International Journal of Advances in Medicine
Khese VB et al. Int J Adv Med. 2021 Apr;8(4):523-529
http://www.ijmedicine.com
pISSN 2349-3925 | eISSN 2349-3933
DOI: https://dx.doi.org/10.18203/2349-3933.ijam20211049

Original Research Article

Assessment of pulmonary artery hypertension by Doppler echocardiography and its correlation with right heart catheterization

Vikrant B. Khese, Chandrakant B. Chavan*, Ravi Kalra, Anirudh K. Allam, Abhinav Mohabey

Department of Cardiology, Bharati Hospital and Research Centre, Pune, Maharashtra, India

Received: 13 January 2021
Revised: 17 February 2021
Accepted: 01 March 2021

***Correspondence:**
Dr. Chandrakant B. Chavan,
E-mail: cbchavan19740210@yahoo.com

Copyright: © the author(s), publisher and licensee Medip Academy. This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

ABSTRACT

Background: Definitive diagnosis of pulmonary artery hypertension (PH) requires an elevated mean pulmonary arterial pressure (MPAP) of 25 mmHg at rest measured by right heart catheterization (RHC). As it is invasive mode of investigation, it is declined by many patients, echocardiography was thought to be an acceptable substitute to assess pulmonary arterial pressures. Whether there is a correlation between these measurements is controversial. The aim of this study was to assess PH by echocardiography and its correlation with RHC.
Methods: Twenty-six patients aged ≥18 years with pulmonary artery hypertension with or without tricuspid regurgitation (TR) were included in this cross-sectional study. All the patients underwent a transthoracic echocardiography evaluation and were taken for RHC study within an hour.
Results: The correlation between pulmonary artery acceleration time (PAAT) and pulmonary artery systolic pressure (PASP) and PAAT and MPAP was significant in all degrees of PH. In contrast, correlation between TR jet maximum velocity (TR Vmax) derived estimated pulmonary artery systolic pressure (EPASP) and PASP was significant in moderate and severe PH, while it did not correlate in mild PH.
Conclusions: PAAT is easily measurable parameter and strongly correlates with the values of PASP and the MPAP obtained by right heart catheterization. Implementation of a novel method of determining EPASP from PAAT shall increase significantly the number of patients in whom TTE can be used for the assessment of pulmonary hemodynamic non-invasively.

Keywords: Pulmonary artery acceleration time, Mean pulmonary artery pressure, Pulmonary hypertension

INTRODUCTION

According to the definition, right heart catheterization was needed for accurate diagnosis of the pulmonary artery pressure (PAH).[1] Even though RHC is now a fairly safe procedure, it is invasive and impractical to perform in patients for whom it is not clearly indicated. Hence, there is a need of non-invasive means that aid in diagnosis and allow identification of patients for whom diagnostic RHC is indicated, because of a high likelihood of the existence of PH.[2] Echocardiography is less complicated, less expensive, and less time consuming. Ultrasound imaging has continuously evolved over the recent years, leading to the development of several new echocardiographic indices that allow the evaluation of pulmonary pressures (systolic, mean, and diastolic) and also the estimation of other pulmonary hemodynamic parameters, such as pulmonary vascular resistance (PVR), pulmonary capillary wedge pressure (PCWP) and the pulmonary capacitance and impedance. Thus, it is now possible to obtain a complete and accurate description of the pulmonary hemodynamics using transthoracic echocardiography (TTE) imaging.[3]

Non-invasive assessment of systolic pulmonary artery pressure is a part of assessing cardiac function through echocardiograph.

Purpose of this study is focused on diagnosing PAH through echocardiography can be well-thought-out reliable tool without the need of performing RHC.

METHODS

This prospective observational study was conducted between January 2019 and November 2019. The subjects for the study were selected from cases admitted to the medical wards of Bharati Hospital and Research Centre, Pune. After approval from the scientific advisory committee and institutional ethics committee, written informed consent was obtained from all the patients. Twenty-six patients, twelve males and fourteen females, with a diagnosis of PH established by transthoracic echocardiography, in whom right heart catheterization was clinically indicated were studied. Patients with pulmonary hypertension of various aetiologies were included in the study. Patients with poor echo window, critically ill patients and Paediatric age group patients were excluded.

All the patients included in the study were subjected to detailed history taking and thorough and complete physical examination. Complete blood count, blood sugar and renal function test were done in all patients. A 12-lead electrocardiogram and a chest x ray were obtained in all patients. They were also evaluated for the aetiology of pulmonary hypertension by appropriate tests as indicated: anti-nuclear antibody profile, coagulation profile, human immunodeficiency virus (HIV) testing, high resolution computed tomography (CT) scan of the chest, ultrasound abdomen and pulmonary function testing.

Echocardiographic evaluation

All the patients underwent a detailed transthoracic echocardiographic evaluation using a standard protocol, within one hour of undergoing the right heart catheterization study. Two-dimensional, M-mode, colour flow, spectral Doppler and tissue Doppler examinations were done using GE VIVID E-95 echocardiography machine. Electrocardiography (ECG) monitoring was done during the examination. Examination was done in the left lateral decubitus and in the supine positions. To assess right ventricular (RV) size, function and area, a complete set of standardized views were obtained. These included parasternal long axis (PLAX), parasternal RV inflow, parasternal short axis (PSAX), apical 4 chamber, RV–focused apical 4-chamber, and subcostal views. The basal diameter, defined as the maximal short-axis dimension in the basal one third of the RV was measured on the RV focused apical 4-chamber view. Measurement of right ventricular outflow tract (RVOT) dimensions were made at the proximal or sub valvular level (RVOT-prox) and at the distal or pulmonic valve (RVOT distal) levels in the PLAX RVOT anterior portion view, basal PSAX, and parasternal short-axis of pulmonary bifurcation view (Figure 1). Measurement of end-diastolic right ventricular wall thickness was made in the subcostal 4 chamber view. Right ventricular systolic pressure (RVSP) and therefore the EPASP was determined from the peak TR jet velocity, using the simplified Bernoulli equation and this value was combined with an estimate of the RA pressure:

$$\text{EPASP} = 4(\text{V})2 + \text{RA pressure}$$

Where V is the peak velocity (in meters per second) of the tricuspid valve regurgitant jet.

TR signals were obtained from several windows and the signal with the highest velocity was used. Doppler sweep speed of 100 mm/s was used for all tracings (Figure 2). RA pressure was estimated by IVC diameter and the presence of inspiratory collapse. The subcostal view was used for imaging the IVC, with the IVC viewed in its long axis. The measurement of the IVC diameter was made at end-expiration and just proximal to the junction of the hepatic veins with the IVC (Figure 3). IVC diameter ≤2.1 cm, collapsing >50% was considered to have 0–5 mmHg RA pressure, IVC diameter ≤2.1 cm, collapsing ≤50% was considered to have 5–10 mmHg RA pressure and IVC diameter >2.1 cm decrease >50% was considered to have RA pressure 10–15 mmHg, IVC diameter >2.1 cm decrease ≤50% was considered to have RA pressure 15–20 mmHg.[4] Pulmonary artery diastolic pressure (PADP) was estimated from the velocity of the end-diastolic pulmonary regurgitant jet using the modified Bernoulli equation:

$$\text{PADP} = 4 \times (\text{end} - \text{diastolic pulmonary regurgitant velocity})2 + \text{RA pressure}$$

Pulsed-wave (PW) and continuous-wave (CW) Doppler interrogation of the proximal pulmonary artery was performed in the PSAX with the sample volume placed along the long axis of the main pulmonary artery to maximally align blood flow and Doppler interrogation. This view was also used to exclude pulmonary stenosis. Using the pulse-wave Doppler profile, PAAT, right ventricular ejection time (RVET), and heart rate were measured. PAAT was measured as the interval between the beginning of systolic pulmonary arterial flow and peak flow velocity. RVET was measured as the interval between beginnings of RV ejection to the point of systolic pulmonary arterial flow cessation. All values used for the analysis were the average of three consecutive cardiac cycles, but in patients with atrial fibrillation, five-beat averages were obtained. MPAP was estimated from PAAT measured by PW Doppler of the pulmonary artery in systole:

$$\text{Mean PA pressu} = -(0.6 \times \text{AT})\ \{\text{Mahn and Dabestani formula}\}$$

The percentage RV fractional area change, FAC, which is a measure of RV systolic function, and defined as:

$$\text{FAC} = \text{end} - \text{diastolic area} - \text{end} - \text{systolic area})/\text{end} - \text{diastolic area} \times 100$$

FAC was obtained by tracing the RV endocardium both in systole and diastole. Tricuspid annular plane systolic excursion (TAPSE) was acquired by placing an M mode cursor through the tricuspid annulus and measuring the amount of longitudinal motion of the annulus at peak systole in the apical 4 chamber view (Figure 4).

RV s' or systolic excursion velocity was measured in the apical 4-chamber window, with a tissue Doppler mode region of interest highlighting the RV free wall. The PW Doppler sample volume was placed in either the tricuspid annulus or the middle of the basal segment of the RV free wall. The velocity RV s' was measured as the highest systolic velocity, without over-gaining the Doppler envelope. The parameters used to assess RV diastolic function namely, the Doppler velocities of the trans-tricuspid flow (E, A, and E/A), tissue Doppler velocities of the tricuspid annulus (e', a', E/e') and the E-deceleration time were measured in the apical 4 chamber view.

Right heart catheterization (RHC)

RHC was performed after overnight fasting, at rest in the cardiac catheterization laboratory using the PHILIPS FD-10 machine, without sedation by the standard technique. Catheterisation was done under local anaesthesia, through the right femoral venous route. Multipurpose catheter was used for pressure measurements. Pressure measurements were taken from the pulmonary arterial wedge position, main pulmonary artery, right ventricle and right atrium at the end of expiration. Data collected were entered in Excel 2019 and analysis of data was done using statistical package for social sciences (SPSS) version 20, IBM, USA. The comparison of quantitative variables between the two groups was done using unpaired student's "t" test, comparison of variables between more than 2 groups was done by analysis of variance (ANOVA) test. Comparison of qualitative variables was done by using Chi-square test or Fisher's exact test. Pearson's correlation was used to study correlation. The confidence limit for significance was fixed at 95% level with p value <0.05.

RESULTS

: In our study, 53.8% (14 of 26) of our patients belonged to group 1 pulmonary hypertension. Of them, 42 (11 of 26) had shunt lesion as the cause of PAH, 1 patient due to a coronary AV fistula, 7.7% (2 of 26) had systemic sclerosis. Group 2 PH due to left heart disease was present in 34.6% (9 of 26). Group 3 PH due to lung disease was present in 3.8% (1 of 26) and group 4 due to chronic pulmonary thromboembolism (CPTE) (2 of 26) (Table 1).

Table 1: Baseline characteristics (n=26).

Characteristics	N (%)
Gender	
Male	46 (12)
Female	54 (14)
Age (years)	
Mean±SD	32.8±12.9
<20	15.4 (4)
20-29	30.8 (8)
30-39	23.1 (6)
40-49	23.1 (6)
≥50	7.7 (2)
Symptoms	
Chest pain	38.5 (10)
Shortness of breath	84.6 (22)
RV failure symptoms	7.7 (2)
ECG results	
RVH	30.8 (8)
RBBB	19.2 (5)
BVH	11.5 (3)
LAA	11.5 (3)
Clinical diagnosis	
Group 1	
CHD	42.3 (11)
Cor AVF	3.8 (1)
PPH	0 (0)
SScl	7.7 (2)
Group 2	
RHD	34.6 (9)
Group 3	
Cystic lung	0 (0)
ILD	3.8 (1)
Group 4	
CPTE	7.7 (2)

The mean value of the tricuspid regurgitation peak gradient (TRPG) was 59.9±21.1 and that of the ESPAP was 64.9±21.3. The mean PAAT was 79.4±13.9 and the calculated mean pulmonary artery pressure was 41±8.75 (Table 2).

The mean basal RV diameter was 3.6±0.45 (normal <4.2 cm). The mean proximal RVOT diameter in PLAX view was 3.23±0.5 (normal <3.3 cm). The mean distal RVOT diameter in PSAX view was 2.87±0.5 (normal <2.7 cm) and the mean RV free wall thickness in subcostal view was 6.5±1.0 (normal <5 mm) (Table 3). Only the mean RVOT distal diameter was found to be above normal (Table 4). The RV systolic function as measured by TAPSE was normal in 88.4% (23 of 26) of the patients. Tricuspid s' was normal in 84.6% (22 of 26) of the patients. The RV fractional area change was normal in 84.6% (22 of 26) of the patients. Overall, 11.5% (3 of 26) of the patients had RV systolic dysfunction. The RV diastolic function was normal in 26.9% (7 of 26) patients. Impaired relaxation was present in 19.2% (5 of 26) patients. 50.9 % (14 of 26)

patients had pseudo normal filling pattern and restrictive filling pattern was present in none of the patients studied.

Table 2: TTE derived pulmonary hemodynamic.

Parameter	Range	Mean±SD
Tricuspid regurgitation peak gradient	32-99	59.96±21.17
Estimated right atrial pressure	3-8	5±0.75
Estimated systolic pulmonary artery pressure	37-104	64.92±21.37
Pulmonary artery acceleration time	57-100	79.42±13.98
Right ventricle ejection time	239-294	259.81±19.14
PAAT/RVET ratio	0.2-0.41	0.31±0.07
PR derived PA diastolic pressure	15-36	23±7.02
Estimated mean pulmonary artery pressure	28-54.6	41.02±8.75

Table 3: Right ventricle dimensions.

Right ventricle diameter	Range	Mean±SD
Basal (cm)	2.8-4.3	3.63±0.45
RV outflow tract – proximal (cm)	2.4-4.1	3.23±0.54
RV outflow tract – distal (cm)	2-4	2.87±0.55
RV free wall thickness (mm)	5-9	6.5±1.0

Table 4: RV dimensions: normal and abnormal.

Parameter (abnormal)	Normal		Abnormal	
	No	%	No	%
RV basal diameter cm (>4.2 cm)	25	96.15	1	3.85
RV free wall thickness (>5 mm)	2	7.69	24	92.31
RVOT PSAX distal diameter (>2.7 cm)	10	38.46	16	61.54
RVOT PLAX proximal diameter (>3.3 cm)	15	57.69	11	42.31

The mean value of the PASP measured by RHC was 63.08±18.54 (versus the echo – Doppler estimated PASP of 64.9±21.3) in the study population, that of PADP was 26.6±7.71 (versus echo derived mean value of 23±7) and the MPAP was 40.96±8.96 (versus the echo PAAT derived mean value of 41±8.75). The mean right atrial pressure measured invasively had a mean value of 8.23±3.02 (versus echo derived value of 5±0.75) (Table 5).

The PH was mild in 30.77%, moderate in 38.46% and severe in 30.77% of the cases.

Table 5: Invasively obtained right heart pressures.

Invasive pressures	Range	Mean±SD
PC wedge pressure	8-30	14.19±7.60
P.A. systolic pressure	38-104	63.08±18.54
P.A. diastolic pressure	17-43	26.65±7.71
Mean pulmonary artery pressure	29-58	40.96±8.96
RV systolic pressure	40-100	64.42±19.08
RV end diastolic pressure	3-15	8.92±2.90
Right atrial pressure	4-18	8.23±3.02

Association between echo derived findings and invasively derived findings

There was strong inverse correlation between the echo measured PAAT and both the invasively measured PASP and the MPAP. This correlation was stronger for MPAP. This strong inverse correlation remained thus even after correction for heart rate, between PAAT/RVET and the invasively measured PASP and the MPAP. This was comparable to that between echo TR Vmax derived EPASP and invasively measured PASP. Our study also presented strong direct correlation between echo derived PADP and the invasively measured PADP. The strength of correlation between PAAT and PASP and between PAAT and MPAP was strong among the different groups of PH, and this was also not affected by correction for heart rate and was comparable to the strength of correlation between EPASP and invasively measured PASP (Table 6).

The analysis of results revealed that**: t**he correlation between PAAT and invasive PASP was significant in all degrees of PH while it was less strong in patients with severe PH (Table 7); there was significant correlation between PAAT and invasive MPAP in all degrees of PH; correction for heart rate did not affect the above results (Table 7); in contrast, correlation between TR Vmax derived EPASP and PASP was significant in moderate (moderate correlation) and severe PH (strong correlation), while it was not so in mild PH (Table 7); and correlation between PR derived PADP and invasively obtained PADP was also significant in moderate and severe PH, but not so in mild PH (Table 7).

The strength of correlation between PAAT and PASP and between PAAT and MPAP was strong among the different groups with normal diastolic function, impaired relaxation and pseudo normal filling and this was not affected by correction for heart rate and was comparable to the strength of correlation between EPASP and invasively measured PASP (Table 8).

The strength of correlation between PAAT and PASP and between PAAT and MPAP remained strong after excluding patients with RV systolic dysfunction and also was not affected by correction for heart rate and remained comparable to the strength of correlation between EPASP

and invasively measured PASP. The small (3 of 26) group of patients with RV systolic dysfunction also showed significant correlation between the various above-mentioned parameters analysed (Table 9).

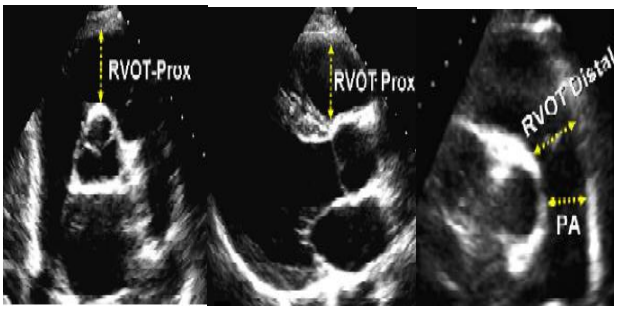

Figure 1: Measurement of right ventricular outflow tract (RVOT) dimensions.

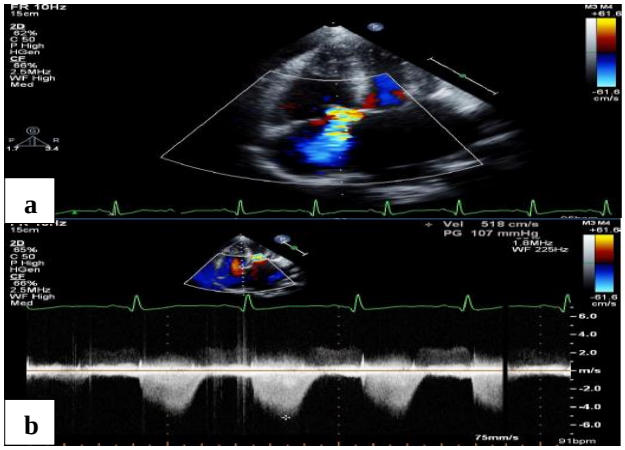

Figure 2: (a) Tricuspid regurgitation and (b) tricuspid regurgitation continuous Doppler.

Table 6: Correlation between echo derived findings and invasively derived findings.

Correlation between	Mean±SD	Correlation coefficient	Correlation
Echo derived PAAT	79.42±13.98	-0.93	Strong inverse correlation
Invasive PSAP	63.08±18.54		
Echo derived PAAT	79.42±13.98	-0.96	Strong inverse correlation
Invasive MPAP	40.96±8.96		
Echo derived PAAT/RVET	0.31±0.07	-0.92	Strong inverse correlation
Invasive PSAP	63.08±18.54		
Echo derived PAAT/RVET	0.31±0.07	-0.95	Strong inverse correlation
Invasive MPAP	40.96±8.96		
Echo TR Vmax derived ESPAP	64.92±21.37	0.94	Strong direct correlation
Invasive PSAP	63.08±18.54		
Echo derived PADP	23±7.02	0.89	Strong direct correlation
Invasive PADP	26.65±7.71		

Table 7: Correlation between echo derived findings and invasively obtained findings in different degrees of PH.

Correlated parameters	Correlation coefficient		
	Mild PH	**Moderate PH**	**Severe PH**
Echo based PAAT and invasive PSAP	-0.70	-0.77	-0.59
Echo based PAAT and invasive MPAP	-0.85	-0.75	-0.93
Echo based PAAT/RVET and invasive PSAP	-0.73	-0.86	-0.57
Echo based PAAT/RVET and invasive MPAP	-0.87	-0.82	-0.89
Echo based ESPAP and invasive PSAP	-0.08	0.69	0.82
Echo based PREDP and invasive PADP	-	0.64	0.98

Table 8: Correlation between echo derived findings and invasively obtained findings in different types of RV diastolic function.

Correlated parameters	Correlation coefficient		
	Normal	**Impaired relaxation**	**Pseudo normal filling**
Echo based PAAT and invasive PSAP	-0.93	-0.96	-0.93
Echo based PAAT and invasive MPAP	-0.89	-0.99	-0.99
Echo based PAAT/RVET and invasive PSAP	-0.92	-0.92	-0.93
Echo based PAAT/RVET and invasive MPAP	-0.86	-0.98	-0.99
Echo based ESPAP and invasive PSAP	0.99	0.87	0.97
Echo based PREDP and invasive PADP	-	1.00	0.89

Table 9: Correlation between echo derived findings and invasively obtained findings in in normal and abnormal RV systolic function.

Correlated parameters	Correlation coefficient	
	Normal systolic function	Systolic dysfunction
Echo based PAAT and invasive PSAP	-0.93	-0.98
Echo based PAAT and invasive MPAP	-0.96	-0.91
Echo based PAAT/RVET and invasive PSAP	-0.93	-0.96
Echo based PAAT/RVET and invasive MPAP	-0.95	-0.86
Echo based ESPAP and invasive PSAP	0.94	0.87
Echo based PREDP and invasive PADP	0.93	0.94

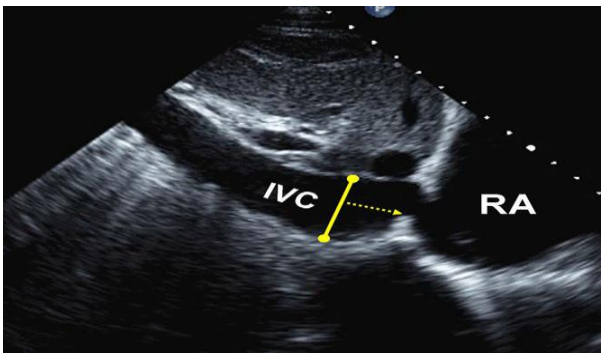

Figure: 3 IVC diameter.

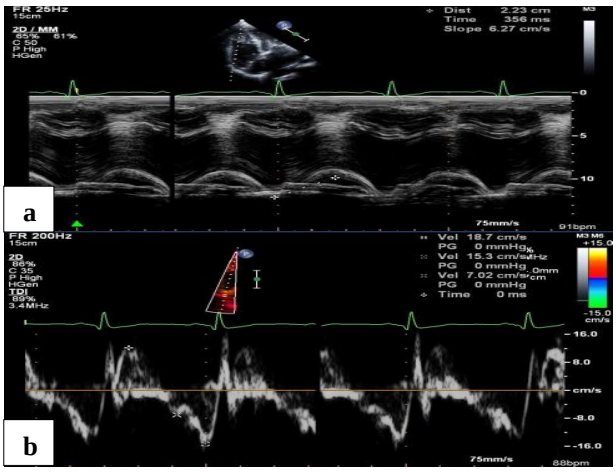

Figure 4: (a) Tricuspid annular plane systolic excursion, and (b) tricuspid annular tissue Doppler velocities.

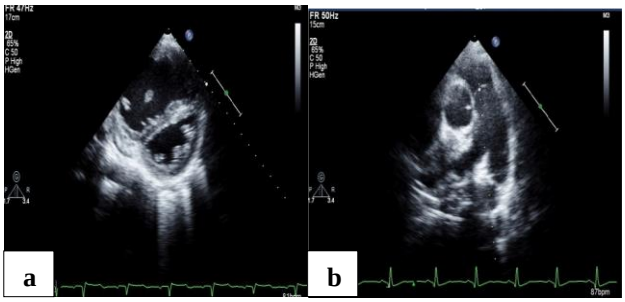

Figure 5: (a) Parasternal short axis view: D shaped septum, and (b) PSAX view: dilated MPA.

DISCUSSION

TTE has been proved to be an important method in the non-invasive assessment of pulmonary artery pressures in a wide range of diseases.[5,6] The most widely accepted and employed transthoracic echocardiographic method for the derivation of pulmonary artery pressures relies on measurement of TR Vmax. But sometimes, TR is not sufficient to perform this measurement, as shown by the fact that 8.7% of patients in our study of patients with PH of varied etiologies had insufficient TR for EPASP estimation. Kitabatake et al showed that PAAT correlates strongly with MPAP and its logarithm (r=0.82 and 0.88, respectively) as measured by cardiac catheterization. They also showed that PAAT corrected for RVET, a method of adjusting for the heart rate, slightly strengthened the correlation between PAAT and MPAP.[7] In a study that followed, Dabestani et al described the same relationship and derived the following equation:

$$MPAP = 90 - (0.62 \times PAAT)$$

In contrast to Kitabatake et al showed that both the logarithmic conversion of MPAP and the correction for RVET did not improve the accuracy of estimating the MPAP using PAAT.[8] Together, these two studies established the usefulness of Doppler derived measurements of pulmonary arterial forward flow in providing an accurate estimate of MPAP. In spite of the above vital publications, the use of PAAT in clinical practice had so far remained quite limited.

This is largely because the vast majority of the publications reporting echocardiographic pulmonary artery pressure estimates had relied on TR Vmax-derived EPASP. As such, physicians who depend on TTE for pulmonary artery pressure measurement have developed a familiarity with and also a preference for peak systolic, and not mean, pulmonary artery pressure values. Our results and their implication on the usefulness of TTE in reviewing the pulmonary hemodynamic non-invasively can be summarized as follows. First, TR Vmax was not measurable in 8.7% of patients, while PAAT was measurable in 100% patients. This percentage may increase further if patients with borderline elevation of PA pressures were included in the study. It has been shown that RV function can affect PAAT. RV systolic

dysfunction tends to lengthen the PAAT.[9] In our study population, only 3 patients had RV dysfunction, and their removal and reanalysis of data did not affect our correlation results. Because of the small number of patients with RV dysfunction in our study, we propose that our derivations from the study may not be accurate in patients with RV systolic dysfunction. The imaging technique plays an important role in the measurement of PAAT that accurately reflects pulmonary pressure. Proper placement of the Doppler sample volume in the middle of the pulmonary artery and also its accurate alignment to the long axis of the main pulmonary artery are essential.

Limitations

Limitations in this study include: the total number of cases include in our study was small; we did not assess PAAT for the estimation of EPASP in conditions with high cardiac output, like exercise or systemic vasodilation as in sepsis; the total number of cases due to lung disease, chronic thromboembolic PH and due to idiopathic PH was small in our study and; the placement of Doppler sample volume during pulse-wave and continuous wave Doppler examination at the centre of the pulmonary artery is vital to optimize the laminar flow pattern. Any deviation from this technique, like placing the sample volume close to the pulmonary artery wall, may change this flow pattern and thus render it unreliable.

CONCLUSION

PAAT is easily quantifiable in patients with pulmonary hypertension and strongly correlates with the values of pulmonary artery systolic pressure and the mean pulmonary artery pressure obtained by right heart catheterisation in a small population of patients with pulmonary hypertension due to a wide spectrum of illness. This is comparable to the strong correlation between the previously well-established method of deriving the EPASP from TR Vmax and the invasively derived PASP. Adoption of a novel method of determining EPASP from PAAT shall increase significantly the number of patients in whom TTE can be used for the assessment of pulmonary hemodynamic non-invasively.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. The consensus agreement of experts, the fourth World Symposium on PH held in 2008 in Dana Point, California. Available at: http://www.4thworldphsymposium.com. Accessed on: 24 December 2020.
2. Habib G, Torbicki A. The role of echocardiography in the diagnosis and management of patients with pulmonary hypertension. Eur Respir Rev. 2010;19(118):288-99.
3. Milan A, Magnino C, Veglio F. Echocardiographic indexes for the non-invasive evaluation of pulmonary hemodynamics. J Am Soc Echocardiogr. 2010;23(3):225-39.
4. Brennan JM, Blair JE, Goonewardena S, Ronan A, Shah D, Vasaiwala S, Kirkpatrick JN, Spencer KT. Reappraisal of the use of inferior vena cava for estimating right atrial pressure. J Am Soc Echocardiogr. 2007;20(7):857-61.
5. Chan KL, Currie PJ, Seward JB, Hagler DJ, Mair DD, Jamil Tajik A. Comparison of three Doppler ultrasound methods in the prediction of pulmonary artery pressure. J Am Coll Cardiol. 1987;9(3):549-54.
6. Currie PJ, Seward JB, Chan KL, Fyfe DA, Hagler DJ, Mair DD, Reeder GS, Nishimura RA, Tajik AJ. Continuous wave Doppler determination of right ventricular pressure: a simultaneous Doppler-catheterization study in 127 patients. J Am Coll Cardiol. 1985;6(4):750-6.
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(2):302-9.
8. Mahan G, Dabestani A, Gardin J, Allfie A, Burn C, Henry W. Estimation of pulmonary-artery pressure by pulsed doppler echocardiography. Circulation 1983;68(4):367.
9. 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(3):653-62.

Cite this article as: Khese VB, Chavan CB, Kalra R, Allam AK, Mohabey A. Assessment of pulmonary artery hypertension by Doppler echocardiography and its correlation with right heart catheterization. Int J Adv Med 2021;8:523-9.