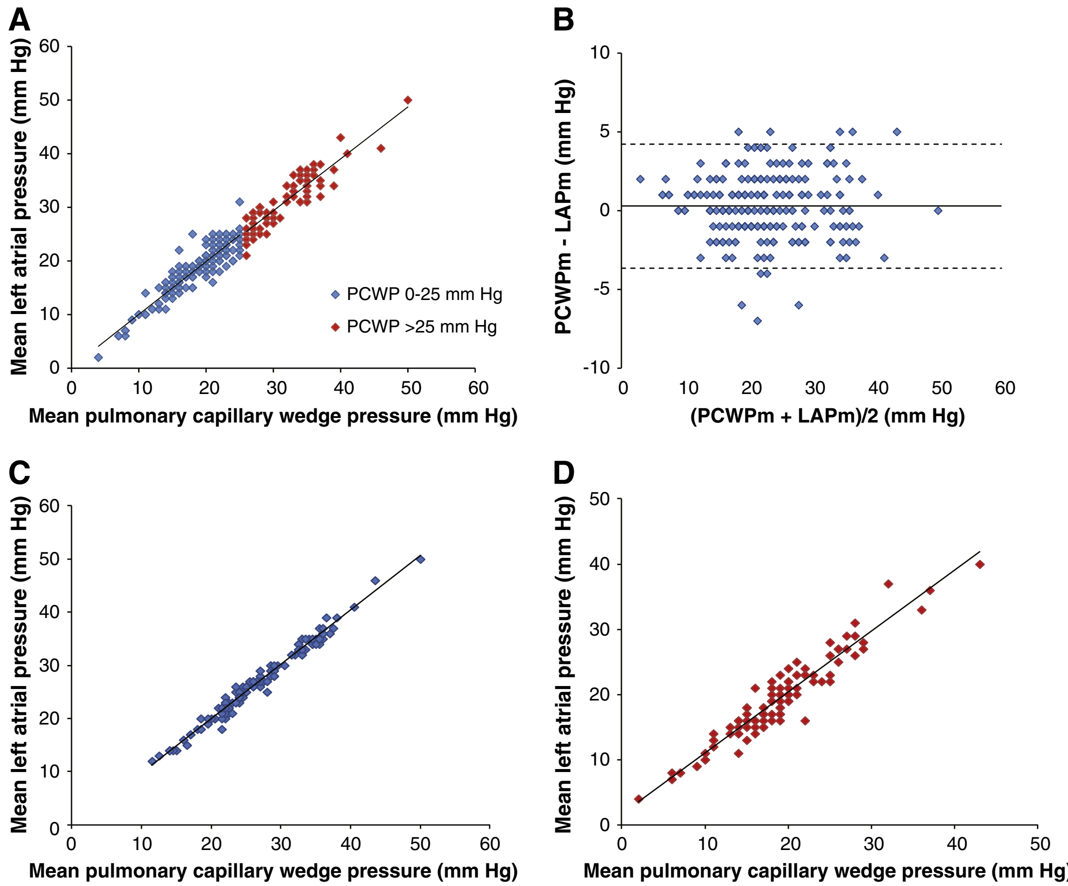
Abbreviations: *BSA*, Body surface area; *SBP*, systolic blood pressure; *DBP*, diastolic blood pressure; *MVA*, mitral valve area; *LVESP*, left ventricular systolic pressure; *LVEDP*, left ventricular end-diastolic pressure; *RVSP*, right ventricular systolic pressure; *RAP*, right atrial pressure.

Mitral valvuloplasty was performed using a 24- to 28-mm Accura balloon catheter (Vascular Concepts, Essex, UK) using standard technique. After the PTMC, pressure recordings were repeated in all patients. Cardiac output (CO) and vascular resistance were measured before and after PTMC. Cardiac output was calculated using the estimated Fick's method. Systemic vascular resistance (SVR) and pulmonary vascular resistance (PVR) were derived from mean arterial and atrial pressures using standard formulae.

Echocardiographic data

All patients underwent standard transthoracic echocardiogram using a GE Vivid E9 system (GE Ultrasound, Horten, Norway) and a 2.5-MHz matrix array transducer.

Figure 1



A, Linear regression analysis of mean LAP vs mean PCWP. Regression line: $0.97x + 0.32$. **B**, Bland-Altman plot of the agreement between mean LAP and mean PCWP. Mean bias (vertical axis): 0.3 mm Hg, 95% limits of agreement (dotted lines): −3.7 to 4.2 mm Hg. **C** and **D**, Linear regression analysis of mean LAP vs mean PCWP before (**C**; regression line: $1.02x - 0.60$) and after (**D**; regression line: $0.94x + 1.64$) PTMC.

Recordings were analyzed according to the recommendations of the American Society of Echocardiography.[13] Images were digitally stored and analyzed offline using dedicated software (EchoPac PC; GE Ultrasound, Waukesha, Wisconsin).

Statistical analysis

Statistical analysis was performed using SPSS version 20.0 for Windows (IBM Inc, Chicago, IL). Data are expressed as mean ± SD. Correlations between variables were determined using the Pearson 2-tailed correlation test. Significance of bias was tested by 1-sample t test. Multiple regression analysis was used to identify independent predictors of the difference between the PCWP and LAP. Statistical significance of the difference between the correlation coefficients was determined as described by Cohen et al.[14] Comparison of receiver operating characteristic (ROC) curves was performed, as described by Hanley and McNeil.[15] All tests were performed at 95% CIs, and a P value less than .05 was considered as statistically significant.

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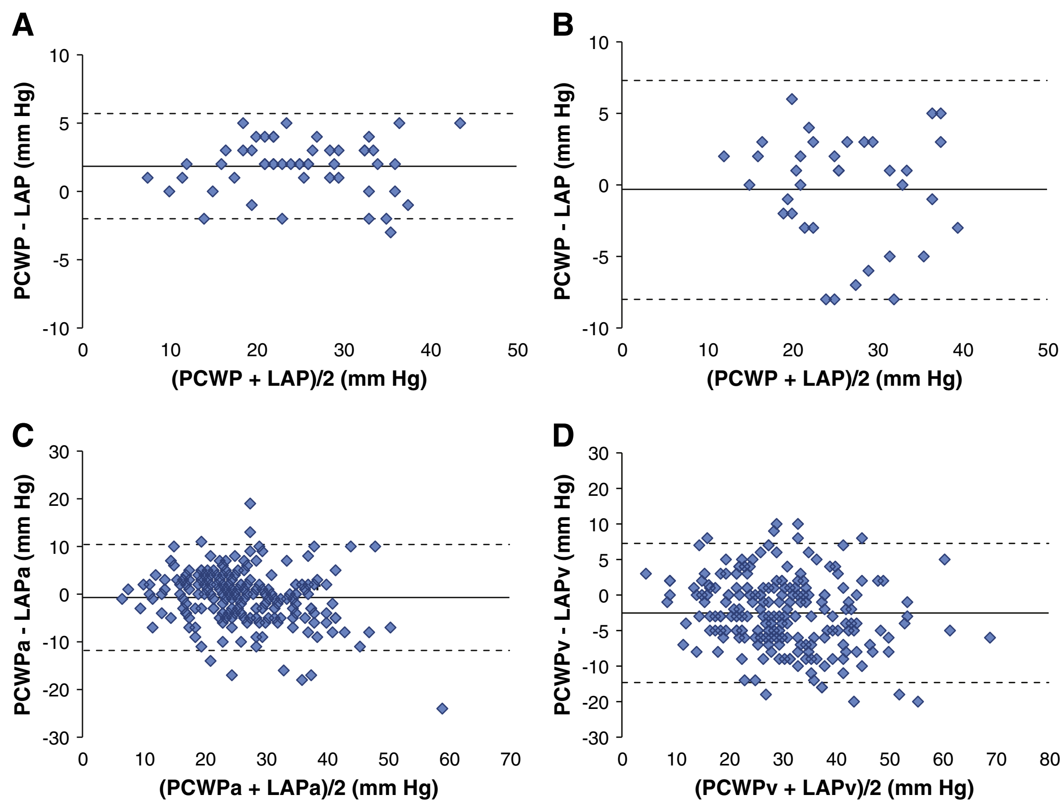
Results

Study population

Hemodynamic and echocardiographic data of 117 patients were analyzed. Preprocedural measurements were available in 116, postprocedural measurements in 110 patients, and simultaneous PCWP and LAP recordings in 51 cases (29 pre- and 22 post-PTCM). Using a cutoff value of 12 mm Hg and of 15 mm Hg, 16 (1 pre- and 15 post-PTMC) and 28 LAP measurements (5 pre- and 23 post-PTMC) were within the reference range, respectively.

Figure 2



A and **B**, Bland-Altman analysis of PCWP and LAP measurements in case of simultaneous (**A**) and subsequent (**B**) recordings. **C** and **D**, Bland-Altman plots demonstrating the agreement between PCWP and left atrial *a* (**C**) and *v* (**D**) wave peaks. Vertical axis: mean bias, dotted lines: limits of agreement.

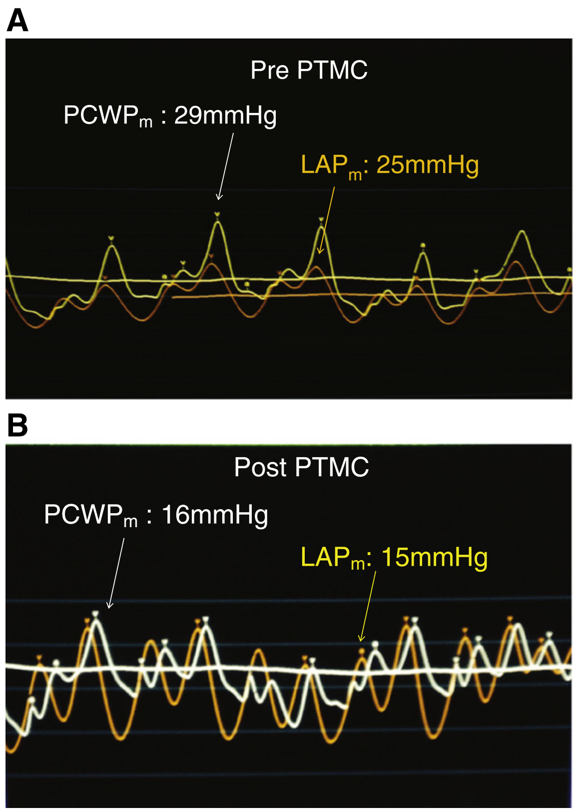
Demographic data, echocardiographic parameters, and hemodynamic measurements of the subjects are summarized in Table I.

Comparison of the PCWP and LAP measurements

The correlation of mean PCWP and mean LAP measurements, with values ranging between 4 and 50 mm Hg and 2 and 50 mm Hg, respectively, is depicted in Figure 1A. Linear regression analysis revealed a strong association between the 2 pressures, with an overall correlation coefficient of 0.97. To test the impact of elevated pressures on the above relation, the data were grouped based on PCWP values. The statistical analysis showed that the 2 measurements demonstrated consistent correlation, independent of the value of pulmonary wedge pressure, as evidenced by an identical correlation coefficient in case of PCWP ranging between 0 and 25 mm Hg and greater than 25 mm Hg ($r = 0.92$ and $r = 0.91$, respectively; $P = .67$). Bland-Altman analysis revealed a barely discernible mean difference of 0.3 mm Hg between PCWP and LAP measurements, with 95% limits of agreement of −3.7 and 4.2 mm Hg (Figure 1B). Despite some level of discrepancy between the 2 measurements, the mean error of PCWP in predicting LAP was no more than ± 2.1 mm Hg (Figure 1B), independent of the range of PCWP. The variance of bias was essentially equivalent for PCWP of less than and greater than 25 mm Hg (3.67 and 4.67, respectively; $P = .26$). The correlation between PCWP and LAP measurements was identical in measurements performed before and after PTMC ($r = 0.96$ vs 0.96, $P = .86$) (Figure 1C, D).

Comparison of the effect of subsequent vs totally simultaneous measurements revealed a significantly stronger correlation ($r = 0.86$ vs $r = 0.97$, $P < .001$) and better agreement with narrower prediction intervals (mean bias −0.31 ± 3.92 vs 1.82 ± 1.98, $P = .0012$) in case of the simultaneous recordings (Figure 2A, B). Illustrative pressure curves of the simultaneous PCWP and LAP recordings are provided in Figure 3. Accordingly, based on the results of the simultaneous measurements, using PCWP yields a slight but systematic and statistically

Figure 3



Illustrative pressure curves of simultaneous PCWP and LAP recordings before (**A**) and after (**B**) PTMC.

significant overestimation of LAP (mean bias compared with zero: $P < .01$ for simultaneous and $P = .65$ for sequential recordings). The degree of overestimation was independent of the value of PCWP ($r = 0.14$, $P = .31$).

Analysis of the concordance between waveforms of PCWP and LAP recordings revealed a good correlation of the peak *a* and *v* wave values derived by the 2 methods ($r = 0.81$ and $r = 0.90$, respectively), although compared with mean wedge and LA pressures, Bland-Altman plots showed larger mean bias with wider limits of agreement (mean bias −0.7 mm Hg [limits of agreement −11.8 to 10.4 mm Hg] and mean bias −3 mm Hg [limits of agreement −12.5 to 7.5 mm Hg], respectively) (Figure 2C, D).

To investigate hemodynamic confounders of the degree of discrepancy between PCWP and LAP, multiple regression analysis was performed including heart rate (HR), left ventricular ejection fraction (EF), CO, RVSP, SVR, and PVR as independent variables in the prediction model. Using backward linear regression analysis, none of these variables showed a significant association with the bias between the 2 invasive measurements.

Diagnostic use

To assess whether the observed slight discrepancy between the measured values of LAP and PCWP still allowed reliable differentiation between subjects with normal and elevated LAP, the diagnostic accuracy of PCWP using 2 different thresholds for normal LAP (12 and 15 mm Hg) was determined. In total, 16 measurements of LAP were ≤12 mm Hg. Using a cutoff value for PCWP of ≤13 mm Hg, 2 of these subjects were erroneously identified as having elevated LAP, whereas 1 patient with elevated LAP was identified as normal (sensitivity 99.5%, specificity 88%, accuracy 99%). At a PCWP cutoff of 14 mm Hg, 1 healthy subject was misclassified as having elevated LAP and 2 patients with overthreshold pressures as having normal LAP (sensitivity 99%, specificity 94%, and accuracy 99%).

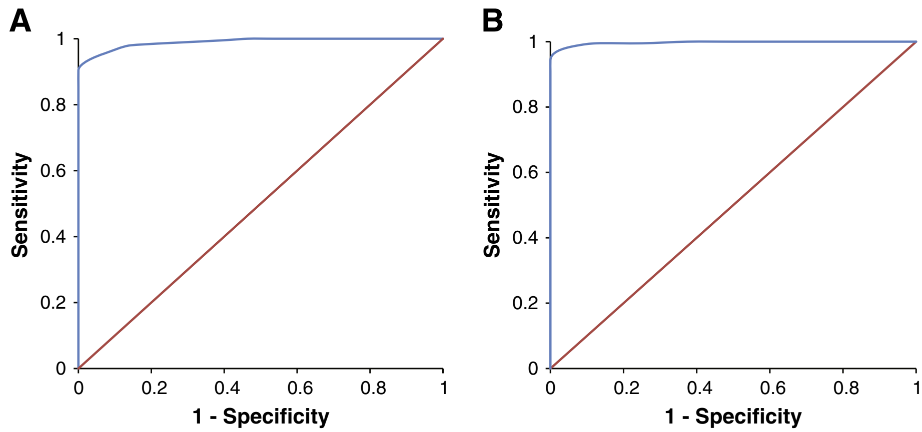
As previously described, 28 subjects had normal LAP when a limit for elevated LAP of >15 mm Hg was used. The receiver operator characteristics analysis with cutoff values of PCWP set at 15 or 16 mm Hg, provided sensitivity, specificity, and accuracy of 98%, 86%, and 97% and 95%, 93%, and 95%, respectively. The area under the ROC curves constructed with threshold values for LAP of >12 or >15 mm Hg demonstrated essentially identical diagnostic power in the 2 cases with AUC of 0.996 and 0.989, respectively ($P = .288$) (Figure 4).

Discussion

Accurate LAP assessment is essential in hemodynamic studies not only in the clinical but also in the experimental setting. Since the introduction of the Swan-Ganz catheter, direct LAP measurement is essentially replaced by measurement of the PCWP. However, it has been repeatedly demonstrated that PCWP does not always accurately reflect the LAP, especially in patients with increased PVR or elevated filling pressures, like those with MS.[8,12,16-18] Because PCWP constitutes the cornerstone in the diagnosis of precapillary vs passive pulmonary hypertension as well as in the evaluation of diastolic left ventricular function, we decided to prospectively study the accuracy of this measure for the assessment of LAP.

Our results demonstrate a strong linear correlation between the mean values of the 2 invasive measurements. More importantly, the concordance between the 2 measurements was considerably better with narrower limits of agreement compared with those previously reported. In a large retrospective study published by Walston and Kendall,[12] the authors reported limits of agreement at the range greater than 15 mm Hg between the direct LAP and corresponding PCWP measurements. Discrepancy of such a degree essentially precludes the interchangeable use of the 2 approaches and renders PCWP unreliable for hemodynamic evaluation. However, our results contradict the aforementioned findings

Figure 4



Receiver operating characteristics of PCWP. **A**, LAP cutoff: 12 mm Hg, AUC = 0.989. **B**, LAP cutoff: 15 mm Hg, AUC = 0.996.

demonstrating good concordance between PCWP and LAP. In addition, in contrast to the observation of Waltson and Kendall,[12] we find that the slight discrepancy between the 2 methods do not increase at higher levels of pressure. In fact, as shown in Figure 1, the correlation coefficient for LAP and PCWP measurements, as well as the variance of bias were essentially equivalent at pressure values up to and over 25 mm Hg.

To investigate whether eliminating the temporal factor would further improve the agreement between the LAP and PCWP recordings, in 51 cases, double registrations of the 2 pressures were performed, one with a time delay up to 5 minutes and another without any time difference (beat-to-beat simultaneous recordings). Not unexpectedly, analysis of the 2 data sets revealed that simultaneous measurements resulted in an even stronger correlation as well as narrower limits of agreement between the 2 methods. This observation may partly explain the considerable discrepancy reported in previous studies on that issue. Furthermore, as derived by the analysis of the simultaneous recordings, measurements of PCWP yield a modest but systematic overestimation of the LAP, whereas no significant difference was observed in the nonsimultaneous data set. The finding of a positive bias of PCWP compared with LAP is concordant with previous observations reported by Luchsinger and coworkers[7] as well as Ankeney,[19] who similarly described a common overestimation of LAP by wedge pressure measurement. A physiological explanation of this phenomenon has been proposed by Rivera-Estrada and coworkers,[20] who suggested that the PCWP is the venous pressure just distal to the pulmonary capillaries and the observed drop in pressure from the wedge position to the left atrium is due to postcapillary vascular resistance. Another physiological factor influencing the relation between the 2 pressure measurements might be the effect of alterations in LA compliance due to long-standing elevations of LAP. According to Hirakawa and coworkers,[21] the compliance of the left atrium decreases, in a nonlinear fashion, at higher LAPs, as estimated by wedge. Changes in compliance might influence the transmission of pressure waves and thus the correlation between PCWP measurements and direct LAP. In a report by Nishimura and colleagues[22] on a very limited number (17) of patients with MS, PCWP was shown to significantly overestimate the transmitral gradient, by 53%, as compared with when direct LAP measurement was used (bias: 3.3 ± 3.5 mmHg). However, in our much larger cohort, the difference in the transmitral gradient calculated using the 2 pressure measurements was barely discernable with only marginal overestimtaion of the transmitral gradient by PCWP (bias ± SD 0.3 ± 2.4 mm Hg).

Even in case of a good agreement between the mean pressures by the 2 methods, *a* and *v* waves might not be accurately transmitted from the left atrium to the arterial side of pulmonary capillaries. In the present study, PCWP traces displayed venous pulse waves similar in form to those recorded in the left atrium. Peak values of the *a* and *v* waves by the 2 measurements showed a strong correlation and fairly good agreement, although with much wider limits of agreement, as compared with the mean pressure values.

Based on the above results, we propose that, for the most part, the large disparities between PCWP and LAP measurements described in previous reports most probably result from methodological rather than physiological factors. It has been shown previously that the PCWP curves show a temporal delay as compared with LAP because of the time needed for waveform transmission through the lung veins.[23] Furthermore, a dampening of the transmitted LA pressure waves occurs.[24] The use of fluid-filled catheters instead of high fidelity manometertipped catheters for pressure measurement might introduce additional error. The increasing degree of error with higher wedge pressures reported in the single large-scale, retrospective study is most likely explained by nonsimultaneously performed measurements with larger pressure values naturally varying within a wider range over time.

It may be argued whether the reported limits of agreement, even if being substantially narrower as compared with previous studies, could favor the use of PCWP for estimation of LAP. To investigate the clinical value of using PCWP as an index of LAP, the predictive ability of PCWP was assessed using ROC curve analyses. For an optimal PCWP cutoff value of 14 mm Hg, the diagnostic potential of PCWP for identifying LAP >12 mm Hg was nearly 100%. Using the currently proposed limit of >15 mm Hg for elevated LAP, setting the cutoff for PCWP at 15 mm Hg or 16 mm Hg yielded essentially identical diagnostic ability. Thus, especially in case of near-normal LAP pressures, a slight overestimation of LAP when using PCWP measurements should be considered, but even in this case, PCWP provides a reliable index of the LAP.

To identify any nonmethodological confounders of the relationship between the 2 invasive measurements, multiple regression analysis was performed. Testing HR, LVEF, CO, SVR, RVSP, and PVR as independent variables in the model, we identified no statistically significant predictor.

Limitations

One criticism of this report might be that the study population comprised a very homogenous group of patients with pure MS. However, MS provides an optimal model for studying the concordance between the investigated 2 measurements under pathological hemodynamic conditions, as the pulmonary vascular changes in this setting do not solely affect the pulmonary venous tree but, in many cases, influence the arterial bed as well. In view of the practical importance of measuring PCWP in patients with elevated LAP, our cohort provides a representative sample.

In the clinical and diagnostic context, the accurate assessment of the LAP is essential, especially when it is at the near-normal pressure ranges. Although our cohort included a sufficient number of patients with near-normal LAP, investigating the agreement between the 2 methods in a large number of subjects with normal PCWP values would be of special importance. A lack of comorbidities in our patients limits the possibility of detecting conditions that may alter the relationship of PCWP and LAP.

Conclusions

Our results indicate that PCWP measurements are in good agreement with the actual LAP measured by direct catheterization of the left atrium. Although the 2 pressures are not identical, our results do support the current approach using PCWP as an estimate for LAP. Importantly, as indicated by the presented data, the relation of the 2 pressure measures is not affected by elevated wedge pressure. Although the described limits of agreement do not allow interchangeability of the 2 measurements, especially in a near-normal LAP range, from a clinical point of view, PCWP provides an accurate diagnostic tool that identifies patients with elevated LAP with high sensitivity and specificity.

Disclosures

The authors take responsibility for all aspects of the reliability and freedom from bias of the data presented and their discussed interpretation.

Conflict of interest: None.

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