**REVIEW ARTICLE**

# Transesophageal Echocardiographic Approach to a Patient with Suspected Pulmonary Hypertension in the Intraoperative Period

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## ABSTRACT

Pulmonary hypertension (PH) defined by a mean pulmonary artery pressure (MPAP) >25 mm Hg at rest is confirmed by right heart catheterization (RHC). With the rapid development of surgical methods and cardiopulmonary bypass strategies, many children and adults are undergoing surgical correction for their cardiac pathologies. Presence of PH in these patients contributes to a major morbidity and poses a great challenge for the operative team during its surgical and postoperative management. With the universal use of transesophageal echocardiography (TEE) in almost all cardiac surgeries, the etiology, severity of PH, and its effect on cardiac structures and function can be evaluated in patients suspected of having PH. Since there is no established algorithm for the intraoperative TEE evaluation of such patients, we proposed an algorithm for the evaluation of PH in patients suspected of having raised pulmonary pressure using TEE in the intraoperative period.

**Keywords:** Pulmonary hypertension, Right ventricle in pulmonary hypertension, Transesophageal echocardiography.

## INTRODUCTION

Pulmonary hypertension is a pathophysiological condition characterized by MPAP > 25 mm of Hg at rest, measured by RHC.[1] A variety of pathological conditions involving the heart, lungs, and connective tissue are associated with elevation of MPAP. Even though all conditions lead to the common endpoint of elevation in MPAP, the clinical feature and treatment depend on the primary etiology of the conditions. The World Health Organization (WHO) has classified PH into five groups, pulmonary arterial hypertension (PAH), PH due to left heart disease, PH due to lung diseases and/or hypoxia, PH associated with chronic thromboembolism, and PH with unclear multifactorial mechanisms (Table 1).[2] For better and

**Table 1:** World Health Organization classification of pulmonary hypertension

| Type | Classification of pulmonary hypertension |
| --- | --- |
| 1 | *Pulmonary arterial hypertension* |
| 1.1 | Idiopathic PAH |
| 1.2 | Heritable PAH |
| 1.2.1 | BMPR2 |
| 1.2.2 | ALK1, ENG, SMAD9, CAV1, KCNK3 |
| 1.2.3 | Unknown |
| 1.3 | Drug and toxin induced |
| 1.4 | Associated with |
| 1.4.1 | Connective tissue disease |
| 1.4.2 | HIV infection |
| 1.4.3 | Portal hypertension |
| 1.4.4 | Congenital heart disease |
| 1.4.5 | Schistosomiasis |
| 1' | Pulmonary veno-occlusive disease and/or pulmonary capillary hemangioma |
| 1" | Persistent pulmonary hypertension of the newborn |
| 2 | *Pulmonary hypertension due to left heart disease* |
| 2.1 | Left ventricular systolic dysfunction |
| 2.2 | Left ventricular diastolic dysfunction |
| 2.3 | Valvular disease |
| 2.4 | Congenital/acquired left ventricle inflow/outflow tract obstruction and congenital cardiomyopathies |
| 3 | *Pulmonary hypertension due to lung disease and/or hypoxia* |
| 3.1 | Chronic obstructive pulmonary disease |
| 3.2 | Interstitial lung disease |
| 3.3 | Other pulmonary disease with mixed restrictive and obstructive pattern |
| 3.4 | Sleep disordered breathing |
| 3.5 | Alveolar hypoventilation disorder |
| 3.6 | Chronic exposure to high altitude |
| 3.7 | Developmental lung disease |
| 4 | *Chronic thromboembolic pulmonary hypertension* |
| 5 | *Pulmonary hypertension with unclear multifactorial mechanism* |
| 5.1 | Hematologic disorders: chronic hemolytic anemia, myeloproliferative disorder, splenectomy |
| 5.2 | Systemic disorders: sarcoidosis, pulmonary histiocytosis, lymphangioleiomyomatosis |
| 5.3 | Metabolic disorders: glycogen storage disorder, gaucher disease, thyroid disorder |
| 5.4 | Others: tumoral obstruction, fibrosing medistinitis, chronic renal failure, segmental PH |

BMPR: Bone morphogenic protein receptor; CAV1: Caveolin 1; ENG: Endoglin; HIV: Human immunodeficiency virus; PAH: Pulmonary arterial hypertension

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simpler understanding, we can consider PH as conditions involving precapillary or postcapillary pulmonary vasculature. As per the WHO classification, groups 1, 3, 4, and 5 involve precapillaries, whereas group 2 involves postcapillary vasculature, leading to elevation in MPAP.[3] In PAH, the elevation in MPAP may be due to relative blood flow obstruction proximal to the lung capillary bed and/or increased pulmonary vascular resistance (PVR).[4] Postcapillary PH can be classified further based on the transpulmonary pressure gradient (TPG) as passive (TPG ≤ 12 mm Hg) or active (TPG > 12 mm Hg),[4] in which the active form has a PH out of proportion to what is expected based on the severity of left heart disease as in mitral valve disease and advanced cardiomyopathy (Table 2).[4] The accurate measurement of precapillary and postcapillary pulmonary pressures mandate the RHC, which forms the gold standard for the diagnosis of PH.[5] Despite recent advances in the catheterization procedures with minimal to no complications in the RHC, the invasive nature and the need of laboratory limit its use in evaluating these patients. With the advent of cardiac surgery in the early 1950s, many patients including children with various lesions undergo surgical correction, in which the accurate diagnosis of PH with its etiology and severity and the consequences of PH on right heart function play an important role in management. Echocardiography forms a valuable tool in these aspects, particularly the TEE, which can be used to evaluate the severity of PH along with clues suggesting the etiology and effect of PH on right ventricular (RV) function in the intraoperative period. Since there is no established protocol for the TEE evaluation of patients suspected of PH in the intraoperative period, we aimed to form a protocol for the same (Flow Chart 1). We will focus on three headings, when to suspect for the presence of PH

based on clinical condition, echocardiographic views to assess the presence of PH, and evaluation of its severity with its limitations and consequences of PH on RV function, which forms a prognostic role in the patient outcome in postoperative period.

## SUSPECT PH

### Signs/Symptoms

The PH usually has an insidious presentation, with dyspnea on exertion being the most common initial symptom.[6] The other symptoms include orthopnea, paroxysmal nocturnal dyspnea (PND) in case of type II PH, hemoptysis, history of venous or arterial thrombosis in type IV PH, connective tissue disorders like systemic lupus erythematosus (SLE), scleroderma and symptoms related to RV dysfunction like early satiety, abdominal distension, pedal edema caused by congestion of the venous system, and angina due to severe RV hypertrophy, and supply demand mismatch to the RV.[7] The vital signs suggestive of the presence of PH include low blood pressure in case of depressed RV function in severe PH, reduced saturation on exercise, elevated V wave in jugular venous pulse due to associated tricuspid regurgitation (TR), presence of parasternal heave suggesting RV hypertrophy, presence of murmur due to TR and/or pulmonary regurgitation (PR) and due to shunts like ventricular or atrial septal defects (VSD/ASD) in case of congenital heart disease (CHD) on auscultation, and hepatomegaly on abdominal palpation.[8] While suspecting PH based on clinical signs and symptoms, the other probable causes of the symptoms like RV outflow tract obstruction (RVOTO) in case of RV hypertrophy and primary pathology of tricuspid valve (TV) and pulmonary valve (PV) causing TR and PR should be kept in mind and should be ruled out in subsequent investigation.

### Laboratory Investigations

The laboratory reports which favor PH include presence of polycythemia, an indirect evidence of desaturation, elevated liver enzymes due to backward congestion in case of RV dysfunction and elevated brain natriuretic peptide level[9] and uric acid level suggesting increased PVR.[10] The presence of dilated pulmonary artery (PA) with peripheral pruning and relatively oligemic lung fields, globular heart due to RV enlargement in chest X-ray (CXR) (Fig. 1), encroachment of retrosternal air space in lateral CXR, RVH and right axis deviation, RV strain pattern in electrocardiogram (ECG) (Fig. 2), and presence of major aortopulmonary collateral arteries in aortography in case of CHD should raise suspicion of PH.[11] In addition, the presence of atrial fibrillation and symptoms related

**Table 2:** Subclassification of PH

| Definitions | Characteristics | Clinical groups |
|---|---|---|
| PH | MPAP ≥ 25 mm Hg | All |
| Precapillary PH | MPAP ≥ 25 mm Hg | PAH |
| | PCWP ≤ 15 mm Hg | PH due to lung disease |
| | CO normal or reduced | Chronic thromboembolic PH |
| | | PH with unclear and/or multifactorial mechanism |
| Postcapillary PH | MPAP ≥ 25 mm Hg PCW > 15 mm Hg | PH due to left heart disease |
| | CO normal or reduced | |
| Passive | TPG ≤ 12 mm Hg | |
| Reactive (out of proportion) | TPG > 12 mm Hg | |
| TPG = MPAP-mean PCWP | | |

**Flow Chart 1:** Transesophageal echocardiographic algorithm in patients with suspected PH
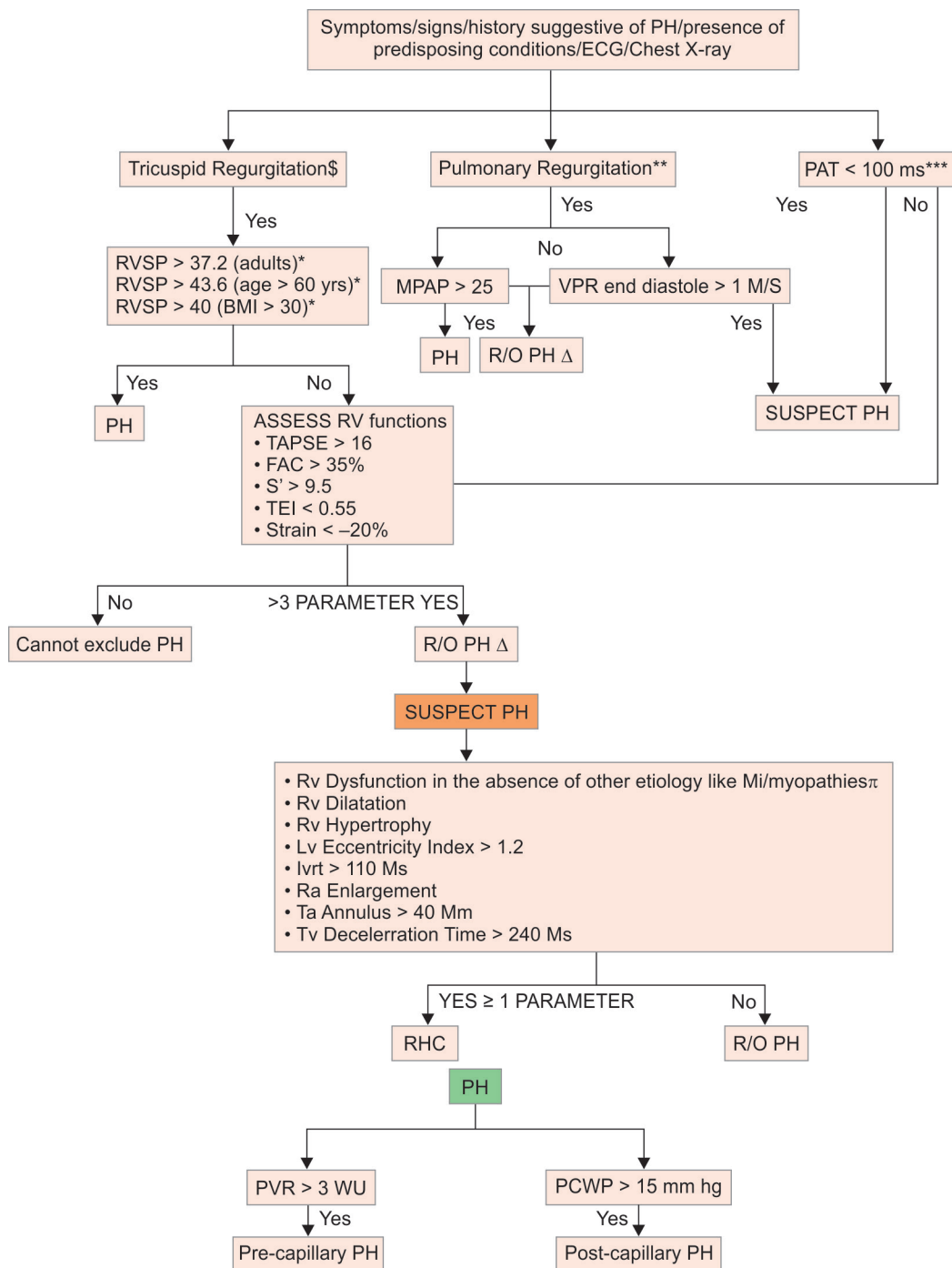


$Rule out RVOTO/intraventricular shunts; *RVSP = 4 $(V_{TR})^2$ + RAP from CVP; **MPAP = 4 $(V_{PR}$ early diastole)$^2$ + RAP; **DPAP = 4 $(V_{PR}$ end diastole)$^2$ + RAP; ***PAT to be corrected for HR (<60/>100) and if PAT> 100 ms—assess RV function [HR corrected PAT = (measured PAT × 75)/patients' HR]; $^\Delta$Only when there is no RV diastolic dysfunction; $^¥$PVR = [10 × $(V_{TR}$/ RVOT VTI)] + 0.16 or PVRI = 9 – 0.07 × PAT; $^£$PCWP = 1.9 + 1.24 × E/E' of MV (OR) from PA catheter; $^\pi$Using parameter given in box with ^

to pulmonary congestion like orthopnea, PND in the absence of right axis deviation, and RVH in ECG suggest the possibility of PH due to left heart disease like mitral valve (MV) and pulmonary venous pathologies.[8]

## Intraoperative Findings

In the intraoperative period, in patients with unknown pulmonary pressure status, the clues suggesting the presence of PH include presence of shunt lesions like ASD/VSD/patent ductus arteriosus (PDA) and aortopulmonary window/tricuspid atresia/partial or total anomalous pulmonary venous connections, which are associated with increased pulmonary blood flow, presence of left ventricular disease like mitral stenosis/regurgitation (MS/MR), which forms one of the most leading causes of PH, presence of masses in the right heart like myxoma,
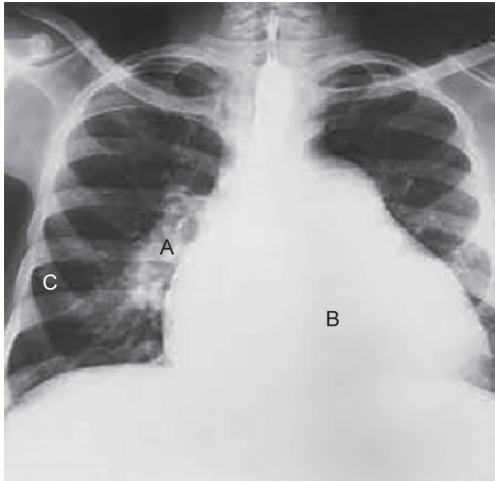
**Figs 1A to C:** Chest X-ray showing prominent pulmonary right artery (A); hypertropied RV (apex shifted left and upward) (B) and oligemic lung fields (C)

thrombus that forms a potential source of recurrent embolic load to the pulmonary circulation, and presence of thrombus in main/left/right pulmonary arteries causing type IV PH. Other findings that prompt for further evaluation of PH includes RV hypertrophy, the circular shape of RV on systole, and dilated RV on diastole, indicating pressure and volume overload respectively, shift of interventricular septum (IVS) toward left ventricle (LV), features suggesting of depressed RV function like presence of TR, reduced RV contractility and presence of PR, and presence of pericardial effusion.[12,13]

## Echocardiographic Analysis for Diagnosis and Assessment of Severity and Consequences of PH

The TEE forms a valuable tool in estimation of PA pressure (PAP) and PVR and helps in diagnosis and classification of PH in the intraoperative period, in patients in whom the PH is suspected and diagnosis not

established by gold standard RHC. While approaching these echocardiographic-based methods in establishing diagnosis, the limitations of the two-dimensional and Doppler methods used should be kept in mind. To increase the accuracy of this method, multiple parameters should be analyzed including the clinical symptoms and signs and other investigational reports like ECG and CXR. Despite this adherence, this method may not be able to detect the PH in few patients like patients with connective tissue disorders like scleroderma or SLE, which necessitates the gold standard tool, i.e., RHC.

Since the definition of PH is based on the MPAP,[1] the initial step in TEE should be the estimation of pressures in the right heart chambers and pulmonary vascular system from which the MPAP, systolic PAP (SPAP), and diastolic PAP (DPAP) can be derived from the Bernoulli's equation, according to which the pressure gradient across a point will be four times the square of the velocity of flow across that point.

## Estimation of SPAP

On applying the above principle to the flow velocity across the TV in TR, the RV systolic pressure (RVSP) can be estimated based on the following formula,

$$\text{RVSP} = 4 \times (\text{peak velocity of TR})^2 + \text{RAP}^{14}$$

where RAP is right atrial pressure, TR is tricuspid regurgitation, which, in turn, equals the SPAP provided there is no obstruction in RVOT and no shunt between RV and pulmonary vasculature.

This can be obtained by placing the Doppler sample volume at the tip of the TV leaflet and applying continuous wave Doppler from which the peak TR velocity can be measured. The major limitations of this method of estimating SPAP is the requirement of TR for evaluation,
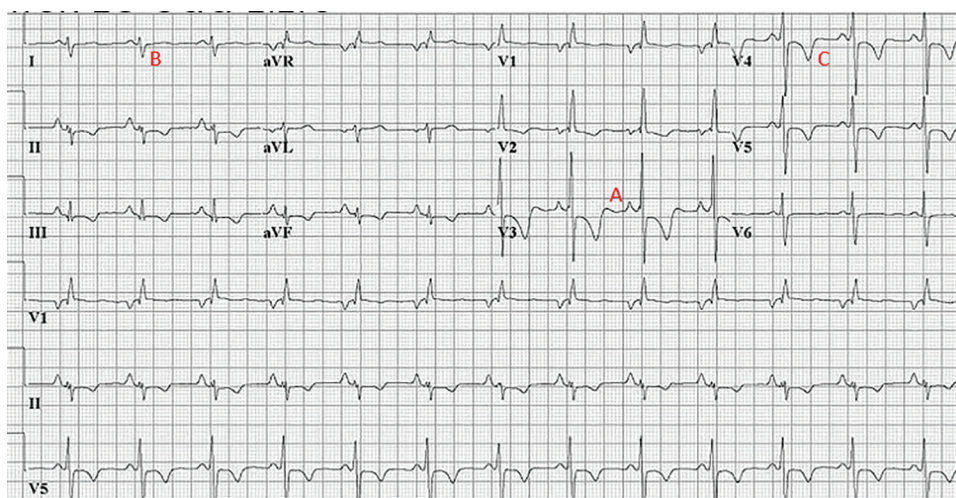


**Figs 2A to C:** Electrocardiogram of pulmonary hypertension patient showing RVH (A), right axis deviation (B) and RV strain indicated by T wave inversion in anterior chest leads (C)

which is available only in 60 to 70% of patients with PH[15] and the direction of the regurgitated jet should be in line with the Doppler. The alignment can be increased by applying color Doppler across the TV to identify the direction of the TR jet and placement of continuous wave Doppler in that direction in multiple TEE views (Fig. 3). The another limitation of this method is in the case of severe TR in which the right atrium (RA) pressures equalize very rapidly, with the RVSP leading to reduced peak TR velocity, and in case of Gerbode shunt, in which the TR velocity may estimate the LV systolic pressure (LVSP).[16] Finally, the limitations in the measurement of RAP also should be considered. This formula holds true in the evaluation of RVSP when the above limitations are taken care of even in patients with primary TV pathology and RV dysfunction, in which case the SPAP is low because of reduced flow in the pulmonary system despite having PH defined in terms of PVR > 3 Woods unit (WU).

The different TEE views used for this purpose include midesophageal four chamber (ME4C), RV inflow–outflow (RVI/O), transgastric RV inflow (TGRVI), transgastric RVI/O (TGRVI/O), and deep transgastric long axis (DT-GLAX) views. The view with better alignment with maximum peak velocity can be taken into account for the estimation of RVSP. Studies have shown a positive correlation between SPAP and MPAP and MPAP can be derived from the formula, $MPAP = 0.61 \times SPAP + 2$ mm Hg.[17] The normal upper limit of SPAP depends on age and body mass. The estimated upper 95% limit for SPAP was 37.2 mm Hg in low-risk subjects ($V_{TR}$ = 2.6 m/sec), whereas the estimated upper 95% limit for subjects aged > 60 years was 43.6 mm Hg ($V_{TR}$ = 2.9 m/sec) and in patients with a body mass index > 30 $kg/m^2$, the limit was 40 mm Hg ($V_{TR}$ = 2.8 m/sec),[13,18] where $V_{TR}$ is the peak velocity of TR jet.

In case of shunt lesions like VSD, by measuring the peak velocity across the shunt and LVSP, which is equal to the systemic blood pressure measured from invasive catheter in the absence of any LV outflow tract (LVOT) or aortic valve obstruction, the RVSP can be calculated using the formula, RVSP = LVSP/systolic blood pressure $- 4$ (peak velocity across the shunt)$^2$ (Fig. 4). The major limitation here is getting the proper alignment between
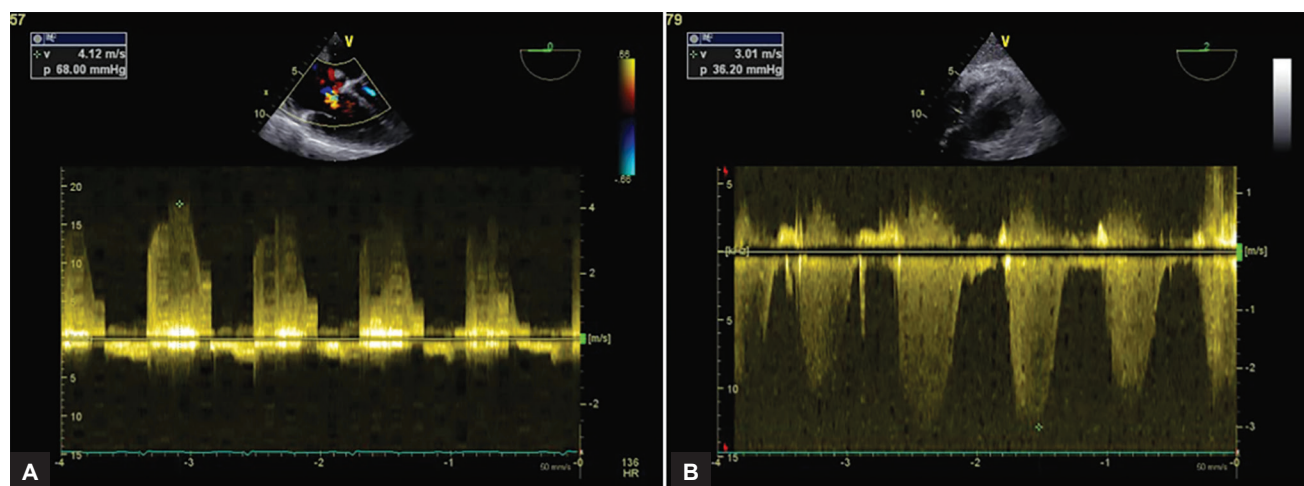


**Figs 3A and B:** Peak systolic gradient across tricuspid valve measured from regurgitation jet in ME4C (A) and DTG (B) views
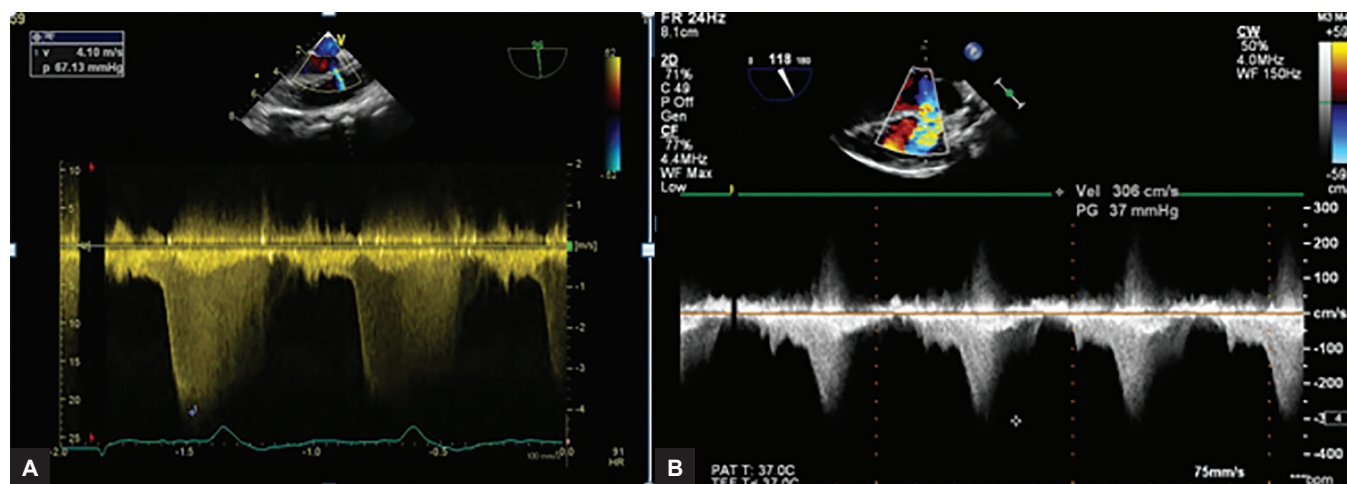


**Figs 4A and B:** Peak systolic gradient across VSD measured in MELAX view

the flow and Doppler beam, which can be obtained in transgastric two-chamber view in few patients.

## Estimation of RAP

The RAP can be reliably measured from the central venous catheter inserted for the surgical procedure at the end expiration, which can be used to measure SPAP/RVSP from the previously discussed formula. In case of absence of CVP monitor, it can be measured directly from the RA by inserting a needle with a pressure transducer attached to it. The measurement of RAP from the inferior vena cava (IVC) diameter and its collapsibility in the spontaneously breathing patient[19] are not validated in the mechanically ventilated patient. In the nonavailability of the above measures, the RAP can be estimated approximately by evaluation of TV inflow pattern in which the systolic filling fraction (systolic VTI/[systolic VTI + diastolic VTI]) < 0.55, where VTI is the velocity time integral and on hepatic flow pattern in which the ratio of systolic to diastolic flow velocity < 1 (Fig. 5) and TV annulus E/E' > 6 indicates elevated RAP > 10 mm Hg.[20] The TV E', E velocity, and VTI across TV can be obtained from the ME4C, TGRVI, TGRVI/O, or DTGLAX views (Fig. 6). Similar to any Doppler evaluation, the alignment should be < 20° for accurate estimation of E/E'. Hepatic venous flow velocity can be obtained by placing the pulse Doppler sample volume within the mouth of hepatic vein



**Fig. 5:** Hepatic vein systolic and diastolic forward flow velocity measured using TEE

opening into the IVC with Doppler alignment that can be obtained by rotating the TEE probe to right side from the transgastric basal short-axis view and increasing the omni-plane angle to 60 to 70°.

Once the measured RVSP/SPAP > 37.2 mm Hg or $V_{TR}$ > 2.6 m/sec, we can proceed further for the evaluation of severity and type of PH. The absence of RVSP > 37.2 does not exclude PH as in the case of poor RV function, it will be low due to inability of RV to produce such pressure in the cavity in which case the measurement of PVR and other parameters should be carried out.

## Estimation of MPAP and DPAP

Based on the simplified Bernoulli's equation, the pressure gradient across the PV can be measured from PR and the MPAP and DPAP can be calculated from the early diastolic PR velocity and end diastolic PR velocity respectively, using the formula,

$$MPAP = 4(V_{PR} \text{ early diastole})^2 + RAP^{21}$$

$$DPAP = 4(V_{PR} \text{ end diastole})^2 + RAP^{14}$$

The limitations with this method are requirement of PR and proper alignment of Doppler with regurgitant flow direction similar to any Doppler measurement.

In TEE, the presence of PR and measurement of the pressure gradient can be obtained from placing continuous wave Doppler sample volume at the PV leaflet tips at diastole in upper esophageal aortic arch short-axis (UE AA SAX), RVI/O, and TGRVI/O views (Fig. 7).

While measuring MPAP and DPAP, the diastolic function of RV should be considered, as in case of RV diastolic dysfunction, the peak velocity of early and late PR jets will be low due to increased pressure in the RV cavity, which leads to underestimation of MPAP and DPAP. In case of severe PR, as with severe TR, there may occur rapid equalization of PA and RV pressure leading to low-velocity profile of PR causing underestimation of PAP.

Pulmonary artery acceleration time (PAT) is the time interval between the start to peak of pulmonary artery flow measured from pulse wave Doppler flow pattern. In general, the shape of the Doppler pattern will be
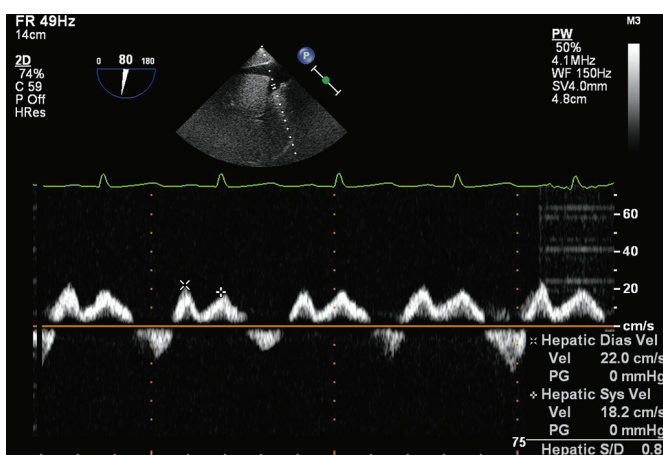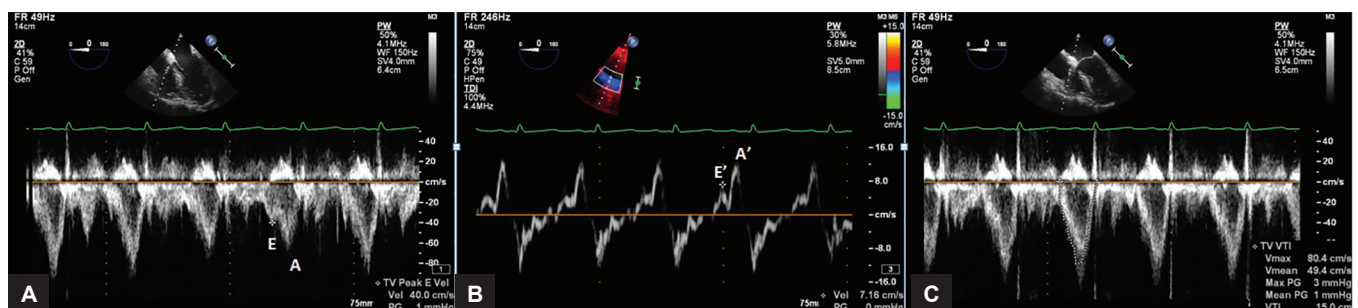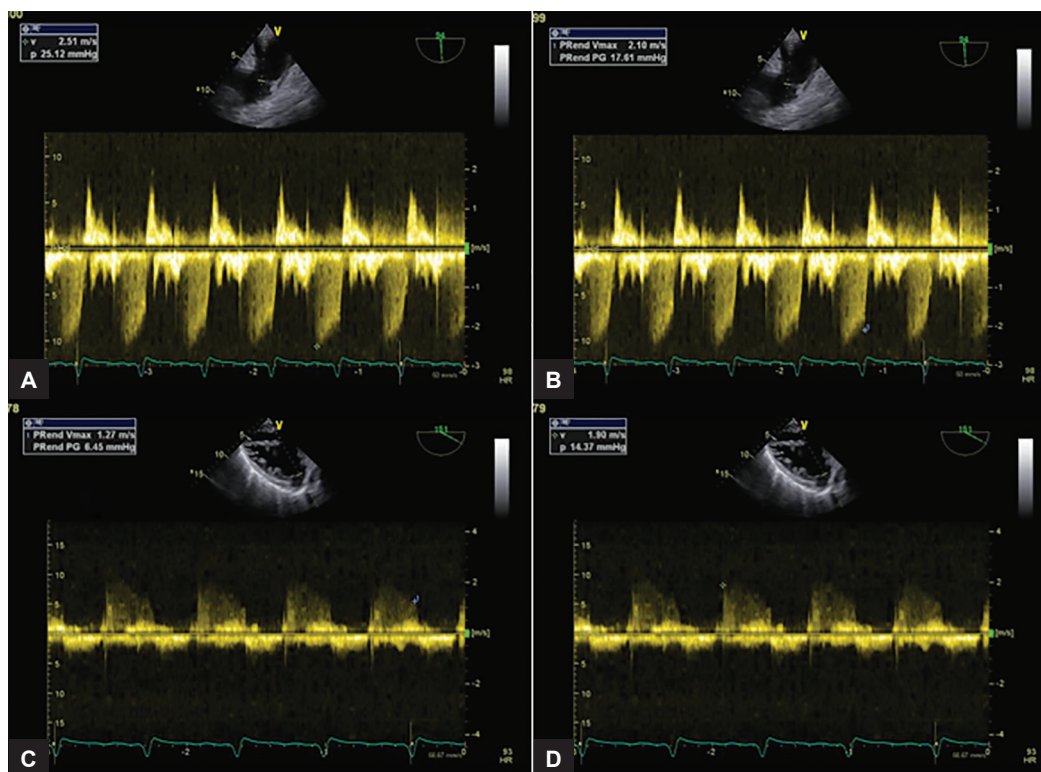


**Figs 6A to C:** Tricuspid valve E velocity: (A) E'; (B) VTI; and (C) measured in ME4C view

**Figs 7A to D:** Continuous Doppler wave applied across the pulmonary regurgitant jet measuring early (A,D) and end (B,C) pulmonary regurgitant velocity in UE AA SAX (A,B) and TGRVI/O (C,D) views

parabolic with smooth contour due to the low pressure system of pulmonary vasculature in comparison with the triangular appearance with early peak in case of Doppler pattern obtained In LVOT. As the PH develops, the Doppler pattern of RVOT also changes its shape similar to LVOT Doppler pattern, with early peak with midsystolic closure.[22] Many studies have identified a positive correlation with PAT and MPAP and various formulas relating PAT and MPAP have been derived by regression equation both in adults and children

$$MPAP = 79 - (0.45 \times PAT)^{23}$$

$$MPAP = 90 - (0.62 \times PAT)^{22}$$

$$MPAP = 48 - (0.28 \times PAT)^{24}$$

$$Log10\ MPAP = 0.0068\ PAT + 2.1^{25}$$

The dependence of RV ejection time on heart rate (HR) mandates the correction of PAT for patient's HR, which can be done using the formula

$$Corrected\ PAT = (measured\ PAT \times 75)/patient\ HR^{13}$$

The advantages of PAT over other measures include the easy availability of Doppler pattern in RVOT in every patient and the more sensitive nature of PAT than $V_{TR}$ to early or latent pulmonary vasculopathy,[26] which may be due to its sensitivity to changes in pulmonary vascular impedance more than to PVR.[27] However, HR and sample location dependency have limited its utility, which can be corrected using HR-corrected PAT when

HR <60 or >100/min and placement of sample volume in the middle of RVOT.

The PAT > 100 ms suggests that there is no PAH, whereas a PAT < 100 ms increases the probability of PAH in adults and PAT < 60 ms signifies the presence of severe PAH (MPAP > 50 mm Hg).[28]

A third method for the estimation of MPAP is from the mean systolic gradient between RA and RV from the formula, MPAP = RAP + $V_{TR}$ mean gradient.[29]

While measuring MPAP from PAT, the RV function should be evaluated because poor RV function in itself reduces the RV ejection time (RVET) and, thereby, PAT leading to overestimation of MPAP, which can corrected by using PAT corrected for RVET.

## ESTIMATION OF PULMONARY VASCULAR RESISTANCE

Since in the definition of PH, PVR > 3 WU in the presence of MPAP > 25 mm Hg and pulmonary capillary wedge pressure (PCWP) < 15 mm Hg is called precapillary PH, the estimation of PVR by TEE adds to the positive predictability of PH. The PVR is defined by the ratio of TPG to the transpulmonary flow.[30]

As PVR indicates the afterload against which the RV ejects, when PVR increases, the enhanced and early reflection of the pressure wave profile of the RVOT appears leading to changes in RVOT VTI and increase
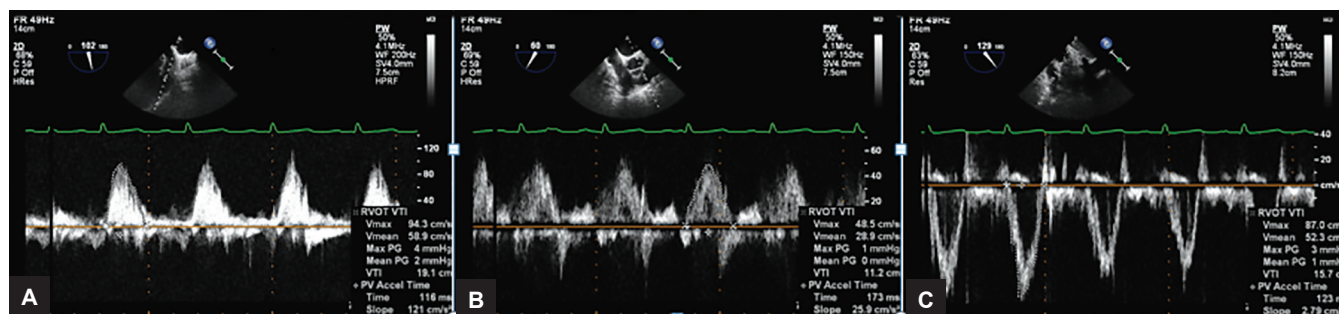
**Figs 8A to C:** The RVOT VTI and PAT measured by pulse wave Doppler with sample volume in the middle of RVOT in UE AA SAX (A); RVIO (B); and TGRVI/O (C) views

in the TR velocity due to increase in PAP associated with increase in PVR.[30-33] Many studies have showed a negative correlation between PVR and RVOT VTI and positive correlation between PVR and TR velocity.[30,34,35] The equation PVR = [10 × ($V_{TR}$/RVOT VTI)] + 0.16 was shown to provide a good estimate of PVR, when PVR < 6 WU. The ratio $V_{TR}$/RVOT VTI > 0.175 is considered abnormal and suggests an elevated PVR (>2 WU), while $V_{TR}$/RVOT TVI > 0.275 indicates a PVR > 6 WU.[36]

In TEE, the RVOT VTI and PAT can be measured by placing the pulse wave Doppler sample volume in the RVOT just below the pulmonary annulus to get the Doppler pattern with proper alignment with bloodflow either in UE AA SAX, RVI/O, or TGRVI/O (Fig. 8). The limitations of this method in estimating PVR include reliability in patients with poor RV function and limitations associated with the TR velocity measurements.

Similarly, PAT can be used as a surrogate for PVR, as increase in PVR is associated with decrease in PAT and, by regression analysis, PVR can be calculated from the PAT using formula, PVRI = 9 – 0.07 × PAT.[24] In particular, PAT <90 ms can identify PVR ≥3 WU with 84% sensitivity and 85% specificity.[29]

From the above discussion, a PAT < 90 ms or ratio $V_{TR}$/RVOT VTI > 0.175 suggest the possibility of elevated PVR, and in turn, PH.

Once we confirmed the presence of elevated MPAP > 25 mm Hg, and/or PVR > 3 WU, the next step is to grade the severity, which is done based on the MPAP (Table 3) and look for the cause of PH and the consequences of PH, which ultimately decide the patient management and outcome.

Even when the measurement of the above determinants does not meet the criteria, PH cannot be ruled out as there are possibilities of underestimation of the MPAP due to the above-explained limitations associated with echocardiography, during which in the presence of clinical suspicions of PH, MPAP should be measured by direct monitoring of PA pressure invasively in the operating room.

## Transesophageal Echocardiography in determining the Cause of PH

Once PH is diagnosed, the next step is to look for the probable etiology, which plays a role in the further management of the patient. Since PCWP and PVR differentiates PH between precapillary and postcapillary PH, in which the precapillary PH has raised PVR and postcapillary has raised PCWP > 15 mm Hg, the measurement of these parameters aids in the classification. Even though the measurement of PVR as explained above can help in classification, it is not free of limitations as explained. Few studies in the past have suggested echocardiographic scales to discriminate between precapillary and postcapillary PH.[37] Left atrial (LA) anteroposterior (AP) diameter < 3.2 cm (score +1), midsystolic notch in RVOT Doppler pattern or PAT < 80 ms (+1) and E/E′ >10 of mitral valve lateral annulus (–1) and LA AP diameter > 4.2 cm (–1) as measured in TEE, and the final total score positively correlated with the PVR and a score > 0 identifies raised PVR and, thereby, precapillary PH with a sensitivity of 100% and specificity of 63%.[37] In the absence of mitral valve pathology, PCWP can be measured using the formula, PCWP = 1.9 + 1.24 × E/E′,[38] where E indicates the early diastolic mitral inflow velocity measured by pulse wave Doppler examination of mitral valve in midesophageal long-axis view (MELAX) view and E′ indicates the lateral annulus early diastolic velocity measured by tissue Doppler examination of the lateral MV annulus in ME4C view (Fig. 9). Once PCWP and PVR are measured, it helps in narrowing our etiology.

Few causative factors of PH along with the history of clinical features will lead to its classification like presence of CHD with increased pulmonary blood flow leading to type I or PAH, presence of left heart disease including

**Table 3:** Severity classification of pulmonary artery hypertension

| MPAP (mm Hg) | Grade |
|---|---|
| >25–40 | Mild PAH |
| 41–50 | Moderate PAH |
| >50 mm Hg | Severe PAH |
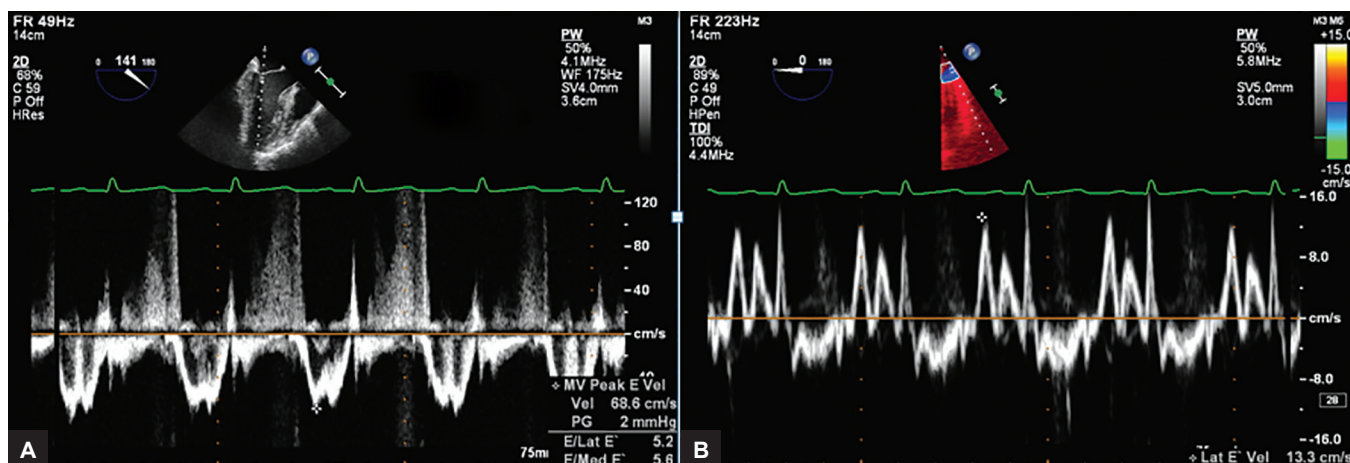
**Figs 9A and B:** Mitral valve E velocity measured by pulse wave Doppler across MV opening in MELAX (A) and lateral E' velocity measured by pulse wave Tissue Doppler in the lateral annulus in ME4C (B) view

MS/MR/pulmonary venous obstruction directing to type II PH, presence of clinical features of chronic lung disease with CXR features directing to type III PH, and presence of features of chronic thromboembolisms with history of deep vein thrombosis/renal cell carcinoma/right-sided myxoma/thrombus, which causes silent recurrent pulmonary thromboembolisms directing to type IV PH.

While this approach aids in the diagnosis of PH with probable etiology, it is not completely foolproof as a patient may present with multiple causative factors contributing to PH, the correct diagnosis of which necessitates RHC, which reliably measures PCWP and PVR.

### Evaluation of Consequence of PH on Right Heart

As we know the measurement of MPAP by TEE has limitations due to the functional status of RV, and at the same time PH affects the performance of RV, knowledge about the RV functional parameter measured by TEE is also essential for the better management of PH patients.

Morphological and functional assessments of RV are more complicated when compared with LV due to its coarse trabeculations, causing difficulty in endocardial border detection and its anterior location behind the sternum. The TEE eliminates the problem associated with its retrosternal position as it examines from esophagus, thus providing better RV assessment than the transthoracic echocardiogram (TTE).[39] Since the RV works against the low-pressure system of pulmonary vasculature, even when both LV and RV produce the same stroke volume, the thickness of RV wall is lesser than LV and the total mass of RV is only 1/5th to 1/6th of LV mass.[19,40] As the pulmonary pressure increases, the thickness of RV increases proportionately, causing RV hypertrophy to a point of compensation after which the further increase in pulmonary pressure causes RV dilatation.[2,41-43] Similarly, the absence of an intermediate

circumferential layer in RV when compared with LV makes the longitudinal movement a predominant contributor, while rotational movement contributes minimally to the ejection.[19]

While assessing the RV, it can be assessed broadly into anatomic parameters and functional parameters.

### Morphological Parameters

As discussed earlier, the normal thickness of RV lateral wall is <5 mm,[44] and as the pressure overload occurs as in PH, it hypertrophies. A thickness of >5 mm measured at the end diastole in the ME4C (Fig. 10) indicates the presence of hypertrophy, and it is positively correlated with RVSP.[45,46] Normally, the apex of the heart is formed by LV and when RV dilates due to chronic PH, the apex will be shared by both the ventricle and with further dilatation, the entire apex of the heart will be formed by RV, which can also be noted in the ME4C view (Fig. 11).[45]
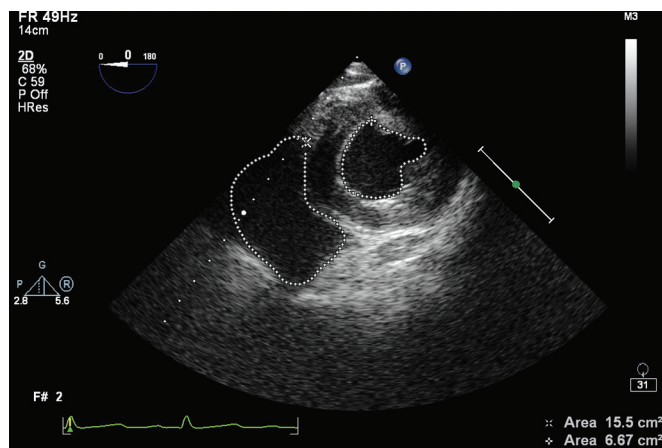
The degree of RV dilatation can be classified based on the ratio of cross-sectional area of RV to LV measured at end diastole in TG mid SAX, as mild when
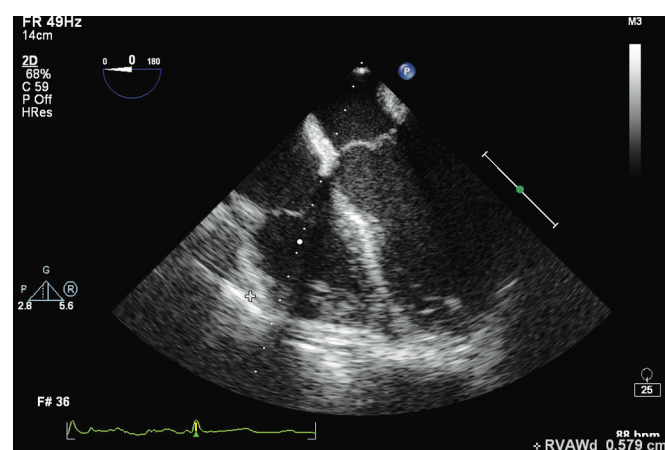


**Fig. 10:** RV free wall thickness measured in ME4C view
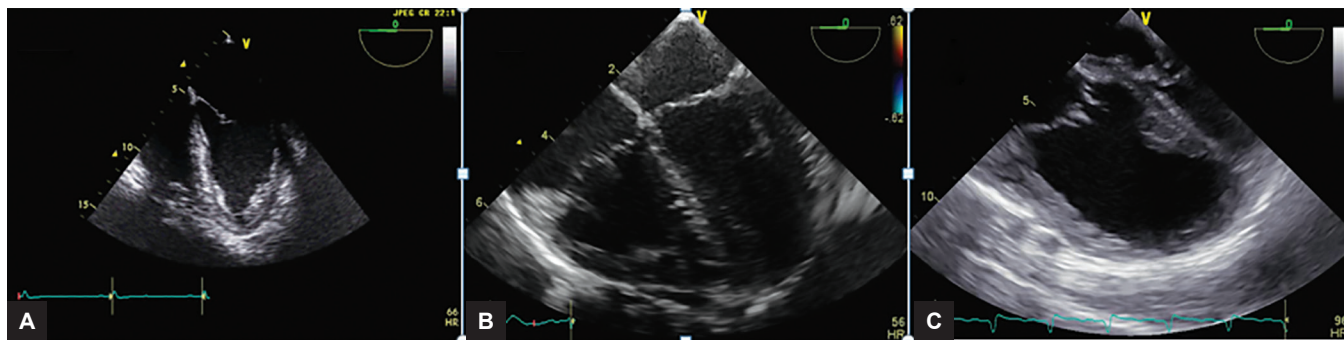
**Figs 11A to C:** ME4C view showing apex of the heart formed left ventricle (A); both the ventricles (B); and right ventricle (C)



**Fig. 12:** Right ventricle and left ventricle end diastolic area measured in TG SAX showing enlarged RV indicating RV volume overload

RV area is 60 to 100%, and moderate when it is 100% or equal, and severe when it is >100% of the LV area.[45] The ratio of RV to LV area is considered to rule out the misinterpretation of global dilatation of heart as RV dilatation (Fig. 12).

The other conditions associated with increase in free wall thickness, such as infiltrative and hypertrophic cardiomyopathies should be kept in mind while performing the measurement.[4,46]

## Functional Parameters

The functional assessment of RV should be analyzed carefully as the RV dysfunction can be the consequence of PH or the calculated pressures in the pulmonary vasculature can be falsely low due to RV dysfunction caused by other primary disease of RV like myocardial infarction or cardiomyopathies.

Tricuspid annular plane systolic excursion (TAPSE), which measures the displacement distance of TV lateral annulus with respect to the RV apex, is shown to be closely correlating with RV ejection fraction (EF)[47] and a value <17 mm is considered abnormal.[27] This can be measured in M mode image of ME4C or TGRVI or TGRVI/O with better alignment, with M mode cursor passing through the TV lateral annulus and RV apex (Fig. 13). The TAPSE changes with growth and increases from preterm infants to healthy adolescents.[48] Reference values of TAPSE measurements in adults and across the pediatric age range are available in the literature.[13] The major limitations with this method are that it measures only the longitudinal function of RV.

Fractional area change (FAC) measured by [(end-diastolic area – end-systolic area)/(end-diastolic area)] × 100, with FAC <35% indicating systolic dysfunction, is a widely used parameter in the assessment of RV
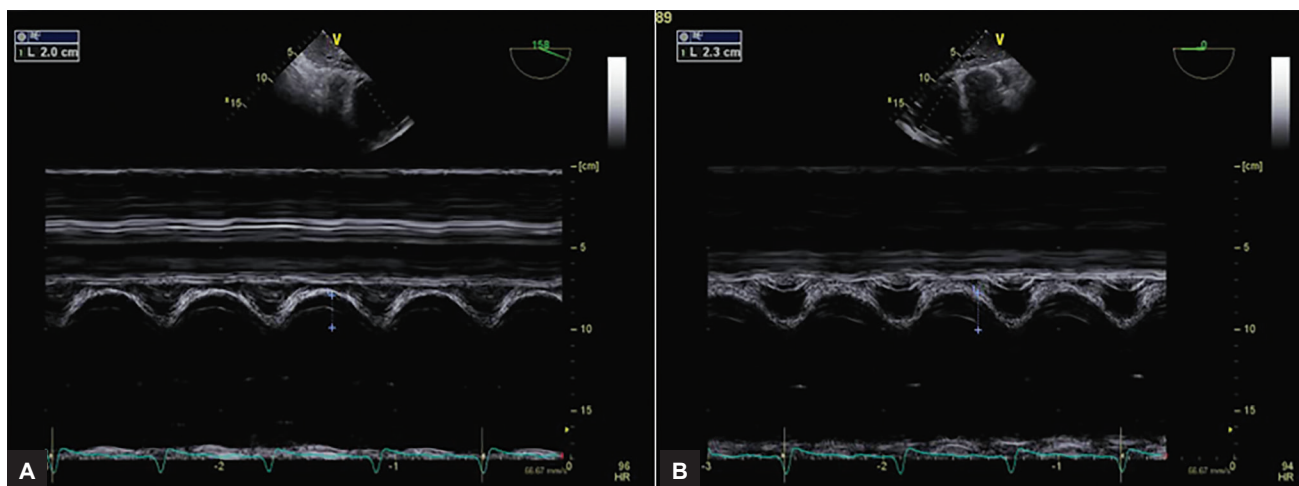


**Figs 13A and B:** The TAPSE measured by M-mode across the lateral annulus of tricuspid valve in TGRVIO (A) and DTG (B) views
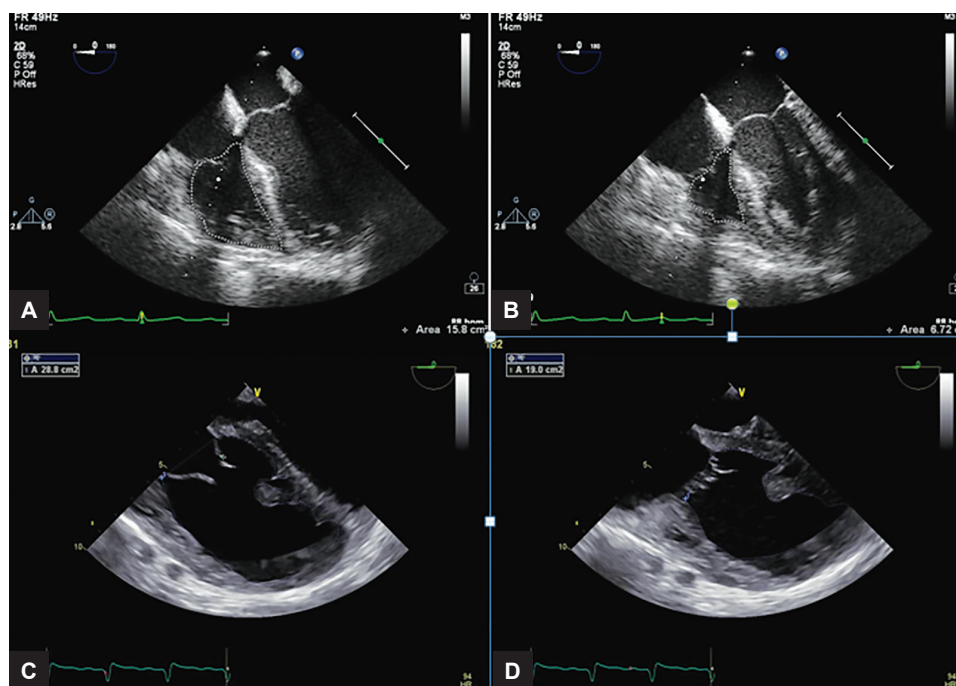
**Figs 14A to D:** Right ventricular end diastolic (A, C) and end systolic (B, D) area measured in two patients for FAC calculation in ME4C view

systolic function, particularly, once the pericardium is opened, and it includes both the longitudinal and radial components of RV function.[14] Studies have shown better correlation of FAC with MRI-measured EF.[46] In TEE, it can be measured by tracing the RV area both in end diastole and end systole in ME4C view (Fig. 14).

The RV S′, which measures the tricuspid annulus systolic excursion, is a measure of longitudinal systolic RV function with <9.5 cm/sec suggesting a decreased RV function. It shares the similar advantage and limitations with that of TAPSE measurement. It is measured by placing the tissue Doppler sample volume at the lateral annulus in the ME4C or TGRVI or TGRVI/O[49] (Fig. 15).

The RV myocardial performance index is a measure of both the systolic and diastolic functions. Even though it can be measured using both pulse wave and tissue Doppler methods, tissue Doppler method minimizes the error related R–R interval. It is obtained by placing the tissue Doppler sample volume at the lateral annulus of TV similar to the measurement of S′, and calculated using the formula (IVRT + IVCT)/RVET, where IVRT is the isovolumic relaxation time, IVCT is isovolumic contraction time, and RVET is RV ejection time. The normal value being <0.54.[14] Studies have shown good correlation of this index with the patient outcome in PH[50] (Fig. 16).

With the recent developments in strain and strain rate, interest had developed in using this parameter
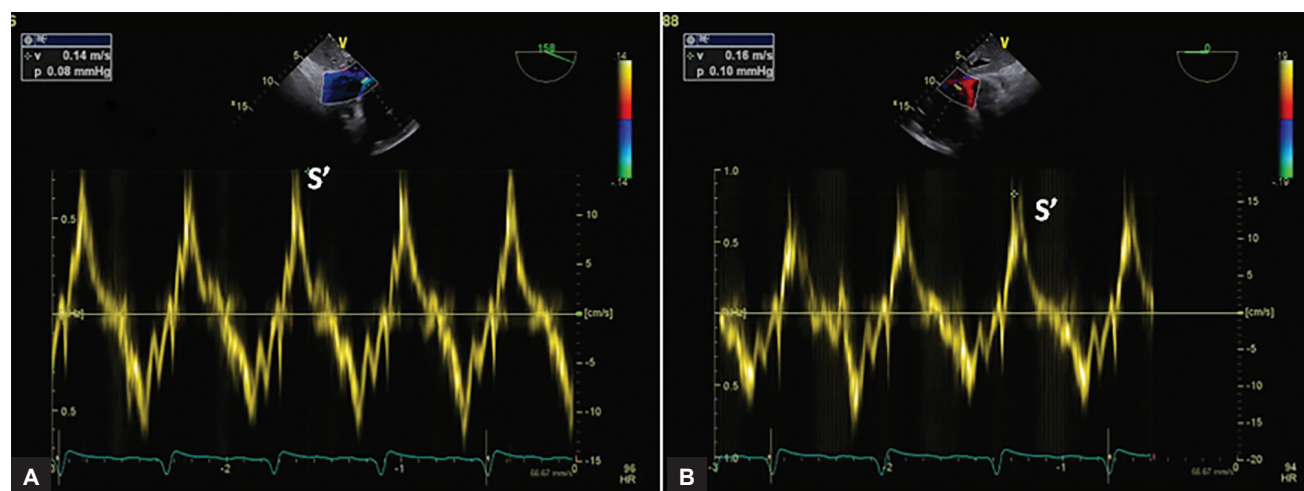


**Figs 15A and B:** Tricuspid valve S′ velocity measured by tissue Doppler imaging with pulse wave sample volume placed in the lateral tricuspid annulus in TGRVIO (A) and DTG (B) views
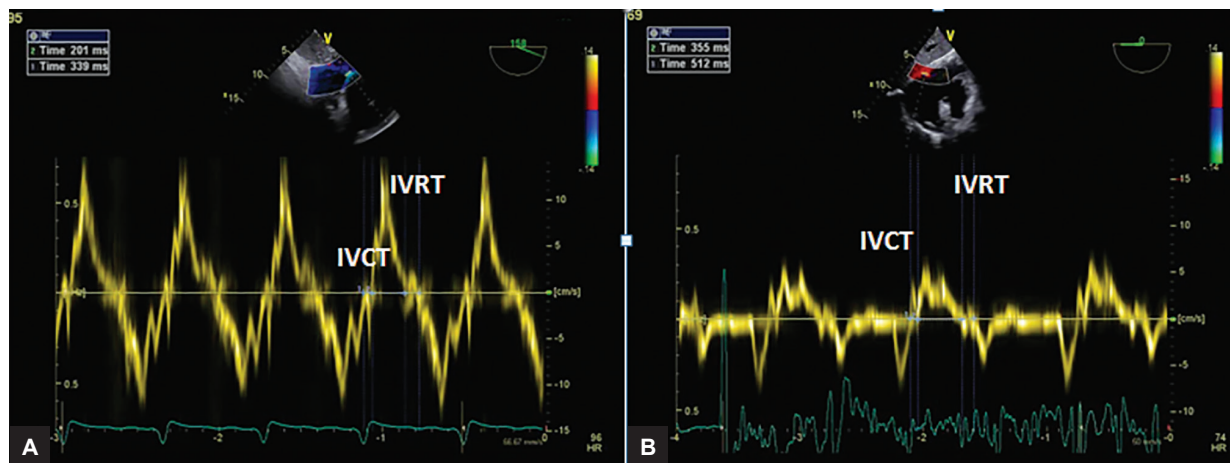
**Figs 16A and B:** The TEI index of right ventricle measured by tissue Doppler method by sampling across the tricuspid lateral annulus in TGRVIO (A) and DTG (B) views

in the evaluation of RV function. The advantage being that strain and strain rate are not angle-dependent like other Doppler measurements.[51] Studies have shown negative correlation with the strain and RV function, with value < −20 % considered significant[13] (Fig. 17).
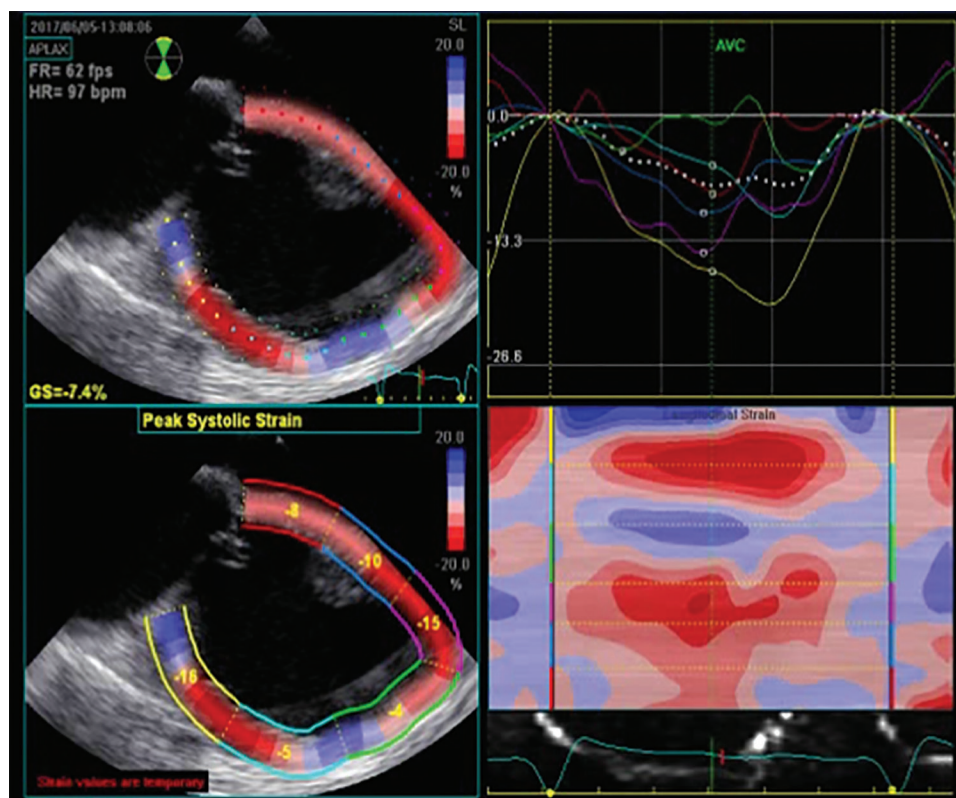
**Right Atrium**

Similar to RV, RA undergoes chronic remodeling due to PH. The thin wall of the RA makes it to dilate rather than hypertrophy with increase in volume or pressure overload

as in TR or PH.[52] Both the RA area and volume give indirect evidence of chronicity of PH. The area is measured by planimetry method and volume by area length or disk summation method in the ME4C view (Fig. 18). The normal value being RA area <18 cm[22] and RA volume 25 ± 7 mL/m$^2$ in men and 21 ± 6 mL/m$^2$ in women.[49]

**Evaluation of Consequence of PH on Left Heart**

Estimation of left heart functions helps in two ways, first in the classification of the PH as type II PH is associated



**Fig. 17:** Right ventricular strain measured in ME4C view showing reduced function by a low strain valve (−7.4%)
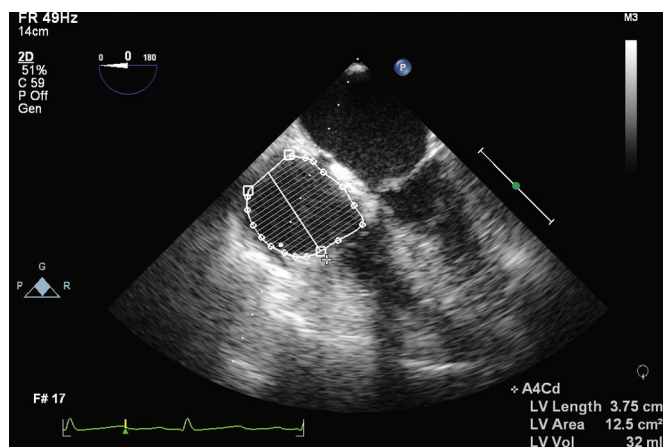
**Fig. 18:** Right atrium area and volume measured by Simpson's method in ME4C view

with left heart disease and elevated PCWP. Secondly to evaluate the effect of PH-induced RV dysfunction on LV, which helps in the management of patient.
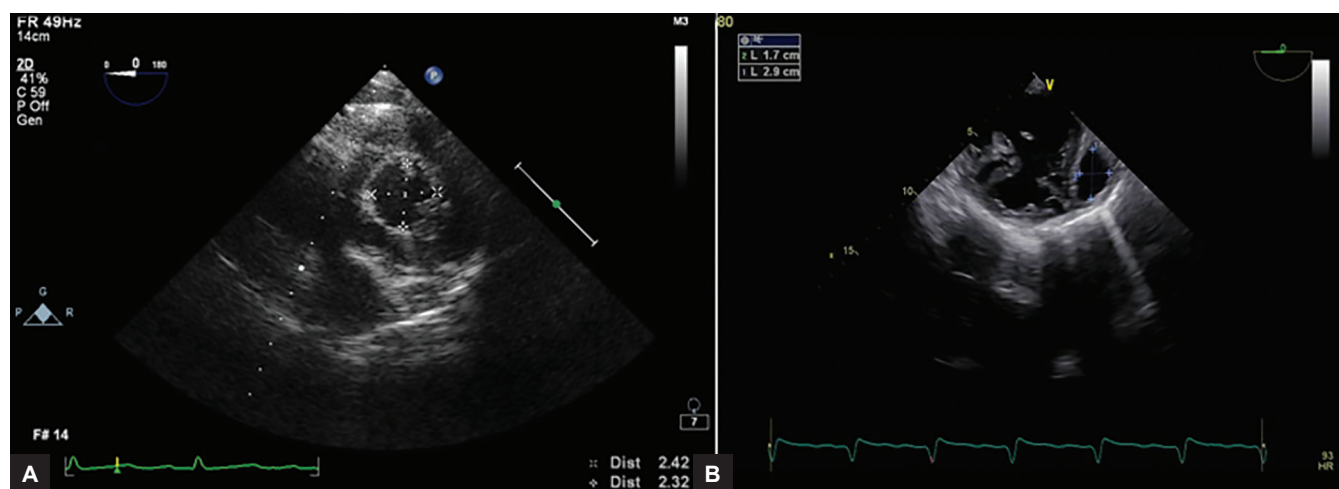
### Left Ventricle

The common pathology of mitral valve like MS/MR and depressed LV systolic and diastolic functions, which contribute to the major proportion of PH, is out of scope for this review. As discussed previously, the PCWP can be calculated using the formula PCWP = $1.9 + 1.24 \times$ E/E'. It helps in differentiating precapillary (PCWP < 15 mm Hg) from postcapillary PH (PCWP > 15 mm Hg). Studies have shown E/E' < 10 indicates pure precapillary PH.[53]

The increased LV cavity pressure compared with RV makes the IVS pushed toward the RV in normal population. As the intracavity pressure increases in RV, it pushes the IVS toward LV depending on the period of cardiac cycle in which the pressure changes, as in case of RV volume overload, it pushes IVS toward LV during mid- and late diastole, whereas in case of

pressure overload in RV as in PH, IVS gets shifted to LV during systole. This phenomenon forms the basis for the eccentricity index calculated in PH patients. It is measured by the formula LV AP diameter/LV septal lateral diameter measured in TG midpapillary SAX at the end diastole. Normally, it will be ≤1. And a value of >1 indicates RV volume overload. Same index can be measured in end systole in which case it indicates RV pressure overload (Fig. 19).

### CONCLUSION

The TEE forms a valuable tool in the intraoperative period in evaluation of both morphological and functional assessments of various cardiac structures. Even though there are few established protocols for the evaluation of PH patients by TTE, there is no such protocol for TEE evaluation in the intraoperative period, which made us to write this algorithm for diagnosis and classification of PH and its consequences on both left and right heart structures. This method is not completely foolproof, suggesting whenever there occur some controversies regarding the diagnosis of PH, RHC should be done in the intraoperative period. The TEE not only helps in the diagnosis and classification, but also in the immediate patient's response in terms of pulmonary pressure in the postsurgical correction of the causative factors of PH like MS/MR/VSD/ASD/PDA closure. A suggested algorithm for the approach of a patient suspected of PH in the intraoperative period is given below. With the development in 3D and strain technology, further studies are needed for the identification and classification of type of PH using TEE to reduce the invasive procedures associated in this patients and for better evaluation of immediate effect of surgical correction on pulmonary pressures in these patients.



**Figs 19A and B:** Left ventricular eccentricity index measured in TG midpapillary short-axis view at end systole (=1) (A) showing absence of right ventricular pressure overload and at end diastole (=1.7) (B) showing right ventricular volume overload

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