

# Pulmonary Artery Acceleration Time Provides an Accurate Estimate of Systolic Pulmonary Arterial Pressure during Transthoracic Echocardiography

Kibar Yared, MD, Peter Noseworthy, MD, Arthur E. Weyman, MD, Elizabeth McCabe, MS, Michael H. Picard, MD, and Aaron L. Baggish, MD, *Boston, Massachusetts; Toronto, Ontario, Canada*

**Background:** Transthoracic echocardiographic estimates of peak systolic pulmonary artery pressure are conventionally calculated from the maximal velocity of the tricuspid regurgitation (TR) jet. Unfortunately, there is insufficient TR to determine estimated peak systolic pulmonary artery pressure (EPSPAP) in a significant number of patients. To date, in the absence of TR, no noninvasive method of deriving EPSPAP has been developed.

**Methods:** Five hundred clinically indicated transthoracic echocardiograms were reviewed over a period of 6 months. Patients with pulmonic stenosis were excluded. Pulsed-wave Doppler was used to measure pulmonary artery acceleration time (PAAT) and right ventricular ejection time. Continuous-wave Doppler was used to measure the peak velocity of TR ( $TR_{V_{max}}$ ), and EPSPAP was calculated as  $4 \times TR_{V_{max}}^2 + 10$  mm Hg (to account for right atrial pressure). The relationship between PAAT and EPSPAP was then assessed.

**Results:** Adequate imaging to measure PAAT was available in 99.6% of patients (498 of 500), but 25.3% (126 of 498) had insufficient TR to determine EPSPAP, and 1 patient had significant pulmonic stenosis. Therefore, 371 were included in the final analysis. Interobserver variability for PAAT was 0.97. There were strong inverse correlations between PAAT and  $TR_{V_{max}}$  ( $r = -0.96$ ), the right atrial/right ventricular pressure gradient ( $r = -0.95$ ), and EPSPAP ( $r = -0.95$ ). The regression equation describing the relationship between PAAT and EPSPAP was  $\log_{10}(EPSPAP) = -0.004 (PAAT) + 2.1$  ( $P < .001$ ).

**Conclusions:** PAAT is routinely obtainable and correlates strongly with both  $TR_{V_{max}}$  and EPSPAP in a large population of randomly selected patients undergoing transthoracic echocardiography. Characterization of the relationship between PAAT and EPSPAP permits PAAT to be used to estimate peak systolic pulmonary artery pressure independent of TR, thereby increasing the percentage of patients in whom transthoracic echocardiography can be used to quantify pulmonary artery pressure. (*J Am Soc Echocardiogr* 2011;24:687-92.)

**Keywords:** Pulmonary artery acceleration time, Right ventricular systolic pressure, Pulmonary artery pressure, Transthoracic echocardiography

Estimates of pulmonary artery pressure obtained using transthoracic echocardiography (TTE) are most commonly performed using continuous-wave Doppler to measure the maximum velocity of tricuspid regurgitation ( $TR_{V_{max}}$ ). This technique of pulmonary artery pressure quantification has been widely adopted for clinical and research purposes because it provides a noninvasive, direct estimate of right ventricular (RV) systolic pressure (RVSP) that correlates closely with invasive hemodynamic measurement.<sup>1</sup> In the absence of significant RV outflow tract obstruction, this measurement provides

From the Division of Cardiology, Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts (K.Y., P.N., A.E.W., E.M., M.H.P., A.L.B.); and the Division of Cardiology, The Scarborough Hospital – General Campus, University of Toronto Medical School, Toronto, Ontario, Canada (K.Y.).

Reprint requests: Kibar Yared, MD, Division of Cardiology, The Scarborough Hospital – General Campus, University of Toronto Medical School, 3050 Lawrence Avenue West, Toronto, ON M1P 2V5, Canada (E-mail: [kibaryared@yahoo.com](mailto:kibaryared@yahoo.com)).

0894-7317/\$36.00

Copyright 2011 by the American Society of Echocardiography.

doi:10.1016/j.echo.2011.03.008

an accurate means of obtaining estimated peak systolic pulmonary artery pressure (EPSPAP). However, this method of pulmonary artery pressure assessment is not feasible when TR is absent or trivial.

Alternative transthoracic echocardiographic methods for pulmonary artery pressure estimation have been proposed. These include the measurement of blood flow through an anatomic defect (ventricular septal defect,<sup>2,3</sup> patent ductus arteriosus,<sup>4</sup> or aortopulmonary shunt<sup>5</sup>), measurement of the peak systolic and end-diastolic pulmonic valve regurgitant velocity,<sup>6</sup> and measurement of the pulmonary artery acceleration time (PAAT).<sup>7</sup> PAAT measurement, previously shown to correlate with invasively measured mean pulmonary artery pressure (MPAP),<sup>7</sup> is a particularly attractive alternative to the  $TR_{V_{max}}$ -dependent method, because it does not rely on the presence of an anatomic defect or valvular regurgitation and is therefore measurable in the vast majority of individuals. Currently, measurement of PAAT cannot be used as an alternative to  $TR_{V_{max}}$  for the derivation of EPSPAP, because the relationship between these two parameters has not been described.

We hypothesized that PAAT would correlate strongly with  $TR_{V_{max}}$  and thus could be used to quantify EPSPAP. To address this hypothesis, we examined the relationship between PAAT and both  $TR_{V_{max}}$

**Abbreviations****EPSPAP** = Estimated peak systolic pulmonary artery pressure**MPAP** = Mean pulmonary artery pressure**PAAT** = Pulmonary artery acceleration time**RV** = Right ventricular**RVET** = Right ventricular ejection time**RVIRT** = Right ventricular isovolumic relaxation time**RVSP** = Right ventricular systolic pressure**SPAP** = Systolic pulmonary artery pressure**TR** = Tricuspid regurgitation**TR<sub>vmax</sub>** = Maximum velocity of tricuspid regurgitation**TTE** = Transthoracic echocardiography

and EPSPAP in a large unselected group of patients undergoing clinically indicated TTE.

**METHODS**

All aspects of this study were approved by our institutional review board (Partner's Human Research Committee). Five hundred (of a total of 9,200) clinically indicated transthoracic echocardiographic studies performed in the Massachusetts General Hospital echocardiography laboratory over a 6-month period (March to September 2009) were randomly selected for retrospective review. To arrive at this total number of participants, a research staff member who was blinded to patient age, gender, and study indication randomly selected 84 studies from each month of the study period for formal re-

pulsed-wave sample volume at the pulmonary valve annulus and not more proximally in the RV outflow tract to maximally align blood flow and Doppler interrogation. The same view was used to exclude pulmonic stenosis. PAAT, RVET, and heart rate were measured using the pulse-wave Doppler profile. PAAT was defined as the interval between the onset of systolic pulmonary arterial flow and peak flow velocity (Figure 2). RVET was defined as the interval between the onset of RV ejection to the point of systolic pulmonary arterial flow cessation. All values used for analysis represent the average of three consecutive cardiac cycles, with the exception of patients with atrial fibrillation, in whom five-beat averages were obtained. All measurements were performed by three study cardiologists (A.L.B., P.N., and K.Y.).

**Statistical Analysis**

Echocardiographic data are reported as mean  $\pm$  SD with the corresponding range of values. The correlation between PAAT and EPSPAP was examined using Pearson's or Spearman's method as appropriate for data distribution. Histograms of PAAT and EPSPAP were constructed and tested for normality using the Shapiro-Wilks test. Although both variables were found to have significant skew, transforming RVSP to its  $\log_{10}$  value fulfilled assumptions for linear regression. PAAT was then plotted against  $\log_{10}$ (RVSP), and linear regression was performed. Interobserver variability for PAAT was assessed using linear regression and Bland-Altman analysis for 50 random individuals from the cohort.

**RESULTS**

Among this cohort of randomly selected clinical studies, pulsed-wave Doppler imaging of the main pulmonary artery was sufficient to measure PAAT in 99.6% of patients (498 of 500). In contrast, 25.2% (126 of 500) did not have sufficient TR to measure TR<sub>vmax</sub>. One individual was excluded from analysis because of the presence of significant pulmonary valve stenosis. As such, 371 individuals were retained in the final analysis.

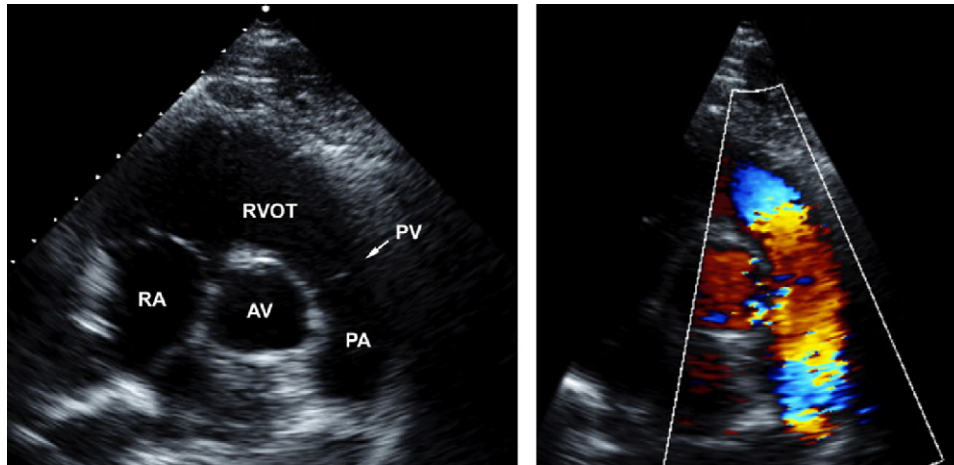
In the final cohort ( $n = 317$ ), the mean age was  $63 \pm 17$  years (range, 17–96 years), and 51% were men (189 of 371). The mean heart rate during TTE was  $70 \pm 10$  beats/min (range, 42–112). Atrial fibrillation or flutter was present in 8% of patients (30 of 371), while the remaining 92% were in normal sinus rhythm. Indications for TTE included assessment of left ventricular function, congestive heart failure, or cardiomyopathy ( $n = 108$ ); assessment of suspected or established valvular heart disease ( $n = 89$ ); dyspnea of uncertain etiology ( $n = 53$ ); chest pain of uncertain etiology ( $n = 46$ ); fever of uncertain etiology ( $n = 23$ ); syncope ( $n = 13$ ); and other ( $n = 39$ ). In 11% of studies (41 of 371), ordering providers specifically requested assessments of RVSP or pulmonary artery pressure.

Group mean values and ranges for all TR-derived and pulmonary artery-derived flow parameters are shown in Table 1. The interobserver variability for PAAT measurement was 0.97. By Bland-Altman analysis, the interobserver variability was no more than  $\pm 7.5$  msec (Figure 3). The group mean TR<sub>vmax</sub> of  $282 \pm 65$  cm/sec (range, 150–480 cm/sec) yielded a mean EPSPAP of  $44 \pm 16$  mm Hg (range, 19–102 mm Hg). The mean PAAT was  $124 \pm 34$  msec (range, 43–186 msec), and the mean RVET was  $362 \pm 51$  msec (range, 216–580 msec). There were strong inverse correlations between PAAT and TR<sub>vmax</sub> ( $r = -0.96$ ), the right atrial/RV pressure gradient ( $4 \times \text{TR}_{\text{vmax}}^2$ ;  $r = -0.95$ ), and EPSPAP ( $r = -0.95$ )

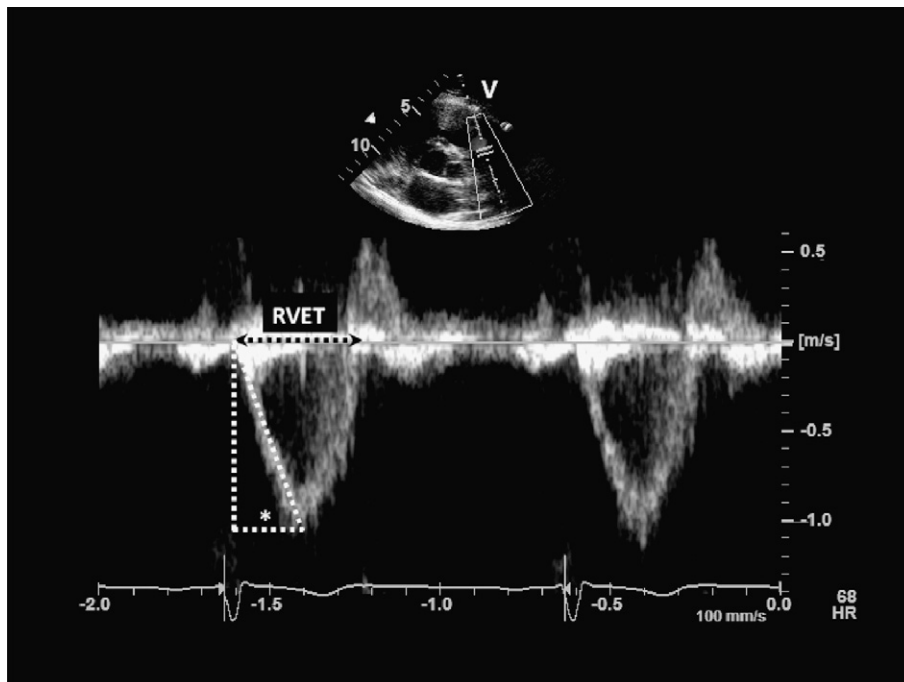
view. Once an a priori established enrollment total of 500 patients was reached, study enrollment ended. As detailed below, TR<sub>vmax</sub>, PAAT, and RV ejection time (RVET) were measured on each study. Studies that did not contain adequate imaging for the measurements of each of these variables were excluded from the final analysis. In addition, patients with significant pulmonic valvular stenosis, as defined by a continuous-wave peak jet velocity  $\geq 2$  m/sec across the pulmonic valve, were excluded from analysis. Studies were performed using commercially available echocardiographic systems (iE33, Philips Medical Systems, Andover, MA; Vivid 7, GE Healthcare, Milwaukee, WI).

To assess the reproducibility of our findings, a separate validation cohort was then constructed. Specifically, 100 consecutive patients referred for clinically indicated TTE who had measurable TR signals (50 from the Massachusetts General Hospital echocardiography laboratory [Boston, MA] and 50 from the Scarborough Hospital echocardiography laboratory [Toronto, ON, Canada]) were prospectively assessed in a manner identical to that used during creation of the derivation cohort.

All measurements were made offline (Xcelera; Philips Medical Systems). Heart rate and rhythm were assessed on the final image of each study. TR<sub>vmax</sub> was defined as the maximal velocity of the TR jet measured using continuous-wave Doppler in the echocardiographic view that yielded the most complete TR Doppler profile. EPSPAP was calculated using the modified Bernoulli equation:  $4 \times \text{TR}_{\text{vmax}}^2 + 10$  mm Hg (to account for right atrial pressure<sup>1,8</sup>). The use of 10 mm Hg as an estimate of right atrial pressure yields similar correlates with catheter-based estimates of RVSP as a method using the Doppler gradient plus mean jugular venous pressure.<sup>8</sup> Pulsed-wave and continuous-wave Doppler interrogation of the proximal pulmonary artery was then performed in the parasternal short-axis view with the sample volume placed at the annulus of the pulmonary valve (Figures 1 and 2). We chose to place the



**Figure 1** Transthoracic echocardiogram in the parasternal short-axis view (left) and with color Doppler interrogation of the pulmonary artery (PA) (right). AV, Aortic valve; PV, pulmonic valve; RA, right atrium; RVOT, RV outflow tract.



**Figure 2** Pulsed-wave Doppler interrogation of the pulmonary artery. RVET is measured from the onset of RV ejection to that of zero flow. PAAT is the interval from the onset of RV ejection to peak flow velocity (asterisk).

(Figure 4). Adjustment of PAAT for RVET ( $r = -0.89$ ) and heart rate ( $r = -0.91$ ) did not lead to stronger correlations with EPSPAP. The relationship between PAAT and  $\log_{10}(\text{EPSPAP})$  is shown in Figure 5. The highly significant ( $P < .001$ ) regression equation describing this relationship was  $\log_{10}(\text{EPSPAP}) = -0.004(\text{PAAT}) + 2.1$ .

In the validation cohort ( $n = 100$ ), the mean age was  $69 \pm 17$  years, 54% were men, and atrial fibrillation was present in 9%. In this population, the mean TR-derived EPSPAP was  $40 \pm 18$  mm Hg (range, 23–80 mm Hg). The mean PAAT was  $126 \pm 28$  msec (range, 52–184 msec). As a result, the mean PAAT-derived EPSPAP was  $42 \pm 13$  mm Hg (range, 22–86 mm Hg). The relationship between TR-derived EPSPAP and PAAT-derived EPSPAP yielded a correlation coefficient of 0.95 (95% confidence interval, 0.933–0.974; Figure 6).

## DISCUSSION

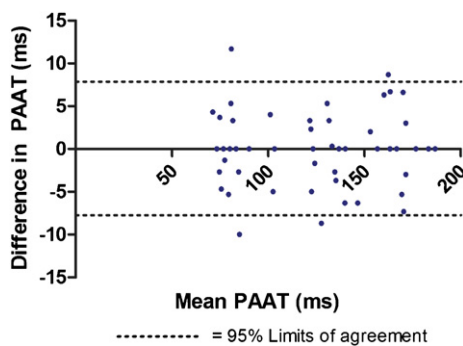
TTE has proven to be an important tool in the noninvasive assessment of pulmonary artery pressures in a wide spectrum of illnesses.<sup>6–18</sup> The most widely accepted transthoracic method for the estimation of pulmonary artery pressures relies on the measurement of  $\text{TR}_{\text{Vmax}}$ . However, TR is frequently not sufficient to perform this measurement, as demonstrated by the fact that 25% of patients in our randomly selected clinical cohort had insufficient TR for EPSPAP determination.

Alternative techniques have been developed to assess pulmonary hemodynamics. Kitabatake *et al.*<sup>7</sup> showed that PAAT correlates well with MPAP and its logarithm ( $r = 0.82$  and  $0.88$ , respectively) as

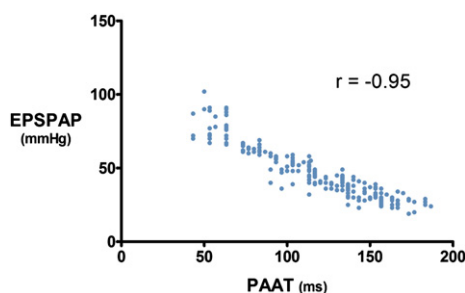
**Table 1** Measurements of TR-derived and pulmonary artery flow-derived measurements in a randomly selected cohort ( $n = 371$ ) of clinically indicated transthoracic echocardiograms

Variable	Mean $\pm$ SD (range)
<b>TR-derived values</b>	
TR <sub>Vmax</sub> (cm/sec)	282 $\pm$ 65 (150–480)
RA/RV gradient (mm Hg)	34 $\pm$ 16 (9–92)
EPSPAP (mm Hg)	44 $\pm$ 16 (19–102)
<b>Pulmonary artery-derived values</b>	
PAAT (ms)	124 $\pm$ 34 (43–186)
Log <sub>10</sub> (PAAT)	2.07 $\pm$ 0.14 (1.64–2.27)
PA <sub>Vmax</sub> (cm/sec)	89 $\pm$ 20 (44–175)
RVET (msec)	362 $\pm$ 51 (216–580)
PAAT/RVET (m/sec <sup>2</sup> )	0.35 $\pm$ 0.11 (0.08–0.64)

PA<sub>Vmax</sub>, Maximal pulmonary artery velocity; RA, right atrial. An RA pressure of 10 mm Hg was assumed for the calculation of RVSP.

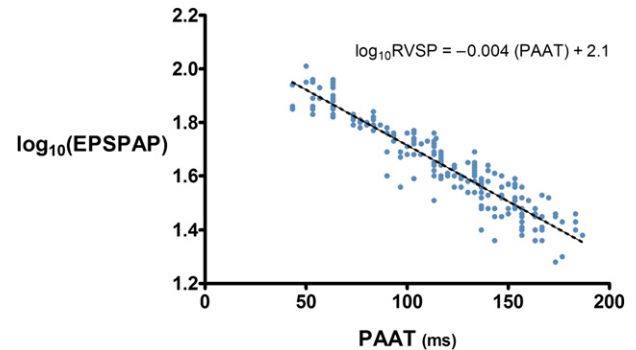


**Figure 3** Bland-Altman analysis showing the limits of agreement between two observers with respect to measurement of PAAT.

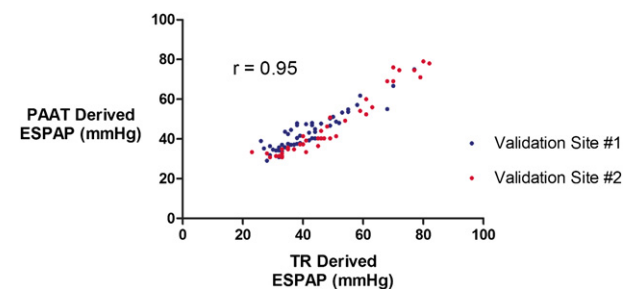


**Figure 4** Relationship between TR-derived EPSPAP and PAAT.

determined by cardiac catheterization. They further demonstrated that PAAT corrected for RVET time, a method of adjusting for heart rate, slightly strengthened the correlation between PAAT and MPAP. In a subsequent study, Dabestani *et al.*<sup>12</sup> described that same relationship as one that observed the following equation: MPAP = 73 – (0.42 × PAAT). In contrast to Kitabatake *et al.*, they found that neither the logarithmic transformation of MPAP nor the correction for RVET improved the accuracy of estimating the MPAP using PAAT. Together, these studies established the capacity of Doppler-



**Figure 5** Relationship between log<sub>10</sub>(TR-derived EPSPAP) and PAAT.



**Figure 6** Prospectively obtained validation cohort examining the relationship between PAAT-derived and TR-derived EPSPAP.

derived measurements of pulmonary arterial flow to provide accurate estimates of MPAP. RV isovolumic relaxation time (RVIRT)<sup>19</sup> and tissue Doppler-derived RVIRT<sup>10</sup> have also been studied to determine their suitability to estimate systolic pulmonary artery pressure (SPAP). Brechot *et al.*<sup>10</sup> studied 26 patients by echocardiography and cardiac catheterization. Doppler-derived RVIRT correlated strongly with SPAP and was able to reliably exclude pulmonary arterial hypertension. Importantly, however, prolonged RVIRT was not specific for pulmonary hypertension, and RVIRT-derived SPAP did not agree well with the catheter-derived value.

Despite these important publications, the use of PAAT in clinical practice has remained relatively limited. This is largely because the vast majority of publications reporting echocardiographic pulmonary artery pressure estimates have relied on TR<sub>Vmax</sub>-derived EPSPAP. As such, clinicians who rely on TTE for pulmonary artery pressure quantification have developed both a familiarity with and a preference for peak systolic, not mean, pulmonary artery pressure values. Despite the well-established role of invasively measured MPAP values with respect to the diagnosis and management of pulmonary hypertension, the use of noninvasively derived EPSPAP values is justifiable given their accuracy, reproducibility, and extensive validation.<sup>20,21</sup> In addition, the relationship between different components of pulmonary artery pressure (SPAP, MPAP, and diastolic pulmonary artery pressure) has been found to remain constant after changes in posture and activity,<sup>22</sup> which may allow one to use any of the three as a surrogate of the other two. Although sound and useful methods of assessing MPAP have been developed, there currently exists no method that can be used to procure an EPSPAP value in the absence of TR. Our data, including the precisely defined quantitative relationship between PAAT and TR-derived EPSPAP, now provide such a tool. The regression equation presented in this report can easily

be integrated in clinical and research echocardiography labs for use in patients with insufficient TR. Results from the current study demonstrate that PAAT can be used as a reliable alternative method for the derivation of EPSPAP.

Our findings and their implications can be summarized as follows. First,  $TR_{V_{max}}$  was not measurable in 25.3% of patients referred for routine, clinically indicated TTE, while PAAT was measurable in 99.6%. Although saline injection may be used to augment a weak or immeasurable TR signal,<sup>23-25</sup> this approach involves the insertion of an intravenous line and coordination of agitated saline injection and color Doppler interrogation of the tricuspid valve. Although this technique may be useful, it is resource and time consuming and thus may not be optimal in a busy echocardiography laboratory. This rationale underscores the need for an approach to pulmonary artery pressure assessment that is independent of the presence of TR and justifies the use of a PAAT-derived method in the clinical echocardiography laboratory.

Second, we observed a strong, inverse correlation between PAAT and EPSPAP among a cohort of patients with a wide range of EPSPAP values. Quantification of this relationship by linear regression led to the derivation of an equation by which PAAT can be used to provide EPSPAP values comparable with those obtained using  $TR_{V_{max}}$ . Thus, we present a novel, TR-independent approach to the quantitative assessment of EPSPAP that appears to perform well across a wide range of pulmonary artery pressures. In a recent series, a significant number of patients with pulmonary pressures >50 mm Hg had mild TR or less, rendering the reliance on  $TR_{V_{max}}$  for the measurement of EPSPAP unfeasible.<sup>26</sup> The use of PAAT for the derivation of EPSPAP has the potential to significantly increase the overall percentage of patients in whom noninvasive assessment of pulmonary hemodynamics can be performed.

Several technical and physiologic issues relevant to this study deserve mention. Although proximal pulmonary artery flow, as assessed by pulsed-wave Doppler, has a characteristic pressure-dependent pattern in which the acceleration phase becomes shorter as the pulmonary artery pressure increases, additional factors, including heart rate, cardiac output, RV function, and imaging technique, may affect its duration.<sup>11</sup> We accounted for the potential impact of heart rate variability in this study by correcting for absolute heart rate and RVET. Similar to previously published studies,<sup>12</sup> we did not find these corrections to be necessary. Because we were not able to control for cardiac output in this study, we cannot comment on how this affected our findings, and we remain uncertain about the accuracy of PAAT for EPSPAP in patients with high cardiac output due to exercise or systemic vasodilation. Next, it has been shown that RV function can affect PAAT with RV systolic dysfunction tending to lengthen the PAAT.<sup>27</sup> In our study population, only 12 patients had mild or greater RV dysfunction, and their removal and the subsequent reanalysis of data did not affect our observed regression and correlation results. Because of the small number of patients with RV dysfunction in our cohort, we caution that our model may not be accurate in patients with RV systolic dysfunction. Finally, we wish to point out that imaging technique plays a significant role in the acquisition of a PAAT that accurately reflects pulmonary pressure. Specifically, proper placement of the Doppler cursor in the middle of the pulmonary artery and accurate alignment to the long axis of the pulmonary artery are both crucial. A retrospective analysis of outlying values in our cohort (Figures 3 and 6) revealed some degree of Doppler beam and pulmonary arterial flow misalignment in a majority of cases. It should be stressed that careful alignment of the Doppler beam with the central, long axis of the pulmonary artery is an essential step in the use of the PAAT for EPSPAP acquisition.

The few limitations of this study are noteworthy. First, our study design involved a comparison of measurements derived from echocardiograms of patients without any correlative invasive hemodynamic measurements. Thus, our findings are limited to a comparison of noninvasive techniques, and confirmation of our findings using invasive measurement of peak systolic pressure is an important goal of future work.

Second, our echocardiographic laboratory's practice emphasizes placement of the Doppler sample volume (during pulsed-wave and continuous-wave Doppler interrogation) in the center of the pulmonary artery to optimize the laminar flow pattern. Deviation from this technique, such as the sampling of a volume close to the pulmonary artery wall, may alter this flow pattern and render it unreliable.

Third, we were unable to determine the performance of PAAT for the estimation of EPSPAP in conditions characterized by high cardiac output, such as volitional exercise or the systemic vasodilation of sepsis. Further work is needed to examine the behavior of PAAT in this context.

## CONCLUSIONS

We have shown that PAAT is easily obtainable and strongly correlated with  $TR_{V_{max}}$  in a large population of randomly selected patients undergoing TTE. Characterization of the relationship between PAAT and EPSPAP now permits PAAT to be used to derive EPSPAP independently of TR. Adoption of this novel method of determining EPSPAP has the capacity to significantly increase the percentage of patients in which TTE can be used to quantify pulmonary artery pressures.

## REFERENCES

1. Yock PG, Popp RL. Noninvasive estimation of right ventricular systolic pressure by Doppler ultrasound in patients with tricuspid regurgitation. *Circulation* 1984;70:657-62.
2. Ge Z, Zhang Y, Kang W, Fan D, Ji X, Duran C. Noninvasive evaluation of right ventricular and pulmonary artery systolic pressures in patients with ventricular septal defects: simultaneous study of Doppler and catheterization data. *Am Heart J* 1993;125:1073-81.
3. Marx GR, Allen HD, Goldberg SJ. Doppler echocardiographic estimation of systolic pulmonary artery pressure in pediatric patients with interventricular communications. *J Am Coll Cardiol* 1985;6:1132-7.
4. Musewe NN, Poppe D, Smallhorn JF, Hellman J, Whyte H, Smith B, et al. Doppler echocardiographic measurement of pulmonary artery pressure from ductal Doppler velocities in the newborn. *J Am Coll Cardiol* 1990;15:446-56.
5. Marx GR, Allen HD, Goldberg SJ. Doppler echocardiographic estimation of systolic pulmonary artery pressure in patients with aortic-pulmonary shunts. *J Am Coll Cardiol* 1986;7:880-5.
6. Masuyama T, Kodama K, Kitabatake A, Sato H, Nanto S, Inoue M. Continuous-wave Doppler echocardiographic detection of pulmonary regurgitation and its application to noninvasive estimation of pulmonary artery pressure. *Circulation* 1986;74:484-92.
7. Kitabatake A, Inoue M, Asao M, Masuyama T, Tanouchi J, Morita T, et al. Noninvasive evaluation of pulmonary hypertension by a pulsed Doppler technique. *Circulation* 1983;68:302-9.
8. Currie PJ, Seward JB, Chan KL, Fyfe DA, Hagler DJ, Mair DD, et al. Continuous wave Doppler determination of right ventricular pressure: a simultaneous Doppler-catheterization study in 127 patients. *J Am Coll Cardiol* 1985;6:750-6.
9. Bouhemad B, Ferrari F, Leleu K, Arbelot C, Lu Q, Roubey JJ. Echocardiographic Doppler estimation of pulmonary artery pressure in critically ill patients with severe hypoxemia. *Anesthesiology* 2008;108:55-62.

10. Brechot N, Gambotti L, Lafitte S, Roudaut R. Usefulness of right ventricular isovolumic relaxation time in predicting systolic pulmonary artery pressure. *Eur J Echocardiogr* 2008;9:547-54.
11. Chan KL, Currie PJ, Seward JB, Hagler DJ, Mair DD, Tajik AJ. Comparison of three Doppler ultrasound methods in the prediction of pulmonary artery pressure. *J Am Coll Cardiol* 1987;9:549-54.
12. Dabestani A, Mahan G, Gardin JM, Takenaka K, Burn C, Allfie A, et al. Evaluation of pulmonary artery pressure and resistance by pulsed Doppler echocardiography. *Am J Cardiol* 1987;59:662-8.
13. Friedberg MK, Feinstein JA, Rosenthal DN. A novel echocardiographic Doppler method for estimation of pulmonary arterial pressures. *J Am Soc Echocardiogr* 2006;19:559-62.
14. Homma A, Anzueto A, Peters JI, Susanto I, Sako E, Zabalgoitia M, et al. Pulmonary artery systolic pressures estimated by echocardiogram vs cardiac catheterization in patients awaiting lung transplantation. *J Heart Lung Transplant* 2001;20:833-9.
15. Lanzarini L, Fontana A, Lucca E, Campana C, Klersy C. Noninvasive estimation of both systolic and diastolic pulmonary artery pressure from Doppler analysis of tricuspid regurgitant velocity spectrum in patients with chronic heart failure. *Am Heart J* 2002;144:1087-94.
16. Posteraro A, Salustri A, Trambaiolo P, Amici E, Gambelli G. Echocardiographic estimation of pulmonary pressures. *J Cardiovasc Med (Hagerstown)* 2006;7:545-54.
17. Skjaerpe T, Hatle L. Noninvasive estimation of systolic pressure in the right ventricle in patients with tricuspid regurgitation. *Eur Heart J* 1986;7:704-10.
18. Stevenson JG. Comparison of several noninvasive methods for estimation of pulmonary artery pressure. *J Am Soc Echocardiogr* 1989;2:157-71.
19. Burstin L. Determination of pressure in the pulmonary artery by external graphic recordings. *Br Heart J* 1967;29:396-404.
20. McGoon M, Gutterman D, Steen V, Barst R, McCrory DC, Fortin TA, et al. Screening, early detection, and diagnosis of pulmonary arterial hypertension: ACCP evidence-based clinical practice guidelines. *Chest* 2004;126:14S-34.
21. Chemla D, Castelain V, Humbert M, Hebert JL, Simonneau G, Lecarpentier Y, et al. New formula for predicting mean pulmonary artery pressure using systolic pulmonary artery pressure. *Chest* 2004;126:1313-7.
22. Syeed R, Reeves JT, Welsh D, Raeside D, Johnson MK, Peacock AJ. The relationship between the components of pulmonary artery pressure remains constant under all conditions in both health and disease. *Chest* 2008;133:633-9.
23. Waggoner AD, Barzilai B, Perez JE. Saline contrast enhancement of tricuspid regurgitant jets detected by Doppler color flow imaging. *Am J Cardiol* 1990;65:1368-71.
24. Torres F, Tye T, Gibbons R, Puryear J, Popp RL. Echocardiographic contrast increases the yield for right ventricular pressure measurement by Doppler echocardiography. *J Am Soc Echocardiogr* 1989;2:419-24.
25. Beard JT II, Byrd BF III. Saline contrast enhancement of trivial Doppler tricuspid regurgitation signals for estimating pulmonary artery pressure. *Am J Cardiol* 1988;62:486-8.
26. Mutlak D, Aronson D, Lessick J, Reisner SA, Dabbah S, Agmon Y. Functional tricuspid regurgitation in patients with pulmonary hypertension: is pulmonary artery pressure the only determinant of regurgitation severity? *Chest* 2009;135:115-21.
27. Stewart WJ, Jiang L, Mich R, Pandian N, Guerrero JL, Weyman AE. Variable effects of changes in flow rate through the aortic, pulmonary and mitral valves on valve area and flow velocity: impact on quantitative Doppler flow calculations. *J Am Coll Cardiol* 1985;6:653-62.

# Did you know?

The screenshot shows the JASE website interface. At the top, there is a search bar and navigation links. The main content area features a 'Current Issue' section for September 2008, with links for 'VIEW CURRENT ISSUE', 'VIEW ALL ISSUES', 'ARTICLES IN PRESS', 'ONLINE', 'SEARCH THIS JOURNAL', 'FREE TRIAL ISSUE', and 'EMAIL ALERTS'. Below this is an 'ASE Information' section. A red arrow points to a link in the 'FREE EMAIL ALERTS' section that says 'Click Here to Sign Up for Alerts'. Other links include 'Register or Log In', 'Forgot Your Password?', 'Institutional subscribers', 'ASE members', 'Subscribe to the Journal', and 'Free Trial Issue'.

You can save your online searches and get the results by email.

Visit [www.onlinejase.com](http://www.onlinejase.com) today!