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Review Article

Echocardiographic Evaluation of Pulmonary Haemodynamics - A Review

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Key words:
Pulmonary Haemodynamics, Echocardiography

Abstract:

Pulmonary haemodynamic status has got significant clinical and prognostic significance both in structural and nonstructural heart diseases. But diagnostic accuracy depends on much care in evaluation and logical correlation of different echocardiographic parameters. Though the Doppler studies play crucial role in estimating Pulmonary haemodynamics, careful observation of 2-D measurements of different structures as well as RV systolic and diastolic function are vital issues to be addressed and correlated for diagnostic accuracy. Tricuspid Valve, RVOT and Pulmonary valve pathologies also demand vivid correlation while estimating PA pressures and pulmonary vascular resistance. Mere presence of elevated pulmonary artery systolic pressure is not enough to define pulmonary hypertension (PH). Before declaring a patient having PH a comprehensive evaluation of right heart haemodynamics is to be performed carefully to avoid unnecessary cardiac catheterization or to avoid inadvertent initiation of PH therapy.

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Introduction:

Right heart is affected frequently by disease of its own, by disease of left heart or by pathologies simultaneously involving both sides of heart. Resulting haemodynamic consequences largely affect clinical scenario and appropriate intervention at right time likely to reduce morbidity and to improve prognosis in a particular case. Considering the common trend of inadequate and inappropriate evaluation, this review article is intended to express a clarification of the different parameters to reach a relatively accurate estimation of haemodynamics of right heart system. Cardiac catheterization is the gold standard for assessing pulmonary haemodynamics and Pulmonary hypertension (PH) which is defined as sustained resting mean pulmonary artery pressure (mPAP) to e" 25 mm Hg determined by right heart catheterization (RHC).[1] In 2019, the sixth world symposium on pulmonary hypertension updated this definition to mPAP > 20 mm Hg.[2] Echocardiography is a reliable and often first tool to suspect evidence of showing an elevated right ventricular systolic pressure (RVSP) or Pulmonary arterial systolic pressure (PASP).[3] An RVSP or PASP e" 40 mm Hg is considered elevated.[4-10] PH (resting mPAP > 20 mm Hg) due to pre-capillary causes shows elevated pulmonary vascular resistance (PVR) to e" 3 Wood units along with pulmonary artery wedge pressure(PAWP) of d" 15 mm Hg and a PH with PVR< 3 Wood units along with PAWP > 15 mm Hg indicates post capillary PH and combined pre & post capillary PH is characterized by PVR e" 3 Wood units along with PAWPe" 15 mmHg.[2] Table I shows updated criteria of different PH groups.[2] Right ventricular (RV) systolic and diastolic dysfunction puts significant impact on pulmonary haemodynamics and thereby are bad prognostic factors for the disease concerned.[1, 2, 11, 12] As we are lacking in having specialized PH clinics to do random cardiac catheterization in patients with PH and as cardiac catheterization is also not always feasible for financial constrain, considering huge advancement of modern technology, dedicated

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echocardiographic evaluation of all the possible parameters would be able to detect pulmonary haemodynamics close to invasive data.

Evaluation Essentials for Pulmonary Haemodynamics:

Evaluation should include all the followings-

1. 2-D measurements of different Chambers and RV free wall thickness
2. Right atrial (RA) pressure estimation
3. Right ventricular systolic pressure (RVSP) estimation
4. Pulmonary artery (PA) Pressure includes
   a) Pulmonary artery systolic pressure (PASP)
   b) Pulmonary artery diastolic pressure (PADP)
   c) Mean pulmonary artery pressure (mPAP)
5. Pulmonary Vascular resistance (PVR)
6. Pulmonary artery compliance (PAC)
7. RV systolic function
8. RV diastolic function
9. PA pressure measurement during exercise

Table I and Table II are prepared from literature review to summarize all the essential parameters that should be evaluated for assessment of pulmonary haemodynamics.[1,2,11,12]

Table-I

Updated criteria of different groups of PH

Definitions	mPAP	PVR	PAWP
Pre-capillary PH	>20 mm Hg	> 3 Wood units	< 15 mm Hg
Isolated Post-capillary PH	>20 mm Hg	< 3 Wood units	>15 mm Hg
Combined pre and postcapillary PH	>20 mm Hg	≥3 Wood units	>15 mm Hg

Table-II

2-D and M-mode measurement related to pulmonary haemodynamics in adults (summarized from literatures).

Measurement	Normal Value	Measurement	Normal Value
RV free wall thickness	<5 mm	RA area	< 18cm^2
Basal RV dimension	<42 mm	RA long axis (major)	Men: 21-27 (mm/ m^2)
(LV dimension at same level)	(LV larger)	dimension	Women:22-28 (mm/ m^2)
Mid RV dimension	<35 mm	RA horizontal	Men: 16-22 (mm/ m^2)
(LV dimension at same level)	(LV larger)	(minor)dimension RA volume	women: 16-22 (mm/ m^2) Men: 25 ± 7 (ml/ m$^{2)}$Women: 21 (ml/m$^{2)}$
RV long axis dimension	<86 mm	IVC diameterend expiration	<21 mm
RVOT dimension (parasternal long axis)	20-30 mm	IVC diameter end inspiration (Collapsibility %)	collapsibility >50%
RVOT-proximal dimension (short axis view)	21-35 mm	MPA size	< 25 mm
RVOT-distal dimension (short axis view)	17-27 mm		
LV eccentricity index	≤1		

Table-III

Measurements for RV function and pulmonary haemodynamics (summarized from literatures).

RV systolic function assessment	Normal Value	RV diastolic function assessment	Normal value	Pulmonary Haemodynamics	Normal value
FAC	≥35%	E/A	0.8-2.0	RA Pressure	0-5 mm Hg
RVEF (2D)	≥44%	E/e'	≤6.0	PASP	<36mm Hg
RVEF (3D)	≥45%			PADP	
TAPSE	≥17 mm			PAcT	
RV global longitudinal strain	≥20%			m PAP	< 20 mm Hg
S'	≥12 cm/S				
RIMP(PW)	≤0.43			PVR	< 3 Wood units
RIMP(TD)	≤0.54			PASP on exercise	<45 mmHg or<20 of resting PASP
dp/dt	≥400mm Hg/sec				
RVSP	<36 mm Hg				

2D measurements:

Although size of different chambers does not indicate any direct evidence of a particular haemodynamic status of right heart, it has got much indirect significance on different issues like RV volume overload or chamber enlargement secondary to PH or RV dysfunction. Significant PH or RV dysfunction is less likely in a normal sized RV. RA size increases with increasing severity of PH, RV dysfunction both systolic and diastolic or with increasing severity of Tricuspid regurgitation (TR).[9, 10] PH is less likely if PA size is <25 mm. Diameter of Inferior vena cava (IVC) and its collapsibility on inspiration is a requirement for estimation of RA pressure. RV hypertrophy (free wall thickness ≥5 mm) indicate RV pressure overload. Presence of Pericardial effusion in a patient with PH signifies obstructed lymphatic drainage secondary to high RV pressure and a bad prognostic factor.[3,11] 2-D measurements with its normal values[11,12] are shown in Table II and Figure -I, II, III and IV a-c shows techniques of measurement.

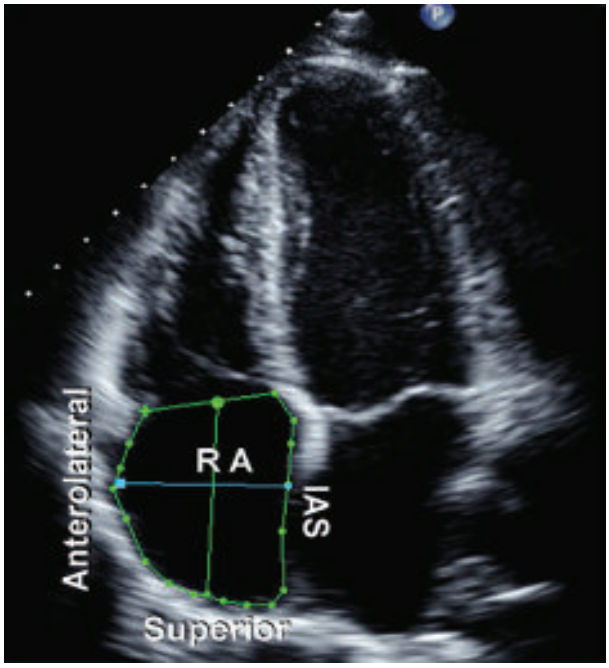

Fig.-1: *Tracing of RA. Green line indicates RA major dimension and Blue line indicates RA minor dimension.*

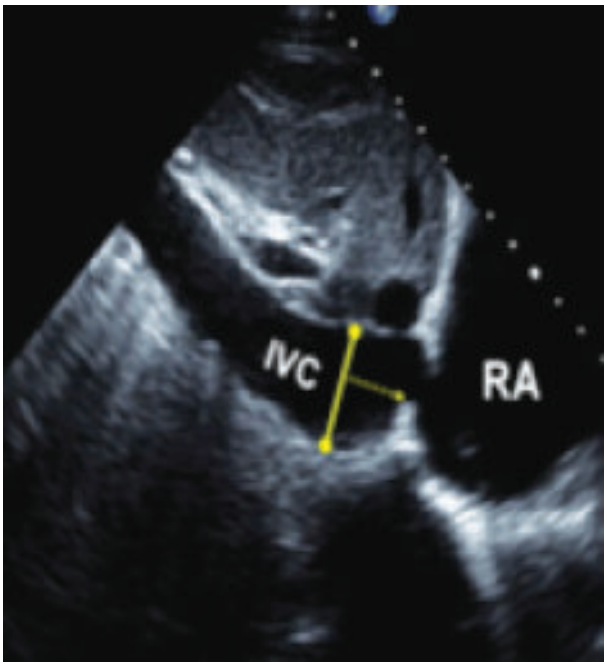

Fig.-2: *Measurement of IVC at end of expiration just proximal to opening of hepatic veins.*

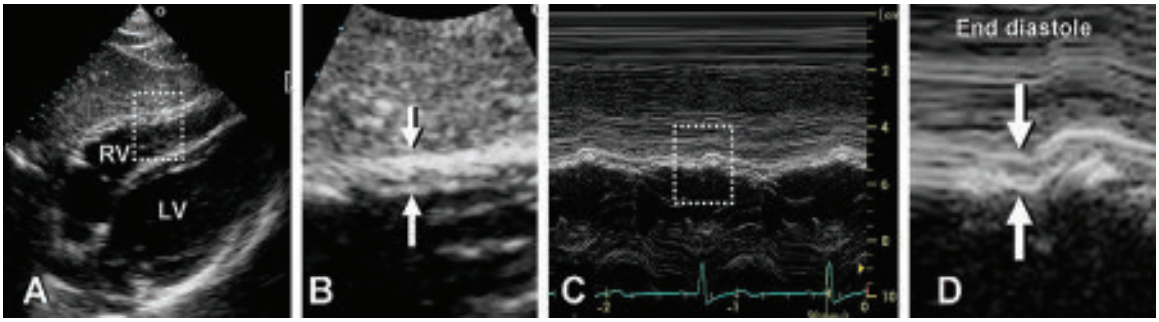

Fig.-3: *Measurement of end-diastolic RV wall thickness. A –Subcostal view, B-Zoom of region outlined in A indicating RV wall thickness. C- M-ode image corresponding to arrows in B, D-Zoom of region outlined in V with arrows indicating wall thickness at end-diastole.*

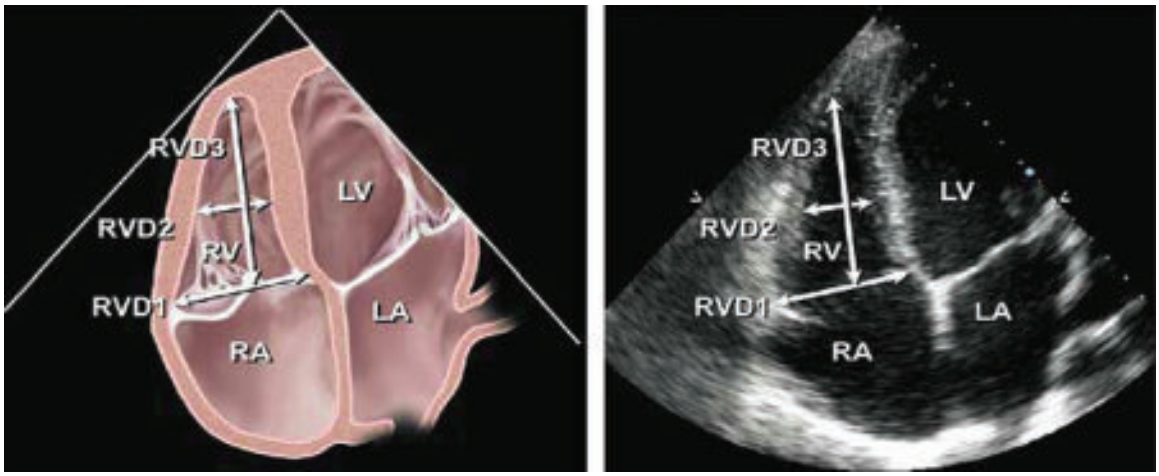

Fig.-4a: *Diagram (left) and corresponding apical 4-chamber RV focused image (right) showing RV basal (RVD1) and mid cavity (RVD2) minor dimensions and the RV longitudinal dimension (RVD3).*

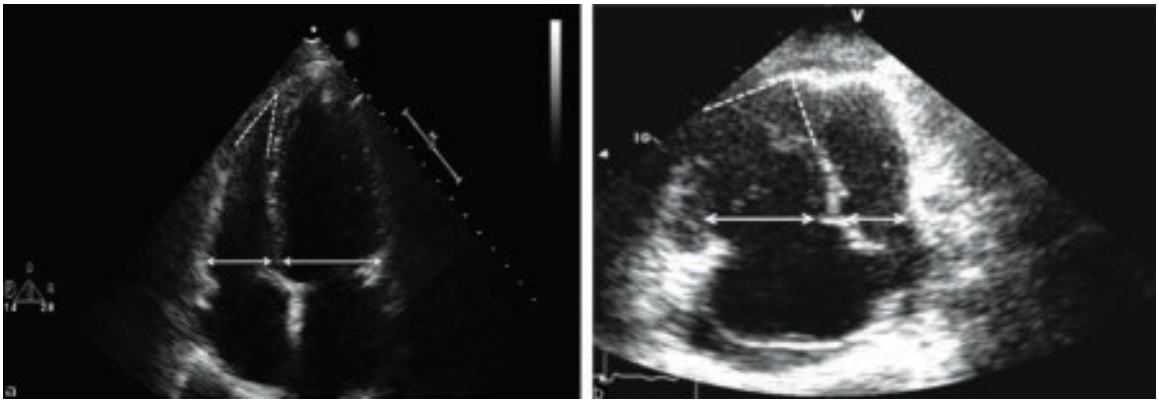

Fig.-4b: *Upper panel shows Apical four chamber view. Panel A demonstrates normal RV: LV size ratio (<1.0) and shape, with a preserved acute angle of the RV apex in a patient either without PH or with pulmonary venous hypertension. Panel B represents a patient with PAH or another form of PH with pulmonary vascular disease. Note the RV: LV ratio is increased (>1.0), the angle of the apex is less acute, and the RV is apex-sharing with the LV. Lower panel shows eccentricity index in short axis (> 1.0).*

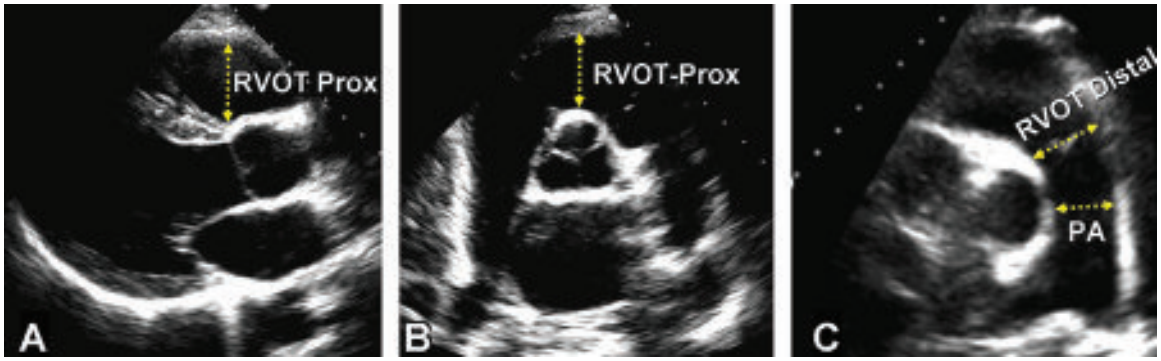

Fig.-4c: *Measurement of RVOT dimension at the proximal or subvalvular level (RVOT-Prox) and at distal or pulmonic valve level (RVOT-distal) in the (A) parasternal long-axis RVOT anterior portion view, (B) basal parasternal short-axis view and (C) parasternal short-axis of pulmonary. Bifurcation view. PA indicates Pulmonary artery dimension between valve and bifurcation.*

2. RA Pressure:

A variety of techniques have been used to estimate RAP, most often using the inferior cava (IVC) dimensions and degree of IVC collapsibility with inspiration or sniff."[14, 15]None of these techniques have proved particularly accurate, with RA pressure overestimation being the more frequently observed limitation. In fact, an overestimated RAP was the primary source of error in nearly 50% of the subjects with an overestimated PASP.[13]

RA pressure can be measured in several ways-[1, 11, 12]

a) Clinically from JVP

b) Size and collapsibility of IVC on inspiration. RA pressure is assumed 3 mm Hg (1-5 mm Hg) when IVC size is normal (<21 mm) with >50% collapsibility, RA pressure is assumed 8 mm Hg (5-10 mm Hg) when IVC is <21 mm but with <50% collapsibility or > 21 mm but >50% collapsibility and RA pressure is considered 15 mm Hg (10-20 mm Hg) when IVC is >21 mm and with <50% collapsibility.

c) Utsunomiya et al. compared right atrial pressure estimates obtained via the ratio of tricuspid inflow E wave velocity to the tricuspid annular tissue Doppler E wave velocity (E/Ea) with near simultaneous invasive pressure values in 50 patients with chronic PAH. [16]

3. RVSP Estimation

RVSP= $4V^2$ + RA pressure; Using the modified Bernoulli equation where "V" is peak TR velocity across tricuspid valve. RVSP is the key parameter for estimating PASP, though absence of TR precludes its estimation by above equation. The peak velocity of the envelope (TR Vmax) is then measured (Fig 5a). A value of ≤2.8m/s suggests low probability, a value of 2.9–3.4 m/s indicates intermediate probability, and a value >3.4 m/s suggests a high probability for PH. Accurate estimations of RVSP demands several criteria to be fulfilled[1,11,12,17,18]

i) A good quality image in parasternal long axis (RV inflow view), apical 4 chamber view, apical 4 chamber RV focused view, short axis view or sometimes in subcostal long or short axis can be used but with a good coaxial TR jet with clear peak having parallel alignment to CW signal.

ii) Agitated saline can be used to have a clear signal in patients with poor quality TR jet.

iii) To have an average of velocity peaks of several beats in case of AF

iv) CW to be used at a sweep speed of 100 mm/s to have good Doppler envelope. Frame rate to be optimized to e"20 HZ/sec, faster the heart rate higher should be the frame rate.

Limitations for using TR Velocity for RVSP (Fig 5b):

-May not be accurate in RV systolic dysfunction and TR quality may not be optimum for evaluation in Lung diseases

-Insignificant or severe TR may lead to inappropriately low RVSP.[1, 11, 17, 18]

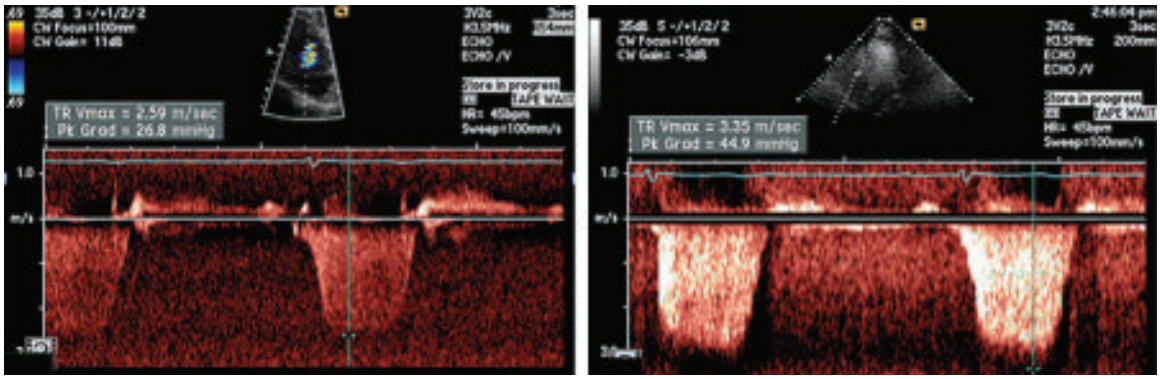

Fig.-5a: *left- Tricuspid regurgitation signal that is not contrast enhanced and correctly measured at the peak velocity. Right- After contrast enhancement, the clear envelope has been obscured by noise, and the reader erroneously estimated a gradient several points higher. As this example shows, it is critical that only well-defined borders be used for velocity measurement, as slight errors are magnified by the second-order relationship between velocity and derived pressure.*

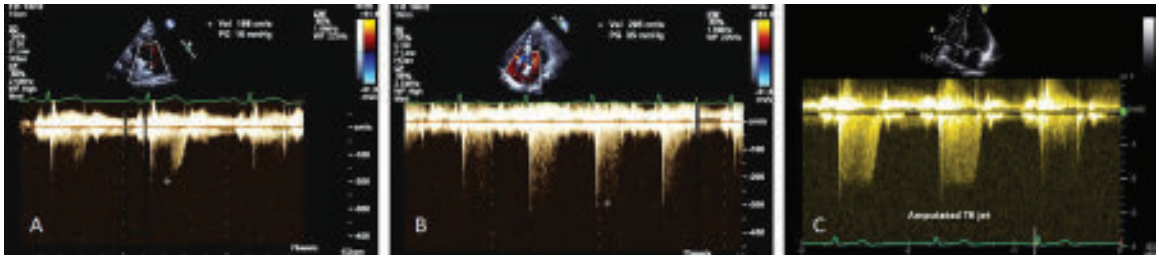

Fig.-5b: *Pitfalls in TR peak measurement. A, B—Peak TR measurement with incomplete trace could lead to underestimation. C—Amputated jet could occur in severe TR that could lead to underestimation.*

4. Estimation of PA pressures

PASP: In absence of Pulmonary stenosis (PS) or RV outflow tract (RVOT) obstruction RVSP is considered as PASP. However, there is a suggestion for just using the Doppler gradient of TR Vmax without additional RA Pressure for determination of probability of PH, as IVC assessment is error prone.[1] RVSP remains stable in both men and women until the age of 50 years. Thereafter, RVSP increases progressively in a linear manner with age and is significantly higher in patients older than 75 years of age. These ranges should be taken into account when using echocardiogram-derived RVSP for the diagnosis of PH in the absence of cardiovascular disease.[19]In presence of PS or RVOT obstruction, PS gradient or RVOT gradient must be deducted from RVSP to get the actual PA pressure.[20]

PADP: PADP can be estimated from the velocity of the end-diastolic pulmonary regurgitant jet using the modified Bernoulli equation: [PADP = 4 × (end-diastolic pulmonary regurgitant velocity) ^{2} + RA pressure].[11, 12] PR is present in most patients with PH, although the converse is not true.[21] Figure VI shows PADP measurement technique.

Limitations:

In severe pulmonary regurgitation, due to a rapid deceleration slope, PR-end velocity may underestimate PADP.[21] This technique may not be useful in the presence constrictive or restrictive physiology.[22, 23]

Mean PA pressure (mPAP): Accurate MPAP estimation needs a comprehensive evaluation and correlation of some Doppler parameters as well as consideration of RV systolic and diastolic function. MPAP can be assessed in following ways-[11, 12]

I) Assessment by visual impression of Doppler envelope:

There are 4 different patterns of RVOT Doppler envelope which are characteristic and signifies increasing severity of PH. Fig 6 shows Pattern I

characterized by a parabolic contour of the ejection flow velocity envelope ; pattern II - a triangular contour as a result of an early systolic peak followed by a slow deceleration time; pattern III and IV - similar to pattern II with the exception that pattern III has mid-systolic notching and pattern IV has significant reduction in signal volume resulting in a spiked appearance. Identification of these RVOT spectral patterns appears to be dependent only on the severity of PH. This particular Doppler marker can certainly aid in the assessment of patients in whom measurement of the maximal tricuspid regurgitation velocity is limited by either a poor transthoracic window or interrogation of eccentric jets.[24]

II) mPAP Measurement by PASP and PADP

Once PA systolic and diastolic pressures are known, mean pressure may be estimated by the standard formula mean PA pressure = 1/3(SPAP) + 2/3(PADP).[11, 12]

III) mPAP by RVOT Doppler acceleration time (RVOT AcT)

Mean PA pressure = 79 × (0.45 × RVOT AcT), When AcT ≥120 ms[25]

Mean PA pressure = 90 × (0.62 ×RVOT AcT), when AcT <120 ms[26]

The absence of TR is not sufficient to exclude significant PH, even though it typically denotes a more compensated right ventricle. RVOT Doppler AcT method is relatively easy to perform, highly reproducible, and unlike pressure estimates based on tricuspid regurgitation velocity, from the RVOT are available in virtually all patients.[11, 12]

Generally, the shorter the AcT the higher the PVR and hence the PA pressure, provided the heart rate is in the normal range of 60 to <100 beats/ mins. A pulse wave signal of pulmonic forward flow is obtained at end expiration, just proximal to the pulmonary valve in the parasternal short axis view for assessment. Right ventricular outflow tract (RVOT) acceleration time is measured from the beginning of the flow to the peak flow velocity (Fig: 7). A value of e"130ms is normal, while <100 msec is highly suggestive of PH.[14] Heart rates outside of the normal range (60 to 100 bpm) may reduce the accuracy of this technique. However, when the mean PAP exceeds 25 mmHg, RVOT acceleration time is accurate even in tachycardia.[15, 16]

An unusually shortened AcT (<60 msec) in association with PASP < 60 mm Hg (60/60 sign) is characteristically found in acute pulmonary embolism due to acute increase in pulmonary resistance and resulting RV systolic dysfunction as McConnell's sign," which is visually appreciated as RV dysfunction along the RV base and mid segments, with a hinge point or "buckling" of the RV free wall near the RV apex.[32, 33]

mPAP measurement by Peak PR gradient

The mean PA pressure can also be estimated as 4× (early peak PR velocity)^2+ estimated RA pressure. [27] (Fig 8)

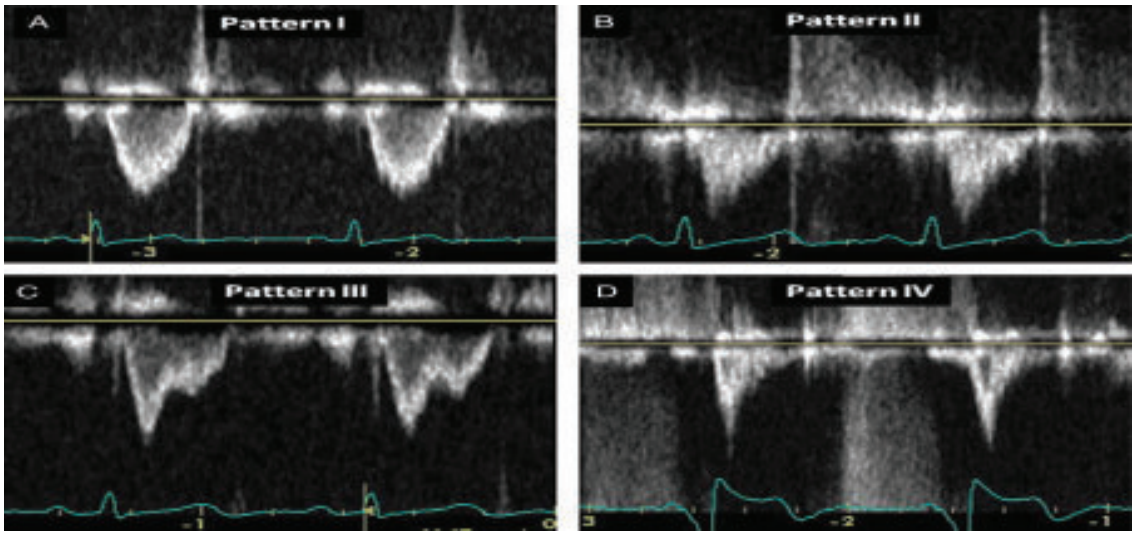

Fig 6: *Right ventricular outflow tract Doppler spectral signals showed four dynamic patterns. (A) Pattern I was characterized by a parabola-like contour of the ejection flow velocity envelope. (B) Pattern II had a triangular contour as a result of an early systolic peak followed by a slow deceleration time. (C) Pattern III has mid-systolic notching and (D) pattern IV has significant reduction in signal volume resulting in a spiked appearance.*

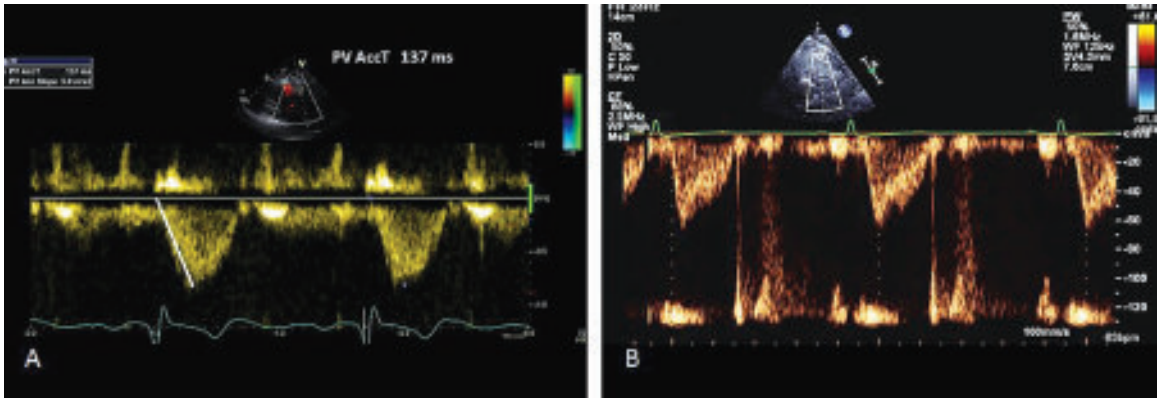

Fig 7: RVOT acceleration time method for assessing pulmonary pressure. A- Pulmonary acceleration time measurement. B- Rapid rise and mid-systolic notching suggesting elevated pulmonary pressure.

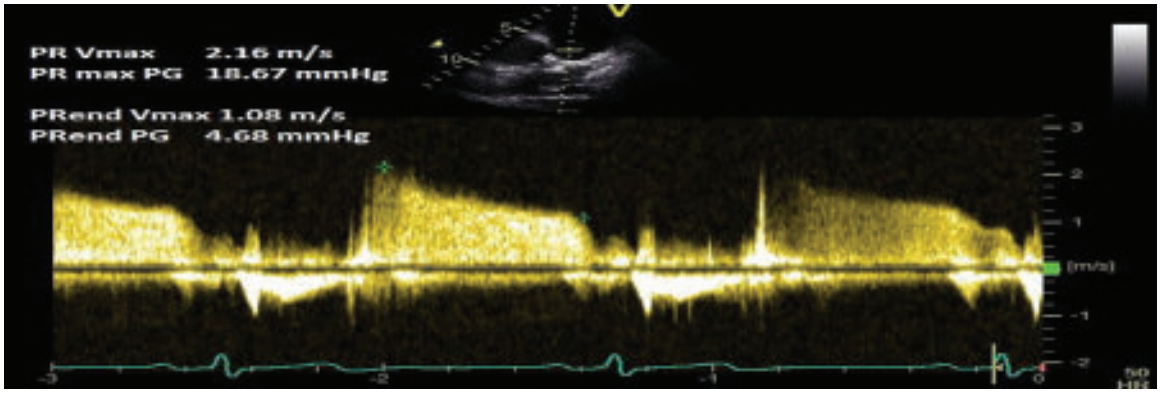

Fig.-8: *Pulmonary regurgitation method for measuring mean and diastolic pulmonary artery pressure. Mean PA pressure correlates with 4× (early PI velocity)* ^{2} *+ estimated RAP, in this example 19 mm Hg + RAP. Point 2 marks the PI velocity at end-diastole. PADP is correlated with 4 × (end PI velocity)* ^{2} *+ estimated RAP. In this example, PADP is 5 mm Hg + RAP.*

V) mPAP measurement by TR Velocity time integral (TR-VTI)

MPAP=Estimated RA pressure plus TR-VTI

This method is relatively recent one and describes addition of estimated RA pressure to the velocity-time integral of the TR jet to calculate mPAP. This method has been validated by right heart catheterization and provides a value closer to one derived hemodynamically than the empirical methods.[28, 29]

VI) Others:

RV free wall strain Sm (S'), SmVTI

Tissue Doppler imaging (TDI) is used on the RV free wall in the apical 4-chamber view, and tricuspid annular systolic myocardial velocity (Sm/S') is recorded (Fig 9 B). Sm (S') velocity < 12 cm/s and SmVTI < 2.5 are highly suggestive of elevated PASP .[30] This method correlates well with TR measured PASP but is yet to be fully validated against the gold standard-invasive right heart catheterization.[30, 31] Although the technique helps to identify patients with pulmonary hypertension, it cannot accurately quantify. [11, 12]

Right ventricular isovolumic relaxation time (rIVRT)

TDI is deployed at the lateral tricuspid annulus with a sweep speed of 100 mm/s. Pulse wave (PW) Doppler with a 6 mm sample window is obtained. Right ventricular isovolumic relaxation time (rIVRT) is measured from the offset of the S2 wave to the onset of the E' wave (Fig 9B and C). rIVRT of ≥75 msec reliably predicts pulmonary hypertension while an rIVRT of <40 msec has a high negative predictive value for pulmonary hypertension. The technique may become unreliable in hypertrophic cardiomyopathy, right bundle branch block and RV

dysfunction because the rIVRT is prolonged for other reasons. On the other hand, rIVRT is pseudo-normalized in the presence of elevated RAP and significant TR .[34, 35, 36]

Tei index and TR measured PASP

Tei index was introduced in 1990s as a Doppler-derived marker of ventricular function. Vonk et al. showed that combining TR-measured PASP ≥35 mmHg with Tei index ≥36 improves echocardiographic sensitivity for the diagnosis of PH. Tei index is proven to prognosticate patients with pulmonary hypertension. Whenever possible, it is helpful to use several methods to assess mean pressure so that the internal consistency of the data can be challenged and confirmed. [11,12,37-39] Further discussion on Tie index follows below with the discussion on RV systolic function.

Estimation of PVR

Cardiac Catheterization is gold standard for estimation of PVR (PVR-Cath). But it may not be done in each and every patient particularly when repeat catheterization is needed. So, echocardiographic measurement of PVR (PVR-echo) is invaluable in assessing patients and guiding therapy in patients with altered pulmonary haemodynamics. Several simple formulas are available in literature which correlates well with invasively measured PVR. PVR estimation is shown in Fig-10. Several formulas (Model) exist for calculation of PVR. (a)PVR =10× TR velocity (m2) / RVOT VTI + 0.16 (Model 1) (b) PVR= 1.2× PASP/ RVOT VTI (Model 2) (c) PVR= PASP/RVOT VTI + 3 (if notched) ;(model 3) (d) PVR= 5.19×TRV2/ TVIRVOT - 0.4 (Model 4). Model 1, 2 and 4 appears to have good correlation with PVR -Cath particularly

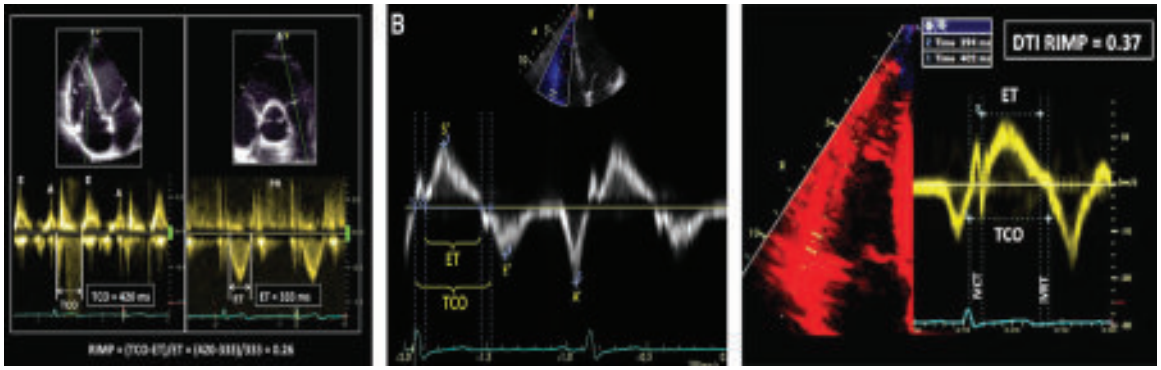

Fig.-9: *Calculation of right ventricular myocardial performance index (MPI) by pulsed Doppler (A) and pulsed tissue Doppler (B and C). The tricuspid (valve) closure opening time (TCO) encompasses isovolumic contraction time, ejection time (ET), and isovolumic relaxation time. In the pulsed Doppler method, TCO can also be measured by the duration of the tricuspid regurgitation continuous-wave Doppler signal. MPI = (TCO - ET) / ET. Note that S', E', and A' are also measured from the same pulsed Doppler tissue image.*

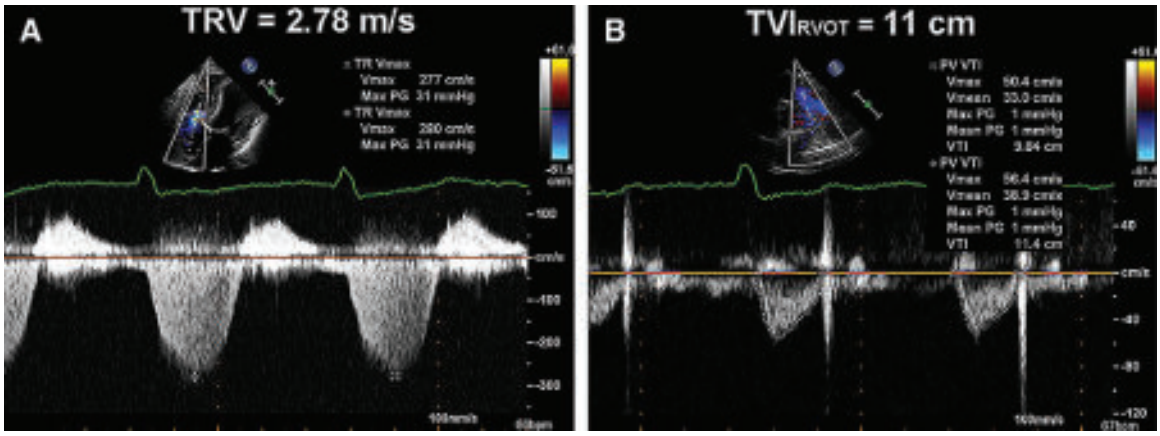

Fig.-10: *Estimation of PVR. The ratio of peak tricuspid regurgitant velocity (TRV) (2.78 m / s) to the time-velocity integral (TVI) (11 cm) in the right ventricular outflow tract (RVOT) is abnormal at 0.25 (normal, #0.15). The estimated PVR is 2.68 using the formula (TRVmax / RVOT TVI) ×10 + 0.16.*

when PVR < 8 Wood Units; And model 3 appears to have better Correlation with PVR (Cath) than model 1 and 2 at any PVR level. [11, 12, 40, 41]

Pulmonary artery compliance (PAC):

PAC, invasively assessed by the ratio of stroke volume to PA pulse pressure, is a sensitive marker of right ventricular (RV)-PA coupling that differs across the spectrum of pulmonary hypertension (PH) and is predictive of outcomes. A retrospective cohort study shows Echocardiographically derived ratio of RV outflow tract velocity time integral to PA systolic pressure (RVOT-VTI/PASP) (a) correlates with invasive PAC, (b) discriminates heart failure with preserved ejection fraction associated PH (HFpEF-PH) from pulmonary arterial hypertension (PAH). RVOT-VTI/PASP differed significantly across the PH spectrum (PAH: 0.13 [0.010–0.25] vs. combined post and pre-capillary PH(Cpc-PH): 0.20 [0.12–0.25] vs. Isolated postcapillary PH (pc-PH): 0.35 [0.22–0.44]; P<0.001), distinguished HFpEF-PH from PAH (AUC¼0.72, 95% CI¼0.63–0.81) and Cpc-PH from Ipc-PH (AUC¼0.78, 95% CI¼0.68–0.88), and (c) is associated with functional capacity.[42]

RV systolic function

RV size and function are important in assessing pulmonary haemodynamics and carries important prognostic value in cardiac and pulmonary diseases. Patients with RV systolic dysfunction may have low PASP due to reduced stroke volume but may have high PVR in patients with PH. Thin-walled compliant RV is for the dealing of low resistance pulmonary circulation. Conditions that acutely increase PVR, such as pulmonary embolism, result in increases in RV size prior to the augmentation of pulmonary pressures, which ultimately may result as the ventricle hypertrophies. Dilatation of the right ventricle thus is the first marker of increases in PVR followed by rise in RVSP as the RV hypertrophies to overcome the elevated PVR. Regional dyskinesia with resultant RV dysfunction in acute pulmonary embolism is entirely reversible with improvement in pulmonary hemodynamics. In patients with longstanding pulmonary vascular disease or other forms of secondary PH the right ventricle tends to hypertrophy and normalize volumes at first, followed by eventual and progressive dilatation. RV dysfunction is one of the most powerful independent predictors of outcome following myocardial infarction, even in the absence of overt RV infarction. Elevated pulmonary pressures directly may contribute to alterations in RV size and function in patients with heart failure. Substantial TR from disease of intrinsic tricuspid valve may lead to RV dilatation and dysfunction without producing significant PH. Visual assessment of RV systolic function gives the reader an initial qualitative evaluation of RV systolic function but remains insufficient in this era of standardization.[11, 43, 44] Different parameters of RV systolic and diastolic function along with other haemodynamic parameters with their normal values are summarized in Table III.

RV systolic function can be assessed by- I) RV fractional area change (FAC), II) RV ejection fraction (RV EF) -2D or 3D, III) Tricuspid annular plane systolic excursion (TAPSE), IV) RV dp/dt, V) RV index of myocardial performance (RIMP) or MPI Myocardial performance index of RV or Tie Index, VI) S', VII) RV longitudinal strain.[11, 12]

RV FAC-

RV FAC = (end-diastolic area - end-systolic area)/ end-diastolic area × 100. It has got fair correlation of RV systolic function with RV EF by magnetic resonance imaging (MRI). FAC is obtained by tracing the RV endocardium both in systole and diastole from the annulus, along the free wall to the apex, and then back to the annulus, along the interventricular septum (Fig 11). Care must be taken to trace the free wall beneath the trabeculations. An FAC of ≥35% is considered as normal value.[11,12]

RV EF-

RV EF from 2D methods is calculated as (EDV - ESV)/EDV × 100, where EDV is end- diastolic volume, ESV is end- systolic volume. The lower reference limit of pooled studies using these methods for the measurement of RV EF is e"44%. RV volume estimation by 2D echocardiography is very complex for its unique geometry. Calculation of RV volume can be divided into area-length methods, disk summation methods, and others. The area-length methods need an approximation of RV geometry, most commonly based on modified pyramidal or ellipsoidal models. It underestimates MRI-derived RV volume and is inferior in

comparison with 3D echocardiographic methods of RV volume estimation. The disk summation method has also been applied to determine a RV "body" volume, using predominantly the apical 4-chamber view. RV volumes are underestimated because of the exclusion of the RVOT and technical limitations of the echocardiographic images. Two dimensionally derived estimation of RV EF is not recommended for heterogeneity of methods and geometric assumptions. With 3D echocardiography, there is less underestimation of RV end-diastolic and end-systolic volumes and improved test-retest variability compared with 2D echocardiography. Three dimensional

echocardiography has been extensively validated against CMR, and the volumetric semi-automated border detection approach is the recommended method for the assessment of RV EF and a value of ≥45% is regarded as normal. The limitations of 3D assessment of RV EF are load dependency, interventricular changes affecting septal motion, poor windows, and irregular rhythms. [11, 12] Fig 12 shows RV systolic function assessment by EF method.

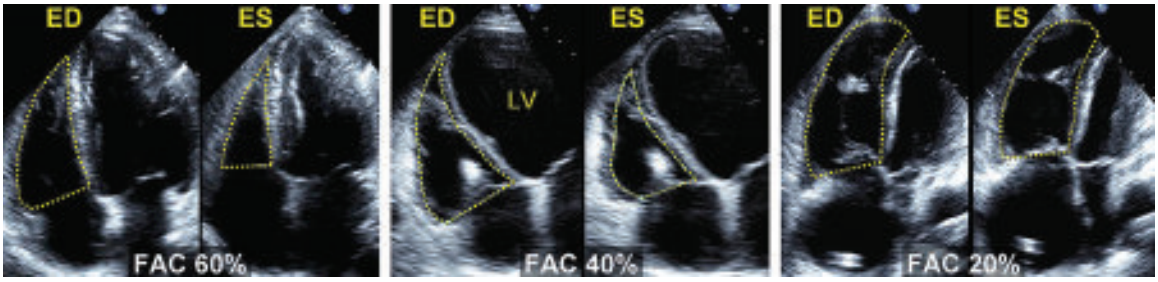

Fig.-11: *Examples of right ventricular fractional area change (FAC). Percentage FAC = 100× end-diastolic area (Area ED) _ end systolic area (Area ES)/end-diastolic area. The endocardial border is traced in apical 4-chamber (A4C) views from the tricuspid annulus along the free wall to the apex, then back to the annulus, along the interventricular septum at end-diastole (ED) and end-systole (ES). Trabeculation, tricuspid leaflets, and chords are included in the chamber. (Left) Normal subject, FAC 60%. (Middle) Moderately dilated right ventricle (RV), FAC 40%, and a markedly dilated left ventricle (LV). (Right) Dilated RV, FAC 20%.*

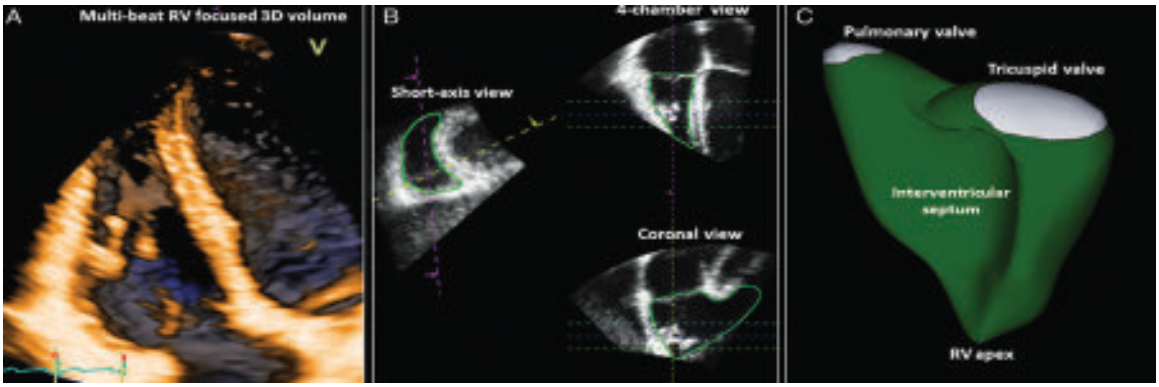

Fig.-12: *Three-dimensional analysis of the right ventricle (RV). (A)A3Ddata set is acquired from a right ventricular-focused apical four-chamber view by stitching together the sub-volumes generated from several (four to six) consecutive beats. (B) The right ventricular endocardial surface is semi-automatically identified after manual initialization in the right ventricular short-axis, four-chamber, and coronal views in both end-systole and end-diastole. (C) The generated 3D surface model of the RV enables the quantification of right ventricular ESV and ESV, stroke volume, and EF.*

TAPSE or Tricuspid Annular Motion (TAM)-
TAPSE or TAM is a method to measure the distance of systolic excursion of the RV annular segment along its longitudinal plane, from a standard apical 4-chamber window. TAPSE or TAM represents longitudinal function of the right ventricle in the same way as mitral annular plane systolic excursion by Doppler tissue imaging does with the left ventricle and is usually achieved by placing M-mode cursor through the tricuspid annulus and measuring the amount of longitudinal motion (Fig 13). TAPSE is simple, less dependent on optimal image quality, reproducible, and it does not require sophisticated equipment or prolonged image analysis. TAPSE has strong correlation with radionuclide angiography and low inter-observer variability. It has also been validated against biplane Simpson RV EF and RV fractional area shortening. It may not be an efficient tool for RV function assessment in cases of regional wall motion abnormalities. TAPSE of ≥18 is considered as normal.[11, 12]

RV dp/dt-
RV dP/dt can be accurately estimated from the ascending limb of the TR continuous-wave Doppler signal. RV dP/dt is commonly calculated by measuring the time required for TR jet to increase in velocity from 1 to 2 m/s (Fig 14). Using the simplified Bernoulli equation, this represents a 12 mm Hg increase in pressure. The dP/dt is therefore calculated as 12 mm Hg divided by this time (in seconds), yielding a value in millimeters of mercury per second. This is a simple technique with a sound physiologic basis. But there are limited data in both normal subjects and pathologic conditions and also it is load dependent. RV dp/dt will be less accurate in severe TR because of neglect of the inertial component of the Bernoulli equation and the rise in RA pressure. RV dP/dt approximately <400 mm Hg/s is likely abnormal and indicates RV systolic dysfunction.[11, 12]

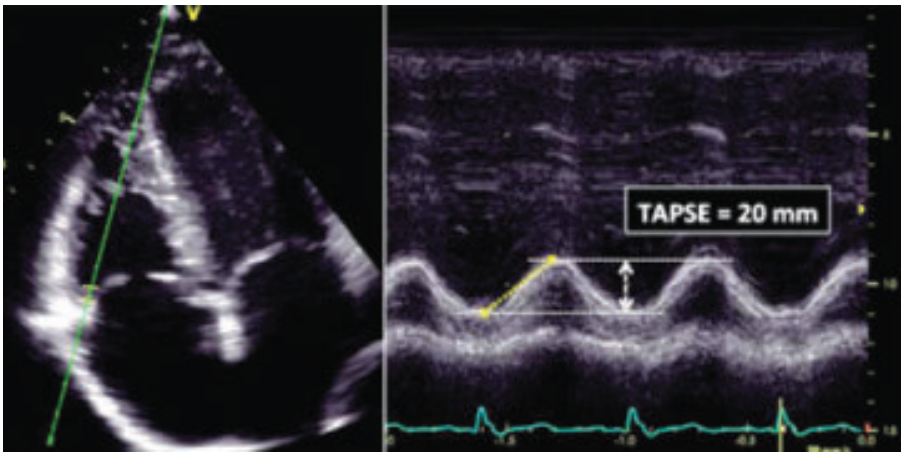

Fig.-13: *Measurement of tricuspid annular plane systolic excursion (TAPSE).*

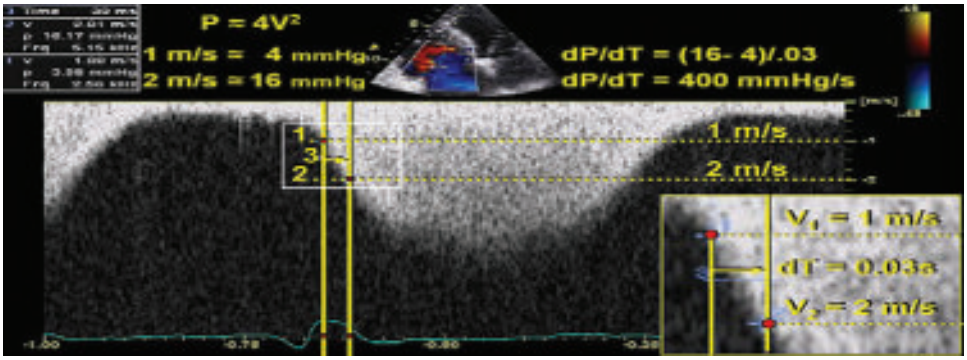

Fig.-14: *Point 1 represents the point at which the tricuspid regurgitation (TR) signal meets the 1 m/s velocity scale marker, while point 2 represents the point at which the TR signal meets the 2 m/s velocity scale marker. Point 3 represents the time required for the TR jet to increase from 1 to 2 m/s. In this example, this time is 30 msec, or 0.03 seconds. The dP/dt is therefore 12mm Hg/0.03 seconds, or 400 mm Hg/s.*

RIMP/MPI of RV/Tie index-

This is a global estimate of both systolic and diastolic function of the right ventricle. It is based on the relationship between ejection and non-ejection work of the heart. The MPI is defined as the ratio of isovolumic time divided by ET, or [(IVRT + IVCT)/ET] where IVRT is isovolumic relaxation time, IVCT is isovolumic contraction time and ET is ejection time. The MPI may be used for initial and serial measurements as an estimate of RV function in complement with other quantitative and non-quantitative measures. The upper reference limit for the MPI is 0.43 using the pulsed Doppler method and 0.54 using the pulsed tissue Doppler method. It should not be used as the sole quantitative method for evaluation of RV function and should not be used with irregular heartbeats. This approach is feasible in a large majority of subjects both with and without TR, RIMP is reproducible and avoids the assumptions and limitations of complex RV geometry. The pulsed tissue Doppler method allows for measurement of MPI as well as S', E', and A', all from a single image (Fig 9). The MPI is unreliable when RV ET and TR time are measured with differing R-R intervals, as in atrial fibrillation. Moreover, it is load dependent and unreliable when RA pressure is elevated. ET is measured with pulsed Doppler of RVOT (time from the onset to the cessation of flow), and the tricuspid valve closure-opening time is measured with either pulsed Doppler of the tricuspid inflow (time from the end of the trans-tricuspid A wave to the beginning of the trans-tricuspid E wave) or continuous Doppler of the TR jet (time from the onset to the cessation of the jet) [Fig 9]. These measurements are taken from different images, and one must therefore attempt to use beats with similar R-R intervals to obtain a more accurate RIMP value. In the tissue Doppler method, all time are measured from a single beat by pulse wave on the tricuspid annulus.[11, 12]

RV Systolic excursion velocity (S')-

Pulsed Doppler sample volume is placed either on lateral tricuspid annulus or middle of the basal segment of RV free wall with a tissue Doppler mode. 4 chamber windows are used. The velocity S' is read as the highest systolic velocity, without over gaining the Doppler envelope. Mean annular velocities in different studies average 8.5 to 10 cm/s, while basal RV free wall velocities are slightly higher at 9.3 to 11 cm/s. S' < 10 cm/s should raise the suspicion for abnormal RV function, particularly in a younger adult patient. This technique is less reproducible at non basal segments, angle dependent and may not be a standard tool of evaluation in RV dysfunctions from regional wall motion abnormalities.[11, 12]

Myocardial Acceleration during Isovolumic Contraction (IVA)-

Myocardial acceleration during isovolumic contraction is defined as the peak isovolumic myocardial velocity divided by time to peak velocity and is typically measured for the right ventricle by Doppler tissue imaging at the lateral tricuspid annulus (Fig 9). RV IVA has been demonstrated to correlate with the severity of illness in conditions affecting right heart function, including obstructive sleep apnea, mitral stenosis, repaired tetralogy of Fallot with pulmonary regurgitation, and transposition of the great arteries following an atrial switch procedure. There are limited normative data available, and though RV IVA is a relatively load independent measurement of global RV systolic function and angle-dependent Doppler measurement that appears to vary with age and heart rate. The lower reference limit by pulsed-wave Doppler tissue imaging is 2.2 m/s^2.[11, 12]

RV Strain and Strain Rate (RV longitudinal strain)-

Strain measurement has been possible using 2D images, resulting in the estimation of 2D strain. This new measure of regional and global contractility uses frame-by-frame tracking of unique speckles in the myocardium with an algorithm that allows tracking the speckle location on sequential images using correlation criteria and sum of absolute differences. Strain and strain rate are useful parameters for estimating RV global and regional systolic function. Longitudinal strain is calculated as the percentage of systolic shortening of the RV free wall from base to apex, while longitudinal strain rate is the rate of this shortening. RV longitudinal strain should be measured in the RV-focused four-chamber view. It can provide regional function estimates, as well as a more "global" function. The term RV global longitudinal strain (GLS) usually refers to either the average of the RV free wall and the septal segments or the RV free wall segments alone. global longitudinal RV free wall strain >20% (i.e., <20% in absolute value) is likely abnormal (Fig 15).[11, 12]

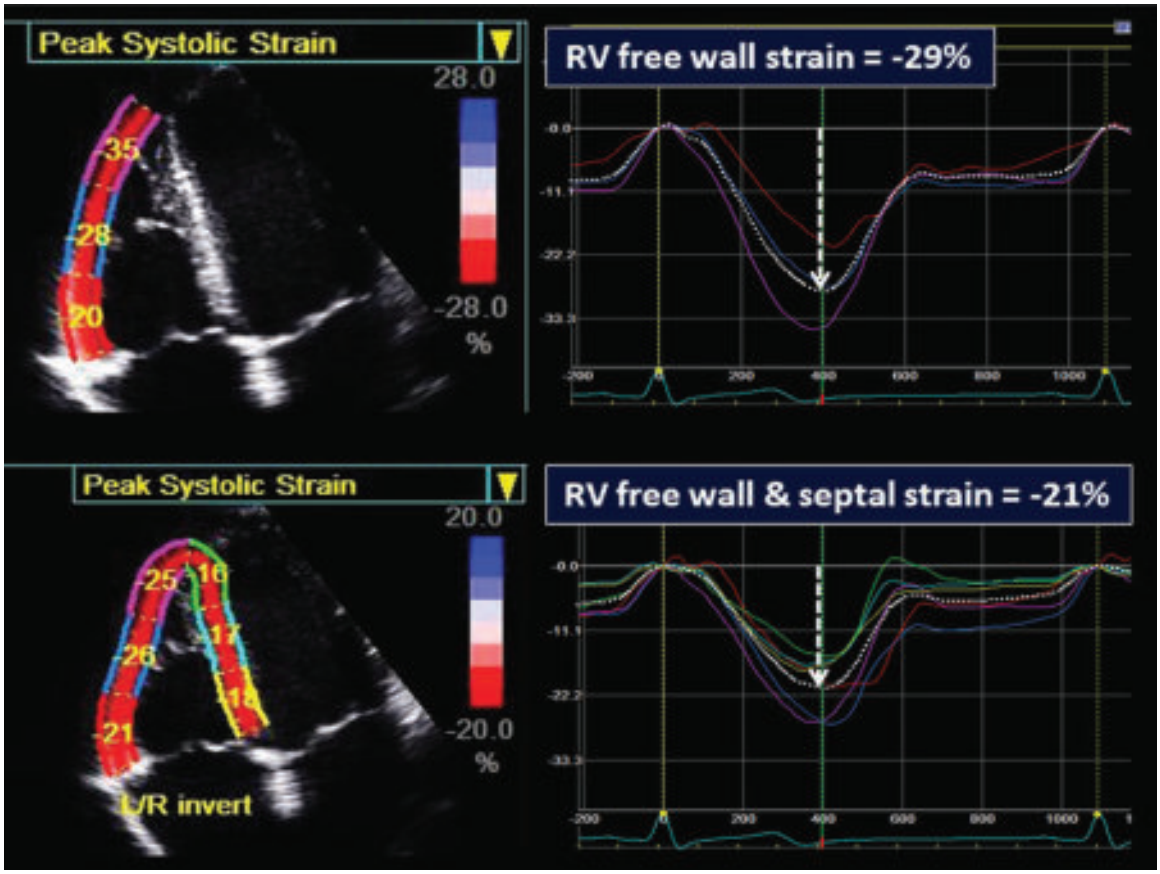

Fig.-15: *Measurement of RV systolic strain by2D STE. The upper panel demonstrates RV "global" free wall strain whereby the three segments of the free wall are averaged, and the lower panel demonstrates "global" longitudinal strain of the six segments of the apical four-chamber view: three free wall and three septal segments.*

RV diastolic dysfunction

Diastolic function of LV is seldom assessed by echocardiography though a number of acute and chronic conditions have been associated with RV diastolic dysfunction like pressure and volume overload pathologies, primary lung disease, ischemic heart disease, congenital heart disease, cardiomyopathies, LV dysfunction, systemic diseases and the physiologic aging process. The parameters used to assess RV diastolic function are essentially the same as those used to assess the left side. Measurement of RV diastolic function should be considered in patients with suspected RV impairment as a marker of early or subtle dysfunction of RV, or in patients with known RV impairment as a marker of poor prognosis. Trans-tricuspid E/A ratio, E/E' ratio, and RA size have been most validated and are the preferred measures (Fig 9). Tricuspid E/A ratio< 0.8 suggests impaired relaxation, a tricuspid E/A ratio of 0.8 to 2.1 with an E/E' ratio > 6 or diastolic flow predominance in the hepatic veins suggests pseudo-normal filling, and a tricuspid E/A ratio > 2.1 with a deceleration time < 120 msec suggests restrictive filling (as does late diastolic ante grade flow in the pulmonary artery). Further studies are warranted to validate the sensitivity and specificity and the prognostic implications of this classification. [11, 12]

PA pressure measurement during exercise

Pulmonary artery pressure rises significantly during exercise in patients with latent PH compared to healthy subjects.[45, 46]This increase occurs early during loaded exercise making it amenable to measurement. TR Vmax measured PASP of >45 mmHg or a rise of >20 mmHg during low-intensity exercise (while not exceeding a

cardiac output of 10 l/min) is diagnostic for latent PH with moderate sensitivity and specificity.[46, 47] However, this cut-off should not be applied to athletes and the elderly who may reach a PASP of 55-60 mmHg on exercise.[48] An E/E2 value of >15 during exercise predicted an elevated catheter-measured PCWP. Ha et al. also showed that exercise mitral E/E2 is a reliable measure in predicting indolent PH due to left heart disease in patients with normal resting pulmonary pressure.[49]A pulmonary hypertensive response during exercise can be clinically important in several conditions, including valvular heart disease, heart failure, and PH.[11, 12]

Conclusion:

Study of Pulmonary haemodynamics is crucial not only for diagnosis of diseases of right heart system but it has got significant diagnostic and prognostic implication of diseases of both sides of the heart and a number of systemic diseases as well. With recent advances in the field, cardiologists should be aware of complete assessment of right heart system and thereby achieving a data very close to invasive data by cardiac catheterization. In future, a scoring system combining various parameters in conjunction with exercise testing might be more helpful to assess and follow up the patients and reduce the need for invasive assessments.

Conflict of Interest - None.

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