Doppler Echocardiographic Indices Are Specific But Not Sensitive to Predict Pulmonary Artery Occlusion Pressure in Critically Ill Patients Under Mechanical Ventilation

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OBJECTIVES: The objective of this study was to prospectively evaluate the ability of transthoracic echocardiography to assess pulmonary artery occlusion pressure in mechanically ventilated critically ill patients.

DESIGN: In a prospective observational study.

SETTING: Amiens University Hospital Medical ICU.

PATIENTS: Fifty-three mechanically ventilated patients in sinus rhythm admitted to our ICU.

INTERVENTION: Transthoracic echocardiography was performed simultaneously to pulmonary artery catheter.

MEASUREMENTS AND MAIN RESULTS: Transmitral early velocity wave recorded using pulsed wave Doppler (E), late transmitral velocity wave recorded using pulsed wave Doppler (A), and deceleration time of E wave were recorded using pulsed Doppler as well as early mitral annulus velocity wave recorded using tissue Doppler imaging (E′). Pulmonary artery occlusion pressure was measured simultaneously using pulmonary artery catheter. There was a significant correlation between pulmonary artery occlusion pressure and lateral ratio between E wave and E′ (E/E′ ratio) ($r = 0.35$; $p < 0.01$), ratio between E wave and A wave (E/A ratio) ($r = 0.41$; $p < 0.002$), and deceleration time of E wave ($r = -0.34$; $p < 0.02$). E/E′ greater than 15 was predictive of pulmonary artery occlusion pressure greater than or equal to 18 mm Hg with a sensitivity of 25% and a specificity of 95%, whereas E/E′ less than 7 was predictive of pulmonary artery occlusion pressure less than 18 mm Hg with a sensitivity of 32% and a specificity of 81%. E/A greater than 1.8 yielded a sensitivity of 44% and a specificity of 95% to predict pulmonary artery occlusion pressure greater than or equal to 18 mm Hg, whereas E/A less than 0.7 was predictive of pulmonary artery occlusion pressure less than 18 mm Hg with a sensitivity of 19% and a specificity of 94%. A similar predictive capacity was observed when the analysis was confined to patients with EF less than 50%. A large proportion of E/E′ measurements 32 (60%) were situated between the two cut-off values obtained by the receiver operating characteristic curves: E/E′ greater than 15 and E/E′ less than 7.

CONCLUSIONS: In mechanically ventilated critically ill patients, Doppler transthoracic echocardiography indices are highly specific but not sensitive to estimate pulmonary artery occlusion pressure.

KEY WORDS: intensive care; pulmonary artery catheter; pulmonary artery occlusion pressure; transthoracic echocardiography

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BACKGROUND

Pulmonary artery occlusion pressure (PAOP) is a valuable variable for the diagnosis and management of critically ill patients, especially those with acute respiratory failure since PAOP allows determining or decline the cardiogenic origin (1, 2). PAOP is measured by right heart catheterization, an invasive procedure that is associated with a risk of serious complications (3). This has led to a decline in the use of this invasive technique in critically ill patients over the last decade (4, 5).

Transthoracic echocardiography (TTE) is a noninvasive technique widely used in the critical care unit for the assessment of critically ill patients with hemodynamic and respiratory failure (6–8). Several Doppler TTE indices have been used to predict PAOP. They are mainly based on analysis of transmitral flow and tissue Doppler imaging (TDI) of mitral annulus motion (9–15).

In nonsedated, spontaneously breathing cardiac patients, numerous studies have shown that TTE is able to successfully predict invasive PAOP (9–12). However, recent published studies question the ability of echocardiography to evaluate PAOP (16, 17). In addition, few studies have been performed in mechanically ventilated critically ill patients, and the predictive capacity of TTE has not been clearly established (13–15). Additionally, experimental and clinical studies have shown that positive pressure ventilation, especially positive end-expiratory pressure (PEEP), can overestimate PAOP measurements (18, 19). Teboul et al (19) proposed a method to correct the effect of PEEP on PAOP measurement: the transmission index (TI). To our knowledge, no study has evaluated the impact of TI-corrected PAOP (PAOP-TI) on the ability of TTE to estimate PAOP.

We therefore decided to evaluate the ability of TTE to assess PAOP and PAOP-TI in mechanically ventilated critically ill patients.

METHODS

Study Population

This prospective, observational study was performed between January 2015 and May 2016 in our ICU in a university hospital (Amiens, France). The study protocol was approved by the local independent ethics committee (CPP Nord Ouest II, Amiens, France). In line with French legislation, all patients (or their surrogates) were provided with information about the study and gave their informed consent to participate. We included all mechanically ventilated patients hospitalized in our ICU and fitted with a pulmonary artery catheter (PAC) in a context of respiratory or hemodynamic failure. The insertion of the PAC was decided by the attending physician when additional hemodynamic information was needed after echocardiography assessment. Exclusion criteria were age less than 18 years old, arrhythmia, severe mitral valve disease, and monophasic mitral flow.

All patients were under mechanical ventilation and deep sedation according to our protocol. Neuromuscular blocking agents was used only in case of spontaneous effort detection. PAOP measurement and TTE were performed simultaneously by two different investigators at the end-expiratory phase. Each measurement was performed blinded to the results of the other technique.

The following data were also recorded: age, gender, Simplified Acute Physiology Score (SAPS) II, the main reason for admission to ICU, and presence or absence of catecholamine infusion at the time of PAOP measurements.

PAC monitoring

A 6.0F PAC (Swan-Ganz Thermodilution PAC 131HF7; Edwards Lifesciences, Irvine, CA) was used for PAOP measurements. All patients underwent a chest radiograph after insertion of the PAC to confirm the correct position and the absence of complications. We also used the methodology described by Teboul et al (18) based on pressure waveforms to check that the tip of the catheter was situated in the third zone of West's classification. PAOP was determined following PAC balloon inflation at end-expiration from an average of three respiratory cycles. The recording was analyzed, and the TI (19) was calculated. TI is an index of the transmission of pressure from the alveolar compartment to the pulmonary veins and therefore reflects PAOP.

$$\mathrm{TI} = \Delta\mathrm{PAOP} / \Delta\mathrm{Palv}$$

ΔPAOP corresponds to the difference between maximum PAOP (PAOPmax) and minimum PAOP (PAOPmin) during a respiratory cycle.

ΔPalv corresponds to the difference between plateau pressure and total PEEP.

Corrected PAOP (PAOP-TI) was calculated as the difference between PAOP and the product of TI and total PEEP.

$$\text{PAOP-TI} = \text{PAOP} - (\text{total PEEP} * \text{TI})$$

Arterial blood pressure was measured invasively in all patients. All patients were monitored in the supine position with the mid-axillary line as zero reference.

TTE

Standard echocardiographic views were acquired using a Vivid S6 echocardiograph (GE Medical Systems, Milwaukee, WI). Mitral inflow pulsed-wave Doppler was obtained and transmitral early velocity wave recorded using pulsed wave Doppler (E), late transmitral velocity wave recorded using pulsed wave Doppler (A), and deceleration time of E wave (DTE) were then measured. Lateral early mitral annulus velocity wave recorded using TDI (E′) was obtained. The E/A and lateral E/E′ (E/E′) ratios were calculated. TTE was performed at the same time as the PAOP measurements by a highly experienced sonographer, blinded to the invasive PAOP measurements.

All TTE measurements were acquired in accordance with the European Association of Cardiovascular Imaging/American Society of Echocardiography task force's recommendations (20, 21).

Statistical analysis

The normality of the data distribution was checked by a Shapiro-Wilk test. Data are expressed as number (%) for categorical variables, mean (SDs) for normally distributed continuous variables, and median (interquartile range [IQR]) for nonnormally distributed variables.

The correlation between PAOP measurements and PAOP-TI and echocardiographic variables (E/A, E/E′, and DTE) was determined by calculating Pearson's coefficient. The ability of TTE (E/A, E/E′) to predict PAOP greater than or equal to 18 mm Hg was analyzed by receiver operating characteristic (ROC) curve analysis.

All statistical analyses were performed with MedCalc software (Version 12.0.4.0; MedCalc Software, Mariakerke, Belgium). The limit of statistical significance was *p* value of less than 0.05.

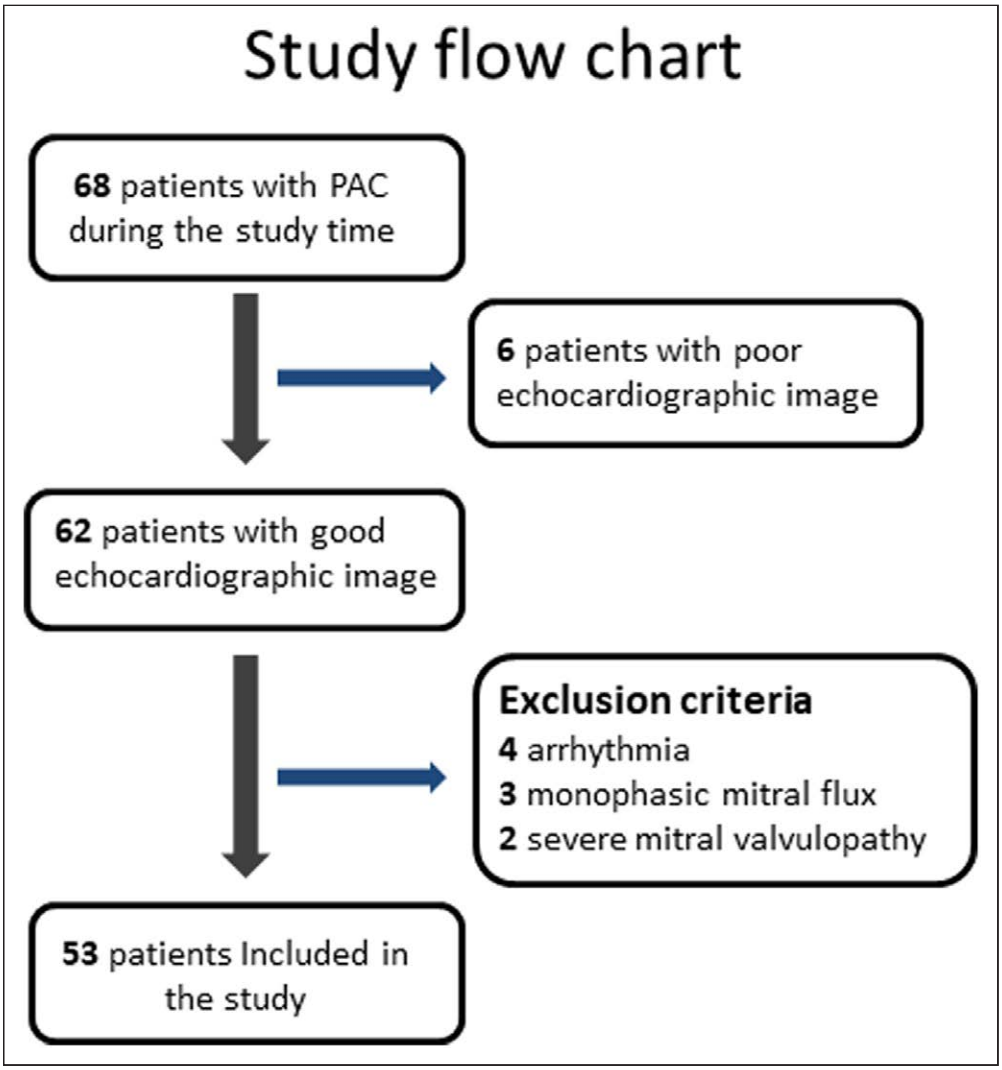

Figure 1. Study flow chart. PAC = pulmonary artery catheter.

RESULTS

Patient Characteristics

During the study period, 364 intubated patients presented circulatory or respiratory failure. A total of 68 sedated, mechanically ventilated patients received a PAC and were included (**Fig. 1**). The following patients were excluded: four patients with arrhythmia, three patients with monophasic mitral flow, and two patients with severe mitral valve disease. Mitral flow or mitral annulus velocity could not be measured in six patients due to poor echocardiographic images. A total of 53 patients were analyzed: 34 male patients (64%) with a median (IQR) age of 64 years (58–74 yr) and a mean SAPS II of 60 ± 23. The most common reason for ICU admission was pneumonia (*n* = 38; 72%). There were 22 patients (42%) with a history of coronary heart disease, of which four were admitted with acute coronary syndrome and four due to sudden death. Of the total, 30 patients had left ventricular (LV) EF less than 50% at the time of echocardiography was performed. Demographic characteristics and hemodynamic, respiratory, and echocardiographic data of the study population are summarized in **Table 1**.

Correlations Between PAOP and TTE Measurements

Fifty-three PAOP and TTE measurements were compared. A loose but significant correlation was demonstrated between PAOP measurements and E/E′ ($r = 0.35$; $p < 0.01$), E/A ($r = 0.41$; $p < 0.002$), and DTE ($r = –0.34$; $p < 0.02$) (**Fig. 2**).

The mean difference between PAOP and PAOP TI was 4.1 (1.7–10) with mean TI of 11 ± 5.8. Fifty-three PAOP-TIs were calculated and were compared with TTE. A loose but significant correlation was demonstrated between PAOP-TI measurements and E/E′ ($r = 0.33$; $p < 0.02$), E/A ($r = 0.42$; $p < 0.002$), and DTE ($r = –0.38$; $p < 0.009$) (Fig. 2).

Capacity of the E/E′ ratio to predict PAOP and PAOP-TI

Sixteen PAOP measurements (30%) were greater than or equal to 18 mm Hg. The area under the ROC curve (95% CI) of E/E′ ratio to predict PAOP greater than or equal to 18 mm Hg was 0.6 (0.45–0.73). An E/E′ greater than 15 was predictive of PAOP greater than or equal to 18 mm Hg with a sensitivity of 25% and a specificity of 95% (**Fig. 3**), whereas an E/E′ less than 7 was predictive of PAOP less than 18 mm Hg with a sensitivity of 32% and a specificity of 81%. A similar predictive capacity was observed when the analysis was confined to patients with EF less than 50% (**Supplemental Fig. 1**, Supplemental Digital Content 1, http://links.lww.com/CCM/F927; **legend**, Supplemental Digital Content 2, http://links.lww.com/CCM/F928).

A large proportion of E/E′ measurements 32 (60%) was situated between the two cut-off values obtained by the ROC curves: E/E′ greater than 15 and E/E′ less than 7 (**Fig. 4**).

Eleven PAOP-TI measurements (21%) were greater than or equal to 18 mm Hg. The area under the ROC curve (95% CI) of E/E′ ratio to predict PAOP-TI greater than or equal to 18 mm Hg was 0.57 (0.42–0.71). E/E′ greater than 15 was predictive of PAOP-TI greater than or equal to 18 mm Hg with a sensitivity of 27% and a specificity of 93%, whereas E/E′ less than 7 was predictive of PAOP-TI less than 18 mm Hg with a sensitivity of 43% and a specificity of 73%. A similar predictive capacity was observed when the analysis was confined to patients with EF less than 50%.

Capacity of E/A to Predict PAOP and PAOP-TI

The area under the ROC curve (95% CI) for the ability of E/A to detect PAOP greater than or equal to 18 mm Hg was 0.69 (0.55–0.81). E/A greater than 1.8 was predictive of PAOP greater than or equal to 18 mm Hg with a sensitivity of 44% and a specificity of 95% (Fig. 3), whereas E/A less than 0.7 was predictive of PAOP less than 18 mm Hg with a sensitivity of 19% and a specificity of 94%. A similar predictive capacity was observed for PAOP-TI and when the analysis was confined to patients with EF less than 50% (Supplemental Fig. 1, Supplemental Digital Content 1, http://links.lww.com/CCM/F927; legend, Supplemental Digital Content 2, http://links.lww.com/CCM/F928). A large proportion of E/A measurements 31 (59%) was situated between the two cut-off values obtained by the ROC curves: E/A greater than 1.8 and E/A less than 0.7 (Fig. 4).

Capacity of E/E′ Lateral and E/A to Predict PAOP Greater Than or Equal to 18

Using both echocardiographic cut-off obtained with the ROC curves, E/E′ greater than 15 and E/A greater than 1.8 were predictive of PAOP greater than or equal to 18 mm Hg with a sensitivity of 25% and a specificity of 97%. While E/E′ less than 7 and E/A less than 0.7 were predictive of PAOP less than 18 mm Hg with a sensitivity of 8% and a specificity of 94%. These ROC curves were not statistically different.

TABLE 1.
Characteristics of the Study Population ($n = 53$)

Characteristics	Study Population ($n = 53$)
Male, *n* (%)	34 (64)
Age, yr, median (IQR)	64 (58–74)
Simplified Acute Physiology Score II, mean ± SD	60 ± 23
Admission, *n* (%)	
Septic shock/systemic inflammatory response syndrome	28 (53)
Cardiogenic shock	7 (13)
Hypovolemic shock	2 (4)
Respiratory failure	16 (30)
Indication for pulmonary artery catheter monitoring, *n* (%)	
Hemodynamic failure	30 (57)
Respiratory failure	23 (43)
Catecholamines, *n*(%)	
Norepinephrine	46 (88)
Dobutamine	13 (25)
Epinephrine	8 (15)
Norepinephrine doses, (ug/kg/min), median (IQR)	0.4 (0.12–1.0)
Surgery, *n* (%)	5 (9)
Heart rate, beats/min	104 ± 20
Mean arterial pressure, mm Hg	71 (66–84)
O_2 saturation, %, median (IQR)	97 (94–98)
Pao_2/Fio_2 ratio, median (IQR)	177 (131–255)
Mechanical ventilation	
Tidal volume, mL, median (IQR)	440 (400–500)
Positive end-expiratory pressure, cm H_2O, median (IQR)	8 (6–11)
Respiratory rate, cycle/min, median (IQR)	22 (20–26)
Plateau pressure, cm H_2O, median (IQR)	21 (18–25)
Fio_2, %, median (IQR)	50 (40-80)
Left ventricular ejection fraction, %, mean ± SD	44 ± 19
Cardiac output, L/min, median (IQR)	4.7 (3.3–6.5)
Mean pulmonary arterial pressure, mm Hg, median (IQR)	29 (22–33)

(*Continued*)

TABLE 1. (*Continued*).
Characteristics of the Study Population ($n = 53$)

Characteristics	Study Population ($n = 53$)
Systolic pulmonary arterial pressure, mm Hg, median (IQR)	40 (32–48)
PAOP, mm Hg, mean ± SD	15 ± 5
Transmission index-corrected PAOP, mm Hg, mean ± SD	11 ± 6
Right auricular pressure, mm Hg, mean ± SD	11 ± 4
Ratio between transmitral early velocity wave and late transmitral velocity wave recorded using pulsed wave Doppler, median (IQR)	1.1 (0.8–1.7)
Early mitral annulus velocity wave recorded using tissue Doppler imaging, cm/s, mean ± SD	8 ± 3
Ratio between transmitral early velocity wave and early mitral annulus velocity wave recorded using tissue Doppler imaging, median (IQR)	9.3 (6.4–12.1)
Deceleration times of the transmitral early velocity wave recorded using pulsed wave Doppler, ms, median (IQR)	154 (137–205)

IQR = interquartile range, PAOP = pulmonary artery occlusion pressure.

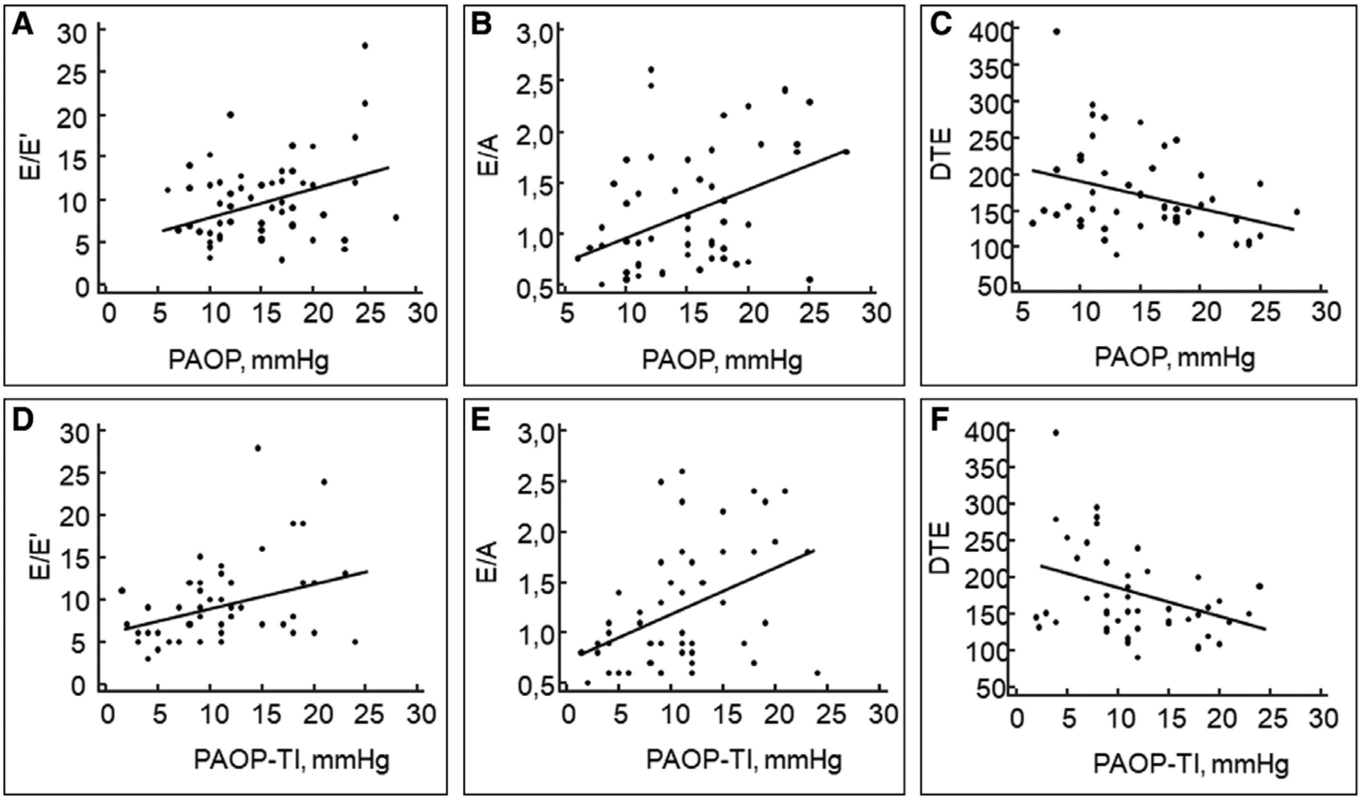

Figure 2. Correlation between pulmonary artery occlusion pressure (PAOP) and transmission index-corrected PAOP (PAOP-TI) and Doppler indices. **A**, Correlation between PAOP and E/E′ lateral ratio ($r = 0.35$; $p < 0.01$). **B**, Correlation between PAOP and E/A ratio ($r = 0.41$; $p < 0.002$). **C**, Correlation between PAOP and DTE ($r = -0.34$; $p < 0.02$). **D**, Correlation between PAOP-TI and E/E′ lateral ratio ($r = 0.33$; $p < 0.02$). **E**, Correlation between PAOP-TI and E/A ratio ($r = 0.42$; $p < 0.002$). **F**, Correlation between PAOP-TI and DTE ($r = -0.38$; $p < 0.009$). A = late transmitral velocity wave recorded using pulsed wave Doppler, DTE = deceleration time of the E wave, E = transmitral early velocity wave recorded using pulsed wave Doppler, E′ = early mitral annulus velocity recorded using tissue Doppler imaging, E/A ratio = ratio between E and A waves, E/E′ lateral ratio = ratio between E wave and E′.

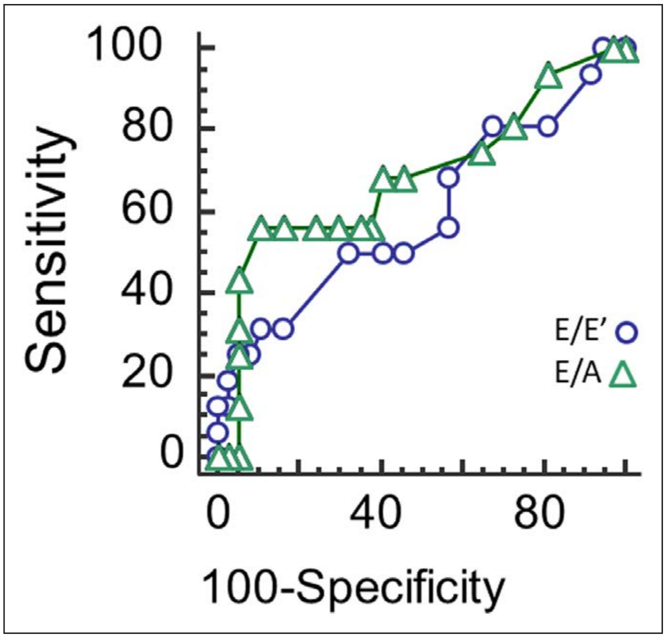

Figure 3. Receiver operating characteristic (ROC) curve of E/E′ and E/A to predict pulmonary artery occlusion pressure greater than or equal to 18 mm Hg. The area under the ROC curve (95% CI) of E/E′ and E/A were 0.6 (0.45–0.73) and 0.69 (0.55–0.81), respectively. E/A ratio = ratio between transmitral early velocity wave (E) and late transmitral velocity wave (A) recorded using pulse wave Doppler, E/E′ lateral ratio = ratio between transmitral early velocity wave (E) and early mitral annulus velocity wave (E′) recorded using tissue Doppler imaging.

DISCUSSION

This study demonstrated that E/E′ greater than 15 and/or E/A greater than 1.8 were highly specific to estimate PAOP and PAOP-TI greater than 18 mm Hg but with low sensitivity. E/A less than 0.7 and/or E/E′ less than 7 predicted a PAOP less than 18 mm Hg with high specificity; 60% and 59% of patients had PAOP between these cut-off values of E/E′ and E/A, respectively.

Doppler TTE indices have been used to assess PAOP, mainly based on analysis of transmitral flow and TDI of mitral annulus motion (9–15). Transmitral flow is composed of two waves, E wave determined by the early diastolic transmitral pressure gradient and A wave determined during atrial contraction. E/A ratio has been shown to be closely correlated with PAOP measured invasively in patients with heart disease and low LVEF (20–24). DTE is the interval from the peak of E velocity to baseline. An increase in left atrial filling pressure results in a higher initial E velocity and a shorter deceleration time, as result of the higher driving pressure across the mitral valve (23).

TDI is a technique that measures myocardial velocities. The E′ has been shown to be a relatively preload-independent measure of myocardial relaxation in patients with myocardial disease (12). When Doppler transmitral flow is combined with TDI, lateral E/E′ ratio has been correlated with PAOP measured invasively (22). These Doppler TTE indices have been shown to reliably predict invasive PAOP in nonsedated, spontaneously breathing cardiac patients. However, this predictive capacity has not been demonstrated in mechanically ventilated critically ill patients.

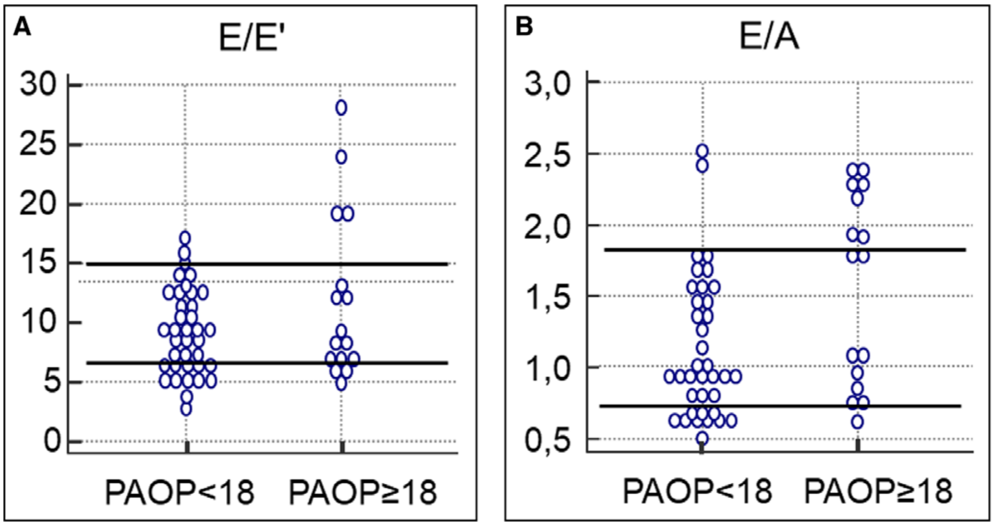

Figure 4. E/E′ and E/A distribution according to pulmonary artery occlusion pressure (PAOP) greater than or equal to 18 mm Hg. The two continuous *lines* correspond to the cut-off values of E/E′ and E/A to predict PAOP, respectively. **A**, A large part of the E/E′ measurements 32 (60%) is between the cut-off values "*gray zone*" E/E′ greater than 15 and E/E′ less than 7. **B**, A large part of the E/A measurements 31 (59%) is between the cut-off values E/A greater than 1.8 and E/A less than 0.7. E/A ratio = ratio between transmitral early velocity wave (E) and late transmitral velocity wave (A) recorded using pulse wave Doppler, E/E′ lateral ratio = ratio between transmitral early velocity wave (E) and early mitral annulus velocity wave (E′) recorded using tissue Doppler imaging.

In the present study on mechanically ventilated critically ill patients, comparative analysis of PAOP and Doppler TTE indices revealed a loose but significant correlation and a poor ability to predict PAOP. PAOP-TI did not improve the predictive value of Doppler TTE indices. There is a large overlap between patients when E/E′ or E/A is used to rule out high PAOP. PAOP greater than or equal to 18 mm Hg can be detected with a high specificity of 95% but with very poor sensitivity. A large proportion of E/E′ and E/A measurements were situated between the two ROC cut-off values, corresponding to a "gray zone", limiting its utility in clinical practice. When we used the two E/E′ and E/A cutoffs obtained with the ROC curves, the specificity was close to 100% but with very poor sensitivity to predict elevated PAOP.

Dokainish et al (15), in a study on 50 ICU patients, reported that E/E′ greater than 15 had a sensitivity of 85% and a specificity of 88% to predict PAOP greater than 15 mm Hg, whereas in the group of the patients with EF less than 50%, E/E′ greater than 15 had a sensitivity of 92% and a specificity of 90% to predict PAOP greater than 15 mm Hg. They found 28% of E/E′ in the "gray zone" between 8 and 15. These better results can be explained by differences in the study populations. In this study, almost half of patients were not ventilated, and a large proportion of the patients were cardiac patients with a mean PAOP of 20.9 mm Hg, considerably higher than that observed in our patients. As indicated above, Doppler TTE indices have been shown to be able to reliably predict invasive PAOP in spontaneously breathing cardiac patients.

Combes et al (13) evaluated the ability of TTE and transesophageal echocardiography (TEE) to estimate PAOP in 23 mechanically ventilated critically ill patients. They showed that E/E′ greater than 7.5 had a sensitivity of 86% and a specificity of 81% to predict PAOP greater than or equal to 15 mm Hg, but less than half of the patients were studied by TTE (nine patients), and, as in the study by Dokainish et al (15), the PAOP cut-off was 15 mm Hg, which, in our opinion, does not have the same clinical applicability as 18 mm Hg. In the same study, a poorer correlation between Doppler indices and PAOP measurements was observed when the analysis was restricted to the patients with the highest PEEP.

The mechanism by which positive pressure mechanical ventilation can affect PAOP measurements can be explained as follows: PAOP is assumed to reflect LV end-diastolic pressure (LVEDP) (25). LVEDP is an intracavitary relative to extracavitary pericardial pressure. LV filling pressure (LVFP) is closely reflected by LVEDP at end-expiration when pericardial pressure is minimal. End-expiratory PAOP is therefore commonly used as a surrogate for LVFP. When PEEP is applied, the pericardial pressure is therefore greater than zero during the expiratory period, which can make PAOP significantly higher than LVFP (26, 27). Pinsky et al (28) demonstrated, in cardiac surgery patients, that PAOP measured after airway disconnection provided a better estimate of LVFP than PAOP when PEEP greater than 10 cm H_2O was used and that this overestimation of LVFP increases as PEEP increases. Teboul et al (19) proposed a method for estimating LVFP from PAOP measurements, without airway disconnection, consisting of an index of transmission (IT) of pressure from the alveolar compartment to the pulmonary veins and therefore to PAOP (IT = ΔPAOP/ΔPalv). In a mechanically ventilated patient with a level of applied PEEP, transmural LVEDP could then be estimated from PAOP and Palv measurements by subtracting the product of total PEEP and IT from the PAOP. This estimation is the PAOP-TI). We calculated the TI and PAOP-TI and found that corrections of PAOP by TI had no impact on the ability of Doppler TTE indices to estimate PAOP.

TEE has also been used to predict high PAOP in mechanically ventilated ICU patients. Vignon et al (29) showed, by ROC analysis of Doppler measurements obtained by TEE, that lateral E/E′ less than or equal to 8 had a sensitivity of 83% and a specificity of 88% to predict PAOP less than or equal to 18, whereas a mitral E/A ratio less than or equal to 1.4 had a sensitivity of 75% and a specificity of 100%. Boussuges et al (14) showed that E/A greater than 2 had a 100% positive predictive value for prediction of PAOP greater than or equal to 18 mm Hg.

We consider that TEE is a less invasive technique than PAC and presents a good predictive capacity for PAOP recording pulmonary venous flow (systolic fraction or S/D ratio) that could be used when PAOP cannot be established with a high degree of confidence by TTE.

Our study has several limitations. The number of patients is limited; however, to our knowledge, this study is one of the studies on this subject with the largest number of patients in the critical care setting. We did not include consecutive patients with either shock or respiratory failure, the intensivist in charge of the patient decided to introduce the PAC, which may induce a selection bias, then the sensitivity may have been underestimated. PAOP and TTE measurements were performed under optimal conditions, with sedation and temporary paralysis, in order to reduce patient-ventilator interactions to a minimum, but these conditions are not always possible in clinical practice. We do not study the ability of TTE assessment to track treatment induced variation of PAOP.

Only a small proportion of patients (30%) had elevated PAOP, in line with the results reported by other studies (29). This proportion also reflects the reality of a noncardiologic ICU population, in which sepsis is the predominant disease. Sepsis is known to be associated with real and relative hypovolemia, which could explain the low ventricular filling pressures observed in our population. Other TTE indices have been used to predict PAOP, including propagation velocity and pulmonary venous flow, were not evaluated in this study. Finally, TTE allows intermittent hemodynamic assessment at the bedside of the critically ill patient; this is a limitation of TTE compared with continuous monitoring tools.

CONCLUSIONS

In a population of mechanically ventilated ICU patients, in sinus rhythm, we demonstrated that Doppler TTE indices are highly specific but not sensitive to estimate PAOP.

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Each author meets the criteria for authorship credit set forth by the International Committee of Medical Journal Editors, as revised in 2013. Drs. Mercado, Maizel, Tribouilloy, and Slama contributed to conception and design. Drs. Mercado, Maizel, Marc, Beyls, Titeca-Beauport, Kontar, Riviere, Bonef, Soupison, and de Cagny contributed to data acquisition. Drs. Mercado, Maizel, and Slama contributed to data analysis. Drs. Mercado, Maizel, Tribouilloy, and Slama contributed to interpretation of data. All authors were involved in drafting the work, revising it critically for important intellectual content and in the final approval of the version submitted for publication.

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The study was approved by the local independent ethics committee (CPP Nord Ouest II, Amiens, France). In line with French legislation, all patients (or their surrogates) were provided with information about the study and gave their informed consent.

All authors have read the article and approved the content.

The data that support the findings of this study are available from the corresponding author upon reasonable request on Excel table.

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