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Echocardiographic Doppler Assessment of Pulmonary Capillary Wedge Pressure in Surgical Patients with Postoperative Circulatory Shock and Acute Lung Injury

Bélaïd Bouhemad, M.D., Armelle Nicolas-Robin, M.D.,* Alain Benois, M.D.,† Sacha Lemaire, M.D.,* Jean-Pierre Goarin, M.D.,‡ Jean-Jacques Rouby, M.D., Ph.D.§*

Background: **In cardiac patients, pulmonary capillary wedge pressure (PCWP) is estimated using color M-mode Doppler study of left ventricular filling and Doppler tissue imaging. The goal of this study was to assess whether echocardiography accurately estimates PCWP in critically ill patients.**

Methods: **Sixty ventilated patients admitted for septic shock and acute lung injury were prospectively studied using simultaneously transesophageal echocardiography and pulmonary artery catheterization. Initial PCWP values and their changes measured invasively were compared to initial values and corresponding changes of early diastolic velocity of mitral annulus displacement measured by Doppler tissue imaging (Ea), flow propagation velocity of early diastolic mitral inflow measured by color M-mode Doppler (Vp), and their respective ratio to early mitral inflow velocity (E) measured by conventional Doppler: E/Ea and E/Vp. Relations between E/Ea, E/Vp, and PCWP were prospectively tested in 20 additional patients.**

Results: **E/Ea and E/Vp gave a rough estimate of initial PCWP values with mean biases of 0.4 ± 2.2 and 0.1 ± 2.9 mmHg, respectively. Receiving operating characteristic curves demonstrated that an E/Ea of 6 or greater is an accurate predictor of a PCWP of 13 mmHg or greater and that an E/Ea of 5.4 is a good predictor of a PCWP of 8 mmHg or less. Changes in PCWP were significantly correlated to changes in E/Ea (Rho = 0.84, $P < 0.0001$).**

Conclusions: **In patients with postoperative circulatory shock and acute lung injury, transesophageal echocardiography estimates noninvasively PCWP. However, echocardiographic estimation of PCWP may not be accurate enough for adjusting therapy.**

BEDSIDE assessment of cardiorespiratory status is a critical issue in surgical patients with postoperative circulatory shock and/or acute lung injury. Indeed, the evaluation of cardiac function, pulmonary hemodynamics, and pulmonary capillary pressure has direct diagnostic and therapeutic implications. In patients with high-permeability–type pulmonary edema, the determination of pulmonary capillary pressure and cardiac output is of critical importance because these two parameters are essential determinants of the amount of fluid accumulating in the alveolointerstitial space.[1–3] Recently, it was demonstrated that the rapid and early normalization of venous oxygen saturation is associated with an improvement in the survival of patients with severe sepsis and septic shock, outlining the importance of cardiorespiratory monitoring.[4] Introduced in clinical practice more than 20 yr ago, the pulmonary artery catheter remains the most popular means for continuous monitoring of cardiac index, mixed venous oxygen saturation, pulmonary artery pressure, and pulmonary capillary wedge pressure (PCWP). However, its insertion is an invasive procedure associated with iatrogenic complications[5–8] that may compromise the final outcome.[9] As a consequence, any noninvasive method for estimating PCWP, if accurate, would be of interest.

In nonsedated, spontaneously breathing cardiac patients, Doppler indices derived from mitral[10] and pulmonary venous flows[11,12] obtained using transthoracic echocardiography have been shown to be acceptable estimations of PCWP. More recently, the flow propagation velocity of early mitral inflow measured by color M-mode Doppler and the ratio between the early mitral inflow measured by conventional Doppler and the displacement of the mitral annulus measured by tissue Doppler imaging[13–15] have been shown to accurately reflect PCWP in cardiac patients.

Surgical patients with postoperative circulatory shock may differ from cardiac patients in several ways: Preload, afterload, and cardiac function can rapidly vary depending on pharmacological support and ventilatory settings; inspiratory and expiratory phases of positive pressure ventilation are associated with cyclic changes of mitral and pulmonary venous flows; increases in heart rate can impair the separate evaluation of early and late diastolic mitral inflows; and segmental wall abnormalities may be present in the absence of ischemic heart disease. These complex cardiorespiratory interactions preclude an extrapolation of the correlations found in cardiac patients to critically ill patients. In addition, the transthoracic approach appears to be of limited value in patients with surgical dressings and chest tubes whose lungs are mechanically ventilated. The transesophageal approach appears quite attractive in heavily sedated, critically ill patients with postoperative surgical complications because it is easy to perform and provides high-quality images of the cardiac chambers that do not have artifacts

* Chef de Clinique Assistant en Anesthésie et Réanimation, † Interne en Anesthésie et Réanimation, ‡ Praticien Hospitalier en Anesthésie et Réanimation, § Professeur d' Anesthésie et Réanimation, Director of the Surgical Intensive Care Unit.

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Address reprint requests to Prof. Rouby: Réanimation Chirurgicale Pierre Viars, Departement d' Anesthésie-Réanimation, Groupe Hospitalier Pitié-Salpêtrière, 47 Boulevard de l' Hôpital, 75651 Paris Cedex 13, France. Address electronic mail to: jean-jacques.rouby@psl.ap-hop-paris.fr or jjrouby.pitie@invivo.edu. Individual article reprints may be purchased through the Journal Web site, www.anesthesiology.org.

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caused by the presence of intrapulmonary gas. The aim of this study was to assess whether PCWP measured from a pulmonary artery catheter could be accurately predicted using echocardiographic indices derived from transesophageal conventional Doppler and tissue Doppler imaging in a series of critically ill noncardiac patients with various forms of postoperative circulatory shock and acute lung injury.

Materials and Methods

Patient Population

Sixty consecutive patients admitted to the Surgical Intensive Care Unit of la Pitié-Salpêtrière Hospital (Paris, France) were prospectively included in the study after institutional review board approval. All patients required insertion of a pulmonary artery catheter and transesophageal echocardiography for diagnostic and therapeutic purposes. In our intensive care unit, an extensive hemodynamic evaluation is routinely performed in each patient with circulatory shock and/or severe hypoxemia resulting from acute lung injury, and no informed consent was required from the patients' next of kin. Exclusion criteria were esophageal and gastric pathology, cervical spine instability, lack of sinus rhythm, right or left bundle branch block, left ventricular systolic dysfunction, and presence of a significant mitral pathology. Patients with a history of coronary artery disease were not included in the study if they had significant segmental wall motion abnormalities detected on preoperative echocardiography.

During the study period, all patients were anesthetized with a continuous intravenous infusion of 5 $\mu g \cdot kg^{-1} \cdot h^{-1}$ fentanyl and 0.1 $mg \cdot kg^{-1} \cdot h^{-1}$ midazolam. All patients were ventilated using controlled mechanical ventilation (Cesar Ventilator, Taema, France). Tidal volume and respiratory rate were adjusted by the clinician in charge of the patient to achieve arterial carbon dioxide tension ($Paco_2$) values between 30 and 50 mmHg. An inspiratory time of 33% and a fraction of inspired oxygen (Fio_2) of 1 were maintained throughout the study period. If possible, positive end-expiratory pressure was discontinued or at least reduced to 5 cm H_2O during the acquisition of hemodynamic and echocardiographic data. Each patient underwent simultaneous Doppler echocardiographic and pulmonary artery catheter studies.

Hemodynamic Measurements

All patients were monitored using a fiberoptic thermodilution pulmonary artery catheter allowing the continuous monitoring of mixed venous oxygen saturation and the repetitive measurement of cardiac output using the semicontinuous thermodilution technique (CCO/Svo_2/VIP TD catheter) and a radial or femoral arterial catheter. During the study period, cardiac output, systemic arterial pressure, right atrial pressure, pulmonary artery pressure, PCWP, and airway pressure were continuously recorded using a BIOPAC MP100 system (Biopac System, Goleta, CA) for later analysis. The wedge position of the pulmonary artery catheter was verified by obtaining characteristic changes in the arterial pressure waveform and oxygen saturation (oxygen saturation $> 95\%$) following balloon inflation. Fluid-filled transducers were positioned at the mid axillary line and connected to the different lines of the pulmonary artery catheter. All recordings were reviewed by an investigator who was unaware of the echocardiographic data and were averaged over five cardiac cycles at the end-expiratory period.

Echocardiographic Studies

All patients were lying in the supine position. A complete transesophageal echocardiography was performed using an HP-SONOS 5500 (Hewlett-Packard, Andover, MA). Images were stored digitally on magneto-optical disks (Hewlett-Packard) for later playback and analysis. Transesophageal echocardiographic data were obtained using standard views and techniques.

The pulmonary venous flow was recorded by pulsed Doppler guided by color Doppler with the sample volume placed 0.5–1 cm into the left pulmonary vein. The mitral inflow was recorded by pulsed Doppler with the sample volume placed at the mitral valve tips (fig. 1A). A color M-mode Doppler image of left ventricular filling inflow in early diastole was obtained with an aliasing velocity set between 50 and 75% of velocity of early mitral (E) filling.[16] The mitral inflow was recorded 4 cm into the left ventricle cavity from the plane of the mitral valve. Velocities of mitral annulus (fig. 1B) were recorded by a tissue Doppler imaging program with a 5-mm sample volume placed at the lateral corner of the mitral annulus[13] rather than at the septal corner as previously recommended.[17] The ultrasound beam was placed as parallel as possible to the mitral or pulmonary venous filling flows. All pulsed and color M-mode Doppler were recorded at a horizontal sweep speed of 100 mm/s.

Echocardiographic measurements were performed offline by an observer who had no knowledge of the clinical data and other hemodynamic measurements. All measurements were made at the end-expiratory period over five consecutive cardiac cycles. The mitral inflow velocity was analyzed for peak velocity of early (E) and late (A) filling (fig. 1a), duration of atrial contraction (dA), and deceleration time of E. Flow propagation velocity of early mitral inflow (Vp) was measured as the slope of the first isovelocity contour line on the recorded color M-mode Doppler image. The recording of pulmonary venous flow was analyzed for the peak velocity and the velocity–time integral of each of the systolic (VTIs) and diastolic (VTId) signals. Duration of pulmonary atrial

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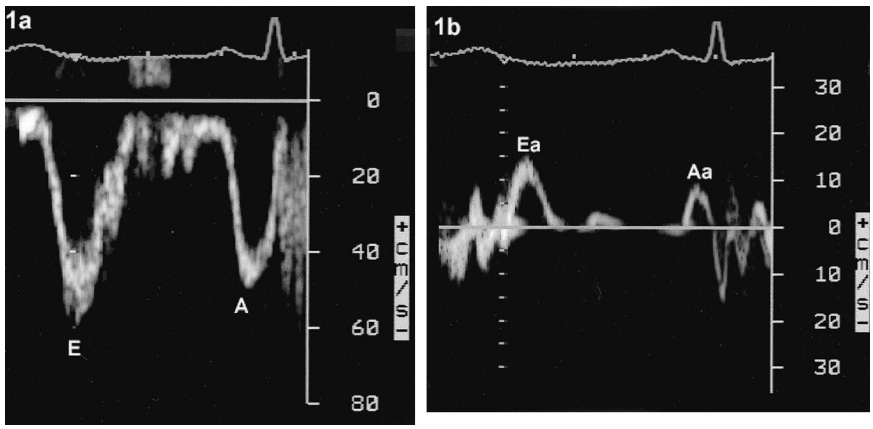

Fig. 1. (*a*) Mitral inflow recorded by pulsed Doppler. E = peak velocity of early mitral inflow; A = peak velocity of late mitral inflow. (*b*) Velocities of mitral annulus recorded by tissue Doppler imaging. Ea = early diastolic velocity of mitral annular displacement; Aa = late diastolic velocity of mitral annular displacement.

reversal flow (drA) was measured. The early diastolic (Ea) and late diastolic (Aa) velocities of mitral annular displacement were measured from the tissue Doppler imaging recording (fig. 1b).

All the following Doppler variables or calculated indices were correlated to PCWP: E/A, (dA − drA), dA/drA ratio, systolic fraction of pulmonary venous flow defined as VTIs/(VTIs + VTId),[18] E/Ea ratio[13] and E/Vp ratio.[16,17]

Left ventricular end-diastolic area (LVEDA) and left ventricular end-systolic area (LVESA) were measured on the short-axis transgastric view at the mid–papillary muscle level. Left ventricular systolic function was assessed by calculating the fractional area change defined as (LVEDA − LVESA)/LVEDA. Left ventricular systolic dysfunction was defined as a fractional area change of less than 50%. Left ventricular diastolic dysfunction was defined as an Ea/Aa of 1 or less with an Ea of less than 10 cm/s.

Assessment of Echocardiographic Measurements for Predicting Therapy-induced Changes of Pulmonary Capillary Wedge Pressure

In 35 patients with persistent shock and/or hypoxemia, the right ventricular catheter was maintained for 48–96 h, and additional echographic measurements were performed following various therapeutic interventions such as fluid depletion or dobutamine administration. According to the same protocol described above, changes of PCWP were compared to changes of echocardiographic indices.

Assessment of Reproducibility of Echocardiographic Measurements

Intraobserver and interobserver reproducibilities for analysis of E, Ea, and VP and their ratios E/Ea and E/Vp were assessed in 10 randomly selected patients. The recorded echocardiographic data were analyzed by two different observers and twice by the same observer at two different time intervals. Reproducibility, expressed as percentage of error, was calculated as the difference between the two sets of measurements divided by the mean value of the measurement.

Statistical Analysis

All data are expressed as mean ± SD. Normality of the distribution of data was assessed by a Kolmogorov-Smirnov test. Correlations between echocardiographic indices and PCWP were made using linear regression analysis (r) for data showing a normal distribution and by means of Spearman correlation rank analysis (Rho) for data without normal distribution.

The best-fitted equations obtained in the first 60 patients were prospectively tested in 20 additional patients. The Bland-Altmann method[19] was used to compare actual PCWP with echocardiographic estimation of PCWP calculated from the equations obtained in the first 60 patients. Receiving operating characteristic curves of these best-correlated echographic indices were constructed to predict the existence of a PCWP of 13 mmHg or greater or 8 mmHg or less.

For data showing a normal distribution, comparison between initial and prospective populations was made using a Student *t* test or a chi-square test. For data without normal distribution, comparison between initial and prospective populations was performed using a Mann–Whitney test or a Fisher exact test.

Statistical analysis was performed using NCSS 6.0 software (Statistical Solutions Ltd., Cork, Ireland), and the statistical significance level was set at 0.05.

Results

Population Characteristics

The characteristics of the initial population are presented in table 1. Most of the patients had undergone major surgical procedures. Circulatory shock was present in 90% of the patients and was mainly of septic origin. Acute respiratory distress syndrome (ARDS)–acute lung injury was present in 76% of the patients, and the overall mortality rate was 32%. Impaired left ventricular systolic function was present in 32% of the patients. More than 90% of the patients had echographic evidence of left ventricular diastolic impairment defined as an Ea/Aa of 1 or less with an Ea of less than 10 cm/s. Left ventricular diastolic dysfunction was related to impaired

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Table 1. Clinical Characteristics of the Patients

	Initial Population (n = 60)	Test Population (n = 20)	Statistical Significance
Age, yr	61 ± 15	58 ± 14	NS
Sex ratio, M/F	47/13	17/3	NS
Outcome, D/S	19/41	5/15	NS
ARDS/ALI	—	—	—
n	32/14	10/3	NS
%	53/23	50/15	NS
Shock, %	92	95	NS
Septic	38	16	NS
Cardiogenic	5	0	NS
Septic and cardiogenic	11	3	NS
Hemorrhagic	1	0	NS
Norepinephrine, %	75	85	NS
Epinephrine, %	7	0	NS
Dobutamine, %	12	5	NS
Type of surgery	—	—	—
Abdominal surgery	18	5	NS
Neurosurgery	1	2	NS
Vascular surgery	30	8	NS
Medical	5	1	NS
Multiple trauma	6	4	NS

Data are presented as mean ± SD or median. Statistical significance was tested with a chi-square test for parametrical parameters or a Fisher exact test when needed.

ALI = acute lung injury; ARDS = acute respiratory distress syndrome; D/S = deceased/survivors; M/F = male/female; NS = not significant.

left ventricular systolic dysfunction, age, or a history of coronary artery disease and/or arterial hypertension. All the patients tolerated the decrease in positive end-expiratory pressure to 5 cm H_2O without desaturation. Hemodynamic and echocardiographic characteristics of the patients are presented in tables 2 and 3. No significant difference was found between the initial population and the test population.

A complete transesophageal echocardiographic study including measurements on standard views and Doppler measurements of mitral inflow and mitral annular displacement was possible in all patients. Pulmonary venous flow could be analyzed in 93% of the patients. The color M-mode Doppler image of flow propagation velocity of early mitral inflow could be obtained in only 80% of the patients.

Correlations between Echocardiographic Indices and PCWP

The interobserver and intraobserver reproducibilities of echocardiographic measurements are shown in table 4. Doppler tissue imaging parameters were measured with excellent reproducibility in contrast to the measurement of Vp in color M-mode Doppler images, which appeared poorly reproducible between the two observers and between two sets of measurements performed by the same observer.

The coefficients characterizing the significant correlations found between PWCP and several echocardiographic Doppler parameters are shown in table 5. In all patients, a significant but weak correlation was found between PCWP and E/A ratio and between PCWP and E/Vp. However, these correlations improved in the subgroups of patients with impaired left ventricular systolic function and/or a distended left ventricle. In patients with left ventricular systolic dysfunction, a weak but significant correlation was found between PCWP and the systolic fraction of pulmonary venous flow, whereas the correlation between PCWP and E/Vp became clinically relevant. As shown in figure 2, the correlation between E/Ea and PCWP was statistically significant including patients with tachycardia and/or left ventricular systolic dysfunction. In patients with tachycardia (heart rate > 100 beats/min), no statistically significant correlation was found between PCWP and E/A, E/Vp, and the systolic fraction of pulmonary venous flow. No relations were found between PCWP and Vp, dA − drA, and dA/drA.

Table 2. Hemodynamic Characteristics of the Patients

	Initial Population (n = 60)	Test Population (n = 20)	Statistical Significance
SAP, mmHg	121 ± 24	124 ± 17	NS
DAP, mmHg	59 ± 19	65 ± 9	0.057
MAP, mmHg	79 ± 17	82 ± 15	NS
Heart rate, beats/min	95 ± 19	98 ± 22	NS
CI ($l \cdot min^{-1} \cdot m^{-2}$)	3.3 ± 1.3	3.8 ± 1.2	NS
SI ($ml \cdot m^{-2}$)	35.5 ± 12.6	39.5 ± 15.3	NS
SPAP, mmHg	34 ± 7	35 ± 7	NS
DPAP, mmHg	17 ± 4	19 ± 4	NS
MPAP, mmHg	25 ± 8	25 ± 5	NS
RAP, mmHg	9 ± 3	9 ± 4	NS
PCWP, mmHg	11 ± 4 (4.5–22)	11 ± 3 (6–17)	NS
SVRI ($dyn \cdot s^{-1} \cdot cm^{-5} \cdot m^2$)	1940 ± 796	1708 ± 643	NS
PVRI ($dyn \cdot s^{-1} \cdot cm^{-5} \cdot m^2$)	354 ± 163	363 ± 188	NS

Data are presented as mean ± SD. Statistical differences between initial population and test population were tested using a Student *t* test for unpaired data.

CI = cardiac index; DAP = diastolic arterial pressure; DPAP = diastolic pulmonary arterial pressure; MAP = mean arterial pressure; MPAP = mean pulmonary arterial pressure; NS = not significant; PCWP = pulmonary capillary wedge pressure; PVRI = pulmonary vascular resistance index; RAP = right atrial pressure; SAP= systolic arterial pressure; SI = stroke index; SPAP = systolic pulmonary arterial pressure; SVRI = systemic vascular resistance index.

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Table 3. Echocardiographic Characteristics of the Patients

Variables	Initial Population (n = 60)	Adequate Doppler Imaging (%)	Test Population (n = 20)	Adequate Doppler Imaging (%)	Statistical Significance
Left ventricular function	—	—	—	—	—
Patients with impaired systolic function	19 (32%)	—	5 (25%)	—	NS
FAC, %	33 ± 12	—	35 ± 11	—	NS
LVEDA, $cm^2 \cdot m^{-2}$	15 ± 4	—	11 ± 3	—	NS
Ea/Aa < 1 + Ea < 10 $cm \cdot s^{-1}$	19 (100%)	—	5 (100%)	—	NS
Patients with normal systolic function	41 (68%)	—	15 (75%)	—	NS
FAC (%)	65 ± 9	—	61 ± 5	—	NS
LVEDA ($cm^2 \cdot m^{-2}$)	10 ± 3	—	12 ± 4	—	NS
Ea/Aa < 1 + Ea < 10 $cm \cdot s^{-1}$	36 (88%)	—	13 (87%)	—	NS
Ea/Aa > 1 + Ea > 10 $cm \cdot s^{-1}$	5 (12%)	—	2 (13%)	—	NS
Mitral inflow	—	—	—	—	—
E ($cm \cdot s^{-1}$)	60 ± 23	100	57 ± 21	100	NS
A ($cm \cdot s^{-1}$)	56 ± 21	100	66 ± 22	100	0.035*
E/A ratio	1.2 ± 0.6	100	0.9 ± 0.5	100	NS
DT, msec	150 ± 52	95	131 ± 41	95	NS
Vp ($cm \cdot s^{-1}$)	43 ± 16	80	42 ± 14	80	NS
E/Vp	1.6 ± 0.6	80	1.6 ± 0.6	80	NS
dA, msec	120 ± 33	100	119 ± 36	100	NS
Pulmonary venous flow	—	—	—	—	—
SFP, %	56 ± 16	93	58 ± 16	85	NS
drA, msec	84 ± 28	90	86 ± 32	75	NS

Data are presented as mean ± SD. Statistical differences between initial and test populations were tested using a Student *t* test for unpaired data* or a Mann–Whitney test.

A = peak velocity of atrial mitral flow; drA = duration of pulmonary atrial reversal flow; DT = deceleration time of early mitral flow; E = peak velocity of early mitral flow; FAC = fractional area change; LVEDA = left ventricular end diastolic area; SFP = systolic fraction of pulmonary venous flow; Vp = flow propagation velocity of early mitral inflow.

Correlations between Variations of Echocardiographic Indices and Variations of PCWP

As shown in figure 3A, the correlation between variations of measured PCWP variations of E/Ea ratio and estimated PCWP was statistically significant and of clinical relevance. As shown in figure 3B, the mean bias between variation of measured PCWP from the pulmonary artery catheter and PCWP derived from Doppler tissue imaging and conventional Doppler according to the formula $PCWP_{Ea}$ = 0.97 E/Ea + 4.34 was of 0.5 mmHg with a precision of 2.0 mmHg.

Estimation of PCWP in the Test Population

As shown in figure 4, the mean bias between PCWP measured from the pulmonary artery catheter and PCWP derived from Doppler tissue imaging and conventional Doppler according to the formula $PCWP_{Ea}$ = 0.97 E/Ea + 4.34 was of 0.4 mmHg with a precision 2.2 mmHg, whereas the mean bias between PCWP measured from the pulmonary artery catheter and PCWP derived from conventional Doppler according to the formula $PCWP_{Vp}$ = 3.91 E/Vp + 4.92 was 0.1 mmHg with a precision of 2.9 mmHg. No relations were found between PCWP and Vp, dA − drA, and dA/drA. Receiving operating characteristic curves for predicting PCWP of 13 mmHg or greater or 8 mmHg or less from E/Ea and E/Vp are shown in figure 5: E/Ea was the most accurate index, with areas under the curve of 0.98 for estimating PCWP of 13 mmHg or greater and 0.80 for estimating PCWP of 8 mmHg or less.

Table 4. Reproducibility of Echocardiographic Measurements

—	Interobserver Error (%)	Intraobserver Error (%)
E ($cm \cdot s^{-1}$)	5 ± 6	4 ± 6
Vp ($cm \cdot s^{-1}$)	21 ± 17	11 ± 9
Ea ($cm \cdot s^{-1}$)	5 ± 3	4 ± 2
E/Vp	21 ± 16	12 ± 6
E/Ea	9 ± 9	7 ± 5

Data are presented as mean ± SD.

E = peak velocity of early mitral flow; Ea = early diastolic velocity of mitral annular displacement; Vp = flow propagation velocity of early mitral inflow.

Discussion

This study, performed in critically ill noncardiac patients with postoperative acute lung injury and circulatory shock, shows that PCWP as well as changes resulting from therapeutic interventions can be estimated using transesophageal echocardiography and Doppler tissue imaging. It confirms and extends similar results found in spontaneously breathing cardiac patients using transthoracic echocardiography.[10–16] A PCWP of 13 mmHg or greater is predicted by an E/Ea of 7 or greater with a sensitivity of 86% and a specificity of 92%. In contrast, E/Ea appears less accurate for predicting PCWP values of 8 mmHg or less. Because one of the critical

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Table 5. Statistical Correlations Existing between Different Echocardiographic Indices and PCWP According to Left Ventricular Function, Heart Rate, and Left Ventricular Preload in the Initial Population

Patient Echocardiographic Indices	E/A Ratio (r)	E/Vp Ratio (r)	SFP % (r)	Ea, cm · s^{-1} (ρ)	E/Ea Ratio (ρ)
Initial population (n = 60)	0.57	0.56	0.41	0.42	0.77
Left ventricular systolic function	—	—	—	—	—
Abnormal (n = 19)	0.61	0.75	0.56	0.56	0.88
Normal (n = 41)	0.57	0.36	0.30	0.32	0.73
Heart rate	—	—	—	—	—
≥100 beats/min (n = 19)	0.28	0.39	0.11	0.66	0.80
<100 beats/min (n = 41)	0.64	0.59	0.53	0.29	0.78
Left ventricular end diastolic area	—	—	—	—	—
>15 cm^2/m^2 (n = 11)	0.64	0.89	0.42	0.47	0.81
≤15 cm^2/m^2 (n = 49)	0.51	0.37	0.32	0.38	0.77

The statistical correlation between the echocardiographic indices and PCWP is expressed as correlation coefficient (r) or Spearman correlation coefficient (ρ). A = peak velocity of atrial mitral flow; E = peak velocity of early mitral flow; Ea = early diastolic velocity of mitral annular displacement; SFP = systolic fraction of pulmonary venous flow; Vp = Flow propagation velocity of early mitral inflow.

therapeutic issues in patients with acute lung injury is to keep pulmonary capillary pressure as low as possible to limit pulmonary edema formation, the possibility of measuring PCWP noninvasively appears to be of clinical interest. However, the echocardiographic estimation of PCWP may not be accurate enough for an acute adjustment of therapy.

Respiratory Consequences of Increased Pulmonary Capillary Pressure in Acute Lung Injury

In acute lung injury, any increase in hydrostatic pulmonary capillary pressure increases the amount of plasma traversing the damaged alveolocapillary barrier.[1,20–23] Increasing left atrial pressure from 3 to 13 mmHg induces an eightfold increase in pulmonary lymphatic flow in dogs with acid-aspiration lung injury as compared to a fourfold increase in animals with normal lungs.[3] In patients with ARDS, the transmicrovascular flux of albumin increases proportionally to the microvascular hydrostatic pulmonary pressure calculated from PCWP and pulmonary artery pressure.[24,25] Although there is no randomized clinical trial reporting an improved mortality of patients with ARDS treated according to pulmonary artery catheter–guided management,[5] some clinical trials have suggested a clinical benefit in minimizing left cardiac filling pressures.[26,27] According to the difficulties of clinically predicting PCWP in the critically ill,[26–28] Swan-Ganz catheters are frequently inserted in patients with ARDS to measure PCWP and reduce as much as possible the "hemodynamic" component of pulmonary edema.[2,20] PCWP tightly correlated with left atrial pressure in patients with ARDS[29] and chronic obstructive pulmonary disease.[30,31] In a minority of patients treated with high positive end-expiratory pressure or who had high levels of intrinsic pressure, PCWP reflects alveolar pressure rather than left atrial pressure.[32] Such a situation can be easily recognized at the bedside when ventilation-induced changes in PCWP largely exceed ventilation-induced changes in pulmonary artery pressure.[33] Because PCWP reflects left atrial pressure, it underestimates pulmonary capillary pressure, the microvascular pressure determining the transvascular flux of plasma in patients with acute lung injury. The effective pulmonary capillary pressure can be estimated from the PCWP either by analyzing the decay of the pulmonary artery pressure following balloon inflation[34] or by wedging the pulmonary artery catheter after deflating the balloon.[35] Even if the accurate evaluation of effective capillary pulmonary pressure remains difficult at the bedside, a logical approach for the clinician re-

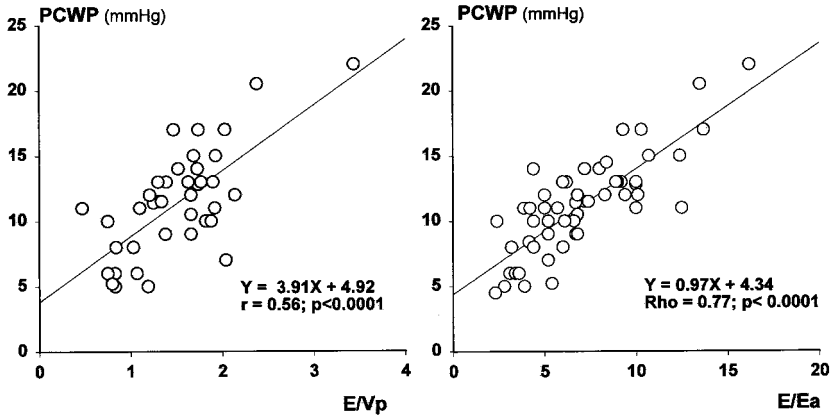

Fig. 2. Correlations existing between pulmonary capillary wedge pressure (PCWP) and color M-mode echocardiography and Doppler tissue imaging indices in the initial population: (*Left*) PCWP (mmHg) is plotted against E/Vp ratio. E = peak velocity of early mitral inflow; Vp = velocity of flow propagation of early mitral inflow. A significant correlation was found between PCWP and E/Vp ratio. (*Right*) PCWP (mmHg) is plotted against E/Ea. Ea = early diastolic velocity of mitral annular displacement. A significant and clinically relevant correlation was found between PCWP and E/Ea ratio.

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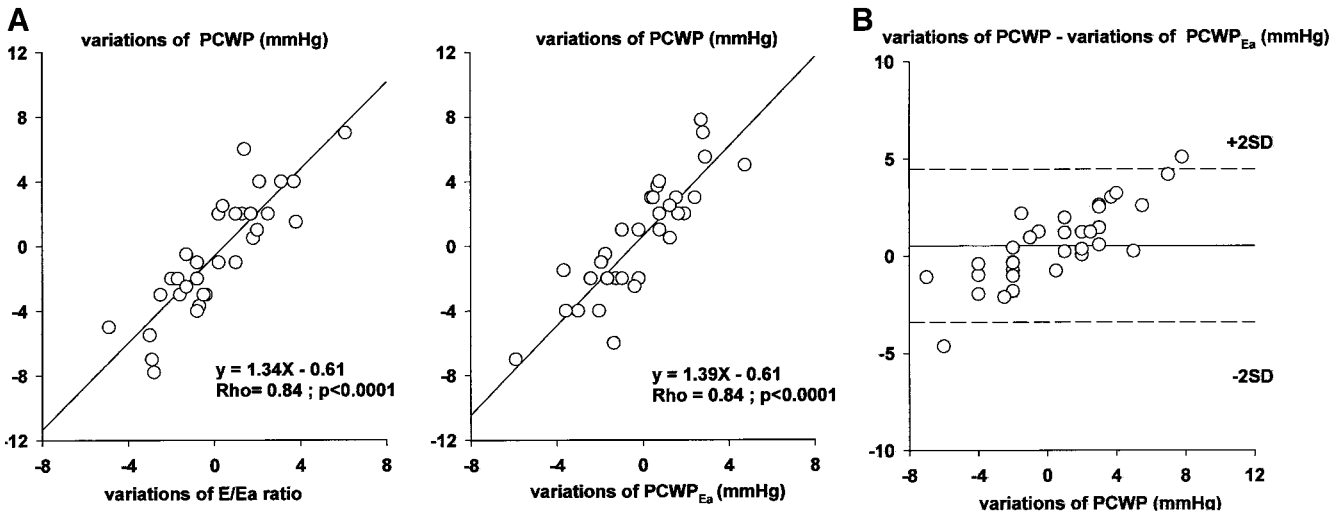

Fig. 3. (*A*) Correlations existing between variations of pulmonary capillary wedge pressure (PCWP) and variations of E/Ea ratio in 35 patients of the initial population. (*Left*) Variations of PWCP measured from the Swan-Ganz catheter and resulting from various therapeutic interventions are plotted against the corresponding variations of E/Ea ratio. E = peak velocity of early mitral inflow; Ea = early diastolic velocity of mitral annular displacement. A significant correlation exists between variations of PCWP and variations of E/Ea ratio. (*Right*) Variations of measured pulmonary capillary wedge pressure (mmHg) are plotted against variations of PCWP estimated from E/Ea ratio ($PCWP_{Ea}$). $PCWP_{Ea}$ was calculated using the following formula: $PCWP_{Ea}$ = 0.97 E/Ea + 4.34. A significant and clinically relevant correlation was found between variations of PCWP and variations of $PCWP_{Ea}$. (*B*) Bias and precision of variations of PCWP estimated from Doppler tissue imaging in 35 patients of the initial population. Differences between variations of PCWP measured from the pulmonary artery catheter and PCWP estimated from E/Ea ratio ($PCWP_{Ea}$). According to the correlation shown in figure 2, $PCWP_{Ea}$ was calculated using the following formula: $PCWP_{Ea}$ = 0.97 E/Ea + 4.34. The mean bias was 0.5 mmHg. The dotted lines represent 2 SDs (precision of the bias, according to the Bland-Altman method).

mains to decrease PCWP to the lowest values preserving systemic perfusion.[36]

Echocardiographic Assessment of Pulmonary Capillary Wedge Pressure in Patients with Acute Lung Injury and Circulatory Shock

Ninety percent of the patients included in the current study had an impaired left ventricular relaxation, and 30% had a transient and reversible alteration of left ventricular systolic function related to sepsis and inflammation.[37,38] The majority of the patients were older than 50 yr; half had a history of arterial hypertension and had undergone major vascular surgical procedures for aortic dissection, thoracoabdominal aortic aneurysms, multiple great vessel stenosis, and aortocoronary grafts; and one third had postoperative septic shock–induced left ventricular systolic dysfunction, all conditions known to be associated with left ventricular diastolic dysfunction.[39–41] All patients had been admitted to the surgical intensive care unit for major postoperative complications such as severe bronchopneumonia, acute acalculous cholecystitis, or ischemic colitis, and the majority had circulatory shock, multiple organ failure, and acute lung injury. Because large fluid loading was administered

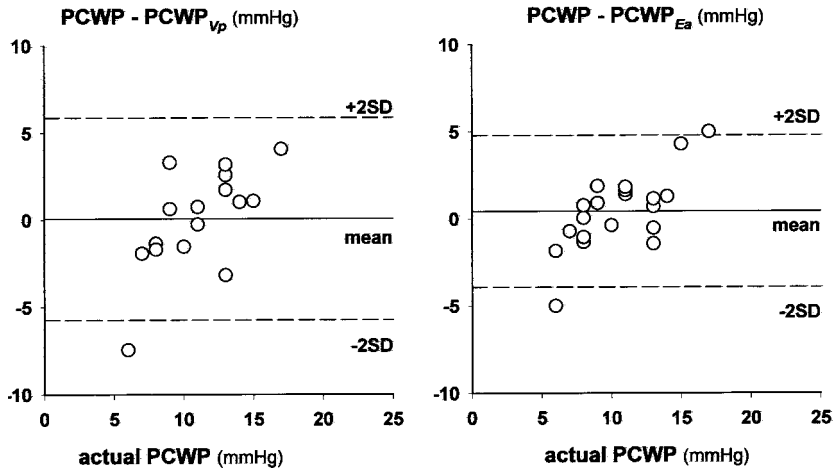

Fig. 4. Bias and precision of pulmonary capillary wedge pressure (PCWP) estimated from conventional Doppler, color M-mode Doppler, and Doppler tissue imaging in the test population. (*Left*) Differences between PCWP measured from the pulmonary artery catheter and PCWP estimated from color M-mode Doppler ($PCWP_{Vp}$). According to the correlation shown in figure 2, $PCWP_{Vp}$ was calculated using the following formula: $PCWP_{Vp}$ = 3.91 E/Vp + 4.92. The mean bias was 0.1 mmHg. The dotted lines represents 2 SDs (precision of the bias, according to the Bland-Altman method). (*Right*) Differences between PCWP measured from the pulmonary artery catheter and PCWP estimated from Doppler tissue imaging ($PCWP_{Ea}$). According to the correlation shown in figure 2, $PCWP_{Ea}$ was calculated using the following formula: $PCWP_{Ea}$ = 0.97 E/Ea + 4.34. The mean bias was 0.4 mmHg. The dotted lines represents 2 SDs (precision of the bias, according to the Bland-Altman method).

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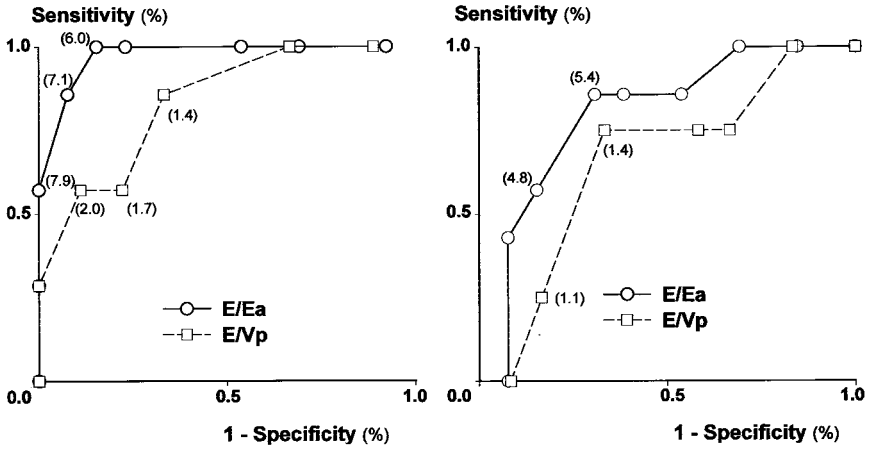

Fig. 5. Receiving operating characteristic curves assessing the ability of Doppler tissue imaging and color M-mode Doppler for predicting a pulmonary capillary wedge pressure (PCWP) of 13 mmHg or greater or 8 mmHg or less in the test population. (*Left*) Receiving operating characteristic curve assessing the ability of E/Ea and E/Vp for predicting a PCWP of 13 mmHg or greater. Areas under the curve (AUC) were, respectively, 0.97 for E/Ea and 0.83 for E/Vp. The numbers in bracket indicate the value of the cutoff. (*Right*) Receiving operating characteristic curve assessing the ability of E/Ea and E/Vp for predicting a PCWP of 8 mmHg or less. AUCs were, respectively, 0.80 for E/Ea and 0.67 for E/Vp. The numbers in bracket indicate the value of the cutoff.

to patients with postoperative septic shock and impaired left ventricular relaxation, a "hemodynamic" worsening of the respiratory status was a permanent risk imposing the monitoring of cardiorespiratory parameters.

A noninvasive approach to diastolic failure and elevated left ventricular filling pressures was developed 10 yr ago and was based on the assessment of transmitral flow using Doppler echocardiography.[10] However, difficulties in making a distinction between normal and pseudonormal patterns of mitral flow[42,43] led to the use of more preload-independent indices of left ventricular diastolic relaxation derived from color M-mode Doppler and Doppler tissue imaging.[42] Several studies performed in spontaneously breathing cardiac patients demonstrated that by combining these new indices with the early diastolic peak velocity of transmitral flow, PCWP could be accurately predicted.[13–16,44] To understand why these rather sophisticated echocardiographic indices are accurate estimations of left cardiac filling pressures in critically ill noncardiac surgical patients, a brief review of the factors influencing each term of the equations is necessary.

The peak velocity of the early mitral flow (E) increases with preload[17] and decreases with impaired relaxation[45] and tachycardia.[14] In healthy volunteers, E is the best predictor of an elevated PCWP.[17] In spontaneously breathing cardiac patients as in noncardiac ventilated critically ill patients with circulatory shock, tachycardia and impaired left ventricular relaxation are frequently observed acting as confounding factors on E, which is no longer an accurate predictor of elevated left cardiac filling pressure.[13–16,44] Mitral annulus early diastolic velocity (Ea) is mainly influenced by left ventricular relaxation.[46,47] In animals and patients with normal left ventricular relaxation, Ea is preload-dependent,[17,48,49] and the ratio E/Ea is not an accurate predictor of PCWP.[17] However, because Ea is quasi–preload-independent in patients or experimental animals with impaired left ventricular relaxation,[48,49] the ratio E/Ea corrects for the influence of relaxation on transmitral E and becomes an accurate predictor of PCWP.[13,15,44] The correlation remains strong in patients with sinus tachycardia.[14]

The same reasoning can be conducted regarding the early diastolic left ventricular flow propagation velocity (Vp) as measured using color M-mode Doppler echography. Vp has been demonstrated to be a preload-independent index of ventricular relaxation.[50,51] As a consequence, the ratio E/Vp corrects for the influence of impaired relaxation on transmitral E and has been shown to be an accurate predictor of PCWP.[16,17] A spatiotemporal velocity map of diastolic filling can be obtained with color M-mode by placing a scan line from the mid left atrium through the mitral valve to the apex of the left ventricle.[52] The entire inflow tract must be visualized from the left atrium across the mitral valve into the left ventricle throughout the entire diastolic filling period. Then, a velocity map with an early wave and an atrial wave is obtained, and flow propagation velocity of early mitral inflow (Vp) is measured as the first isovelocity line. Vp was adequately obtained in 80% of our patients, and a significant correlation was found between E/Vp and PCWP. However, the correlation coefficient ($r = 0.56$) was less than previously reported.[16,17] As shown in figure 3, PCWP could be estimated with a moderate bias of 0.1 mmHg. However, limits of agreement were large, ranging from −6 to + 6 mmHg, and resulted in nonreliable estimation of PCWP. The discrepancy between our results and the results previously reported may be related to difficulties in obtaining an adequate velocity profile in some of our patients. Indeed, E/Vp was best correlated with PCWP in patients with a left ventricular dilatation ($r = 0.89$) and a heart rate of less than 100 beats/min ($r = 0.59$), conditions allowing an optimal alignment on transmitral flow. Reproducibility of measurements was smaller in the current study than previously reported,[51,53] very likely because a transesophageal rather than a transthoracic approach was used. The transesophageal approach limits the possibilities of alignment with the mitral flow in contrast to the transthoracic approach, in which the hand can move freely, offering

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more possibilities of alignment. In addition, during mechanical ventilation, the positive intrathoracic pressure may interfere with intraventricular gradients and velocity profiles, rendering interpretation of aliasing velocities and alignment with mitral flow difficult. As a consequence, in mechanically ventilated patients, color M-mode Doppler derived from transesophageal echocardiography appears less accurate than tissue Doppler imaging for estimating PCWP.

Critique of Method and Limitations

Noninvasive diagnosis of elevated left ventricular filling pressures can be performed at the bedside in critically ill patients using transesophageal echocardiography and Doppler tissue imaging. However, an accurate estimation of PCWP is more problematic. In the current study, PCWP could be estimated with respective biases of 0.4 and 0.1 mmHg for E/Ea ratio and E/Vp. However, limits of agreement were large, ranging from −6 to +6 mmHg for E/Vp and from −4 to + 5 mmHg for E/Ea, rendering the estimation of PCWP hazardous in an individual patient, particularly when PCWP was less than 8 mmHg. When severe hypoxemia resulting from an extended injury of the alveolocapillary barrier is present, the pulmonary capillary pressure is the most important factor determining filtration rate and the amount of pulmonary edema. Because one of the means for limiting pulmonary edema is to lower left atrial pressure as much as possible, the echocardiographic estimation of PCWP may not be accurate enough for optimizing therapy. In addition, two limitations inherent to transesophageal echocardiography should be outlined. First, it does not allow continuous monitoring, which may be required for patients with circulatory shock and acute lung injury. Second, performing and interpreting transesophageal echocardiography requires a long training period aimed at acquiring the necessary knowledge and skills.[54,55] As a consequence, it is unlikely that the majority of intensive care unit teams may permanently have an expert in echocardiography who is able to perform repetitive noninvasive echocardiographic evaluations of PCWP.

It should be pointed out that the good correlation found between PCWP and E/Ea may not be observed in younger patients with a normal left ventricular relaxation. Indeed, in such patients, the mitral annulus early diastolic velocity is preload-dependent and may vary in the same direction as the mitral flow early diastolic velocity. As a consequence, E/Ea may not be a good predictor of PCWP as previously observed in healthy volunteers.[17] Additional studies are required to identify the appropriate echocardiographic index that could be predictive of PCWP in this category of patients.

In surgical patients with postoperative circulatory shock and impaired left ventricular relaxation, transesophageal echocardiography using Doppler tissue imaging allows the identification of patients with elevated PCWP. When acute lung injury is associated with circulatory shock, E/Ea appears to be an acceptable predictor of the actual value of PCWP and can be used at the bedside for guiding therapeutic means aimed at lowering pulmonary capillary pressure and limiting the amount of pulmonary edema.

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