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Assessment of Right Ventricle by Echocardiogram

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Abstract

Assessment of right ventricular (RV) function is important to ascertain clinical outcome in patients with symptoms of right ventricular failure manifested as lower extremity swelling and abdominal congestion. RV function is not routinely assessed and reported in clinical practice. Unlike the bullet-shaped left ventricle (LV), RV has a complex geometry with a triangular shape. RV is further divided into the inlet, trabecular apex, and infundibulum or conus. RV evaluation involves quantifying afterload and preload, assessing the mechanism and severity of tricuspid regurgitation (TR), and quantitative evaluation of RV performance. For quantification of RV size and function, we can use intravenous contrast for endocardial tracing of RV border to measure RV dimensions, tricuspid annular plane systolic excursion (TAPSE), fractional area change (FAC), Doppler index of myocardial performance (Tei index or myocardial performance index), pulsed wave or color Doppler tissue imaging systolic velocity [s'], or strain imaging. For qualitative evaluation of RV, the RV size is compared to the LV size in parasternal, short axis, and subcostal projections.

Keywords: right ventricle, functional evaluation, right heart hemodynamics, echocardiography, clinical significance

1. Introduction

Historically, the importance of right ventricle (RV) has been underestimated and overlooked in clinical practice and literature. Usually, left ventricle (LV) function is most commonly reported and signified. Only in recent years, the importance of assessment of RV size and function in clinical management and treatment of cardiopulmonary disorders has been recognized [1]. RV dysfunction is associated with adverse clinical outcome [2–8] in patients with LV dysfunction,

acute myocardial infarction, pulmonary embolism, pulmonary arterial hypertension, and congenital heart disease [9–11]. Hence, this has generated interest in the evaluation of RV function. RV dysfunction could be secondary to pressure or volume overload; from primary right heart disease or secondary to left heart diseases such as cardiomyopathy or valvular heart disease [12, 13] (**Tables 1** and **2**). RV dysfunction may affect by way of interventricular septal motion (ventricular interdependence) and by affecting LV preload.

RV cardiomyopathy

Arrhythmogenic right ventricular cardiomyopathy (ARVC)
 Dilated cardiomyopathy
 Endomyocardial fibrosis
 Cirrhotic cardiomyopathy
 Eosinophilic myocarditis
 Peripartum cardiomyopathy
 Uhl's anomaly
 Sepsis
 Viral myocarditis
 Coronary artery disease

Table 1. Causes of RV contractile dysfunction.

RV pressure overload

All groups of pulmonary hypertension
 Massive pulmonary embolism
 ARDS
 Eisenmenger syndrome
 RV outflow obstruction
 Pulmonic valve stenosis
 Infundibular stenosis
 (Tetralogy of Fallot, hypertrophic cardiomyopathy)
 Mechanical ventilation
 Hypoventilation

RV volume overload

Left to right shunt
 Atrial septal defect
 Anomalous pulmonary venous drainage
 Pulmonary regurgitation
 Tricuspid regurgitation
 Primary
 Infective endocarditis
 Carcinoid syndrome
 Rheumatic heart disease
 Ebstein's anomaly
 Secondary to tricuspid annular dilation from RV Dilation

Table 2. Causes of right ventricular overload.

Of all the noninvasive imaging modalities, echocardiography remains a mainstay in the evaluation of RV. Moreover, with advances in echocardiography the pathophysiology of RV has been better understood. In this chapter, we aim to review various methods to assess RV anatomy, function, and hemodynamics using two-dimensional (2D) echocardiography, color Doppler echocardiography, tissue Doppler imaging (TDI), three-dimensional (3D) echocardiography, and strain imaging echocardiography [12]. To identify RV pathology, guidelines have been published by the American Society of Echocardiography (ASE) on parameters and normal reference values (Table 5).

2. Location and anatomy of RV

The RV in the normal heart is the most anteriorly situated cardiac chamber located immediately behind the sternum and anterior to LV. It forms the majority of the anterior as well as the inferior border of the cardiac silhouette. Due to this unique anatomical location, assessment of RV size and function by transthoracic echocardiogram (TTE) may appear easy but assessment of RV function is challenging given the odd geometry of the crescent-shaped RV that wraps around conical LV. Furthermore, heavily trabeculated myocardium also limits the delineation of RV endocardial surface.

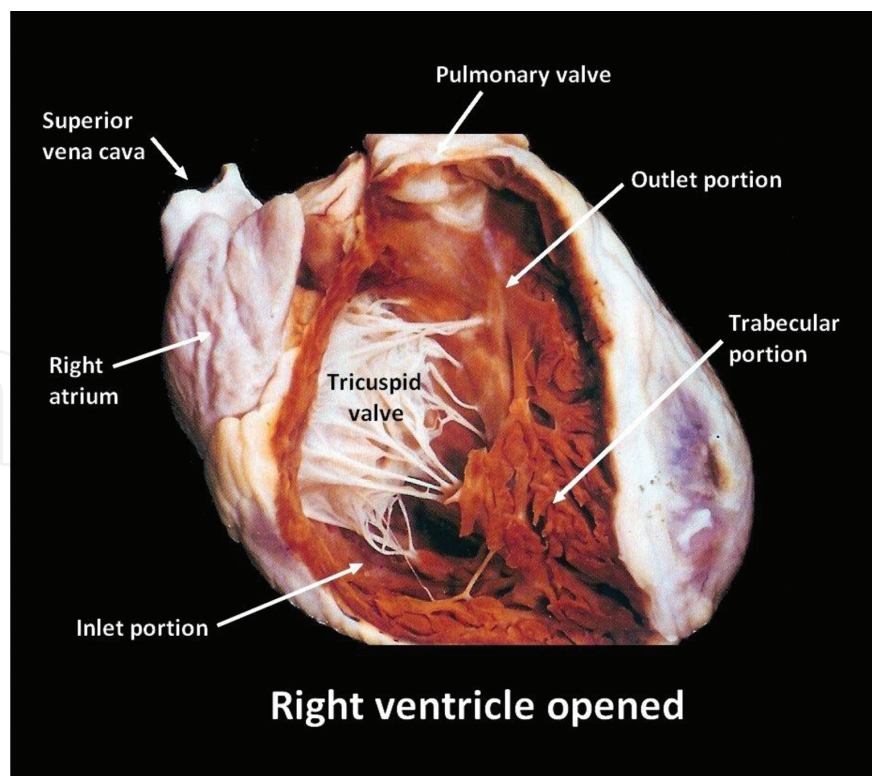


Figure 1. RV anatomy and myocardial fibers. The RV structure: illustrates the inlet, trabecular, and outlet components.

Unlike the LV that is ellipsoid or conical, the RV is crescent shaped or pyramidal, and its cavity has three components [14]: **Figure 1**

1. The muscular inlet comprising of the tricuspid valve, chordae tendineae, and three papillary muscles, which originate in ventricular wall and attach to anterior, posterior, and septal leaflets of the tricuspid valve via chordae tendineae.
2. Immobile apex with heavy, coarse trabeculations; two thick intracavitary muscle bands, the crista supraventricularis [15], and the moderator band attached to the right ventricular outflow tract (RVOT) extending from the interventricular septum (IVS) to the anterior RV wall. The apical part of the RV is heavily trabeculated and virtually an immobile part of the ventricle.
3. Smooth funnel-shaped myocardial outflow tract called infundibulum [14].

The RV is formed by free (anterior and posterior) wall and interventricular septum. Blood supply to the RV is by right coronary artery (equal in systole and diastole except in pressure overload and hypertrophy). The moderator band is supplied by the left anterior descending artery. The tricuspid valve has three papillary muscles and three cusps (anterior, posterior, and septal). The tricuspid valve is 2 mm more apical to the mitral valve. It is very important to be able to differentiate left ventricle from right ventricle based on morphology seen on an echocardiogram (**Table 3**).

Right ventricle is characterized by:

- More *apical position* of the tricuspid valve as compared to the mitral valve
 - Presence of a moderator band
 - Presence of more than three papillary muscles
 - Three leaflets of the tricuspid valve with *septal* papillary attachments
 - Presence of trabeculations (trabeculations can also be seen in the left ventricle in case of pathological noncompaction of the left ventricle)
-

Table 3. Morphological differences between the right ventricle from the left ventricle.

2.1. Musculature of ventricular wall

The RV has one-sixth the muscle mass of LV as it pumps against approximately one-sixth the resistance the LV encounters. However, the RV pumps equal cardiac output as LV. The RV ejection fraction (EF) is lower as RV end-diastolic volume is slightly larger than that of the left ventricle. Appropriately, the RV is adaptable as a volume pump but is likely to fail when subjected to acute pressure overload. The muscular wall of the RV excluding trabeculations is

3–5 mm thick [16]. RV is relatively thin walled having superficial subepicardial circumferential myofibers parallel to the atrioventricular groove that encircles the subpulmonary infundibulum and deeper subendocardial longitudinal myofibers. Unlike the relatively thick-walled LV, the RV lacks the third layer of spiral/oblique myofibers. Longitudinal fibers contract to result in inward/radial thickening. The septal motion is considered to contribute to both LV and RV function [17, 18] and is a major determinant of overall RV performance [17–19].

2.2. RV area

The RV area is measured in the apical four-chamber window at end-diastole by planimetry of the RV cavity. Delineation of the RV endocardium is challenging and should exclude trabeculations or moderator band; however, one should include the apex of the RV to avoid erroneous estimation. The normal reference limit for RV end-diastolic area is ≤ 24 cm² in men and ≤ 20 cm² in women [14].

2.3. RV wall thickness

The RV wall thickness can be measured by M-mode or 2D echocardiography from either the left parasternal window or subcostal window at the level of the tip of the anterior tricuspid leaflet. RV hypertrophy is seen in infiltrative and hypertrophic cardiomyopathy, whereas RV wall thinning is seen in Uhl anomaly and arrhythmogenic RV cardiomyopathy. When measuring the RV wall thickness, it is essential to exclude RV trabeculations, papillary muscle, thickened pericardium, and epicardial fat. The normal cutoff is 0.5 cm from either parasternal long axis (PLAX) or subcostal windows.

2.4. RV linear dimensions

RV size can be measured from the apical four-chamber view at end-diastole wherein RV should appear almost two-thirds of the size of LV on qualitative assessment. The RV is enlarged in acute pressure and volume overload. The absence of standard reference points in RV imaging serves as a limitation in using transthoracic echocardiogram. The RV may appear viable in size when RV imaging is done through various cut planes depending on the probe rotation [20]. In the four-chamber view, the focus should be on the right ventricular chamber “RV Focused view” for better imaging of the RV lateral wall and to maximize the RV size. One should adjust the transducer to attain maximal plane to avert underestimation and to avoid overestimation by positioning the transducer over the cardiac apex with the plane through the left ventricle in the center of the cavity. The basal diameter is the maximal short-axis dimension in the basal, one-third of the right ventricle. The mid-cavity diameter is measured at the level of the LV papillary muscles in mid-third of the RV, and the longitudinal dimension is measured from the plane of the tricuspid annulus to the RV apex. ASE guidelines for the right heart assessment recommend measurement of the following dimensions: RV basal-apical four-chamber view, RV wall thickness (subcostal long axis view), proximal RVOT PSAX (parasternal short axis) view at the great vessels level, and distal RVOT PLAX view (**Figure 2**).

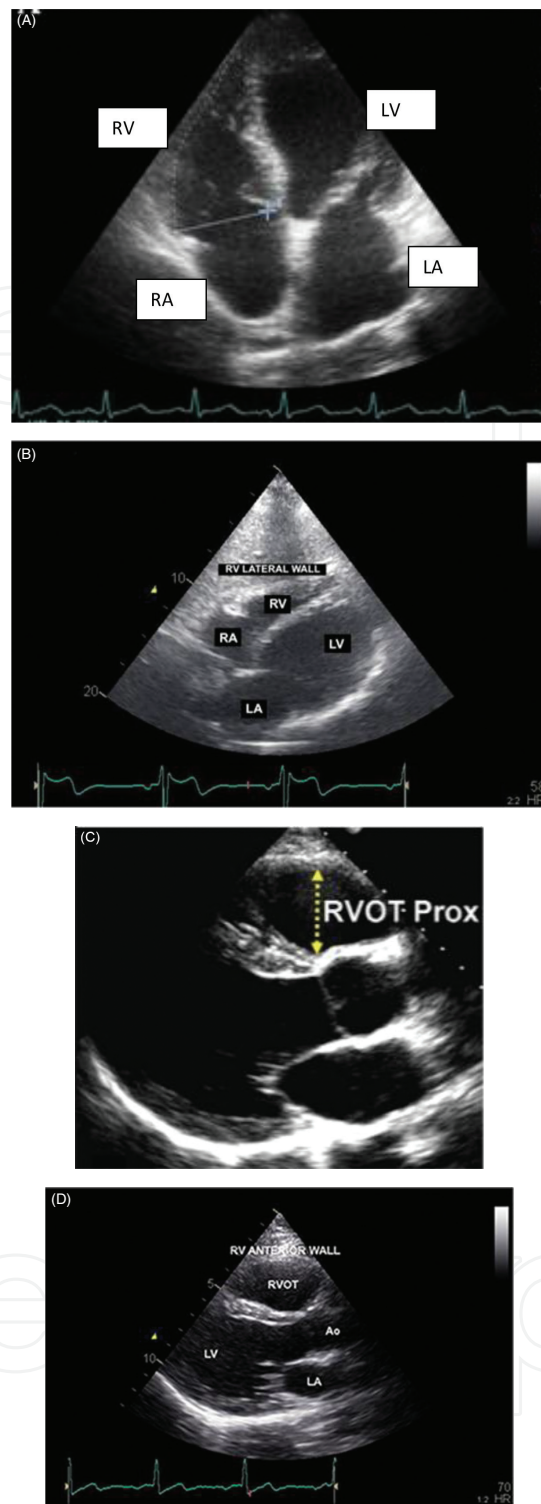


Figure 2. (A) RV basal apical four-chamber view: illustrating the plane of the tricuspid valve and RV endocardial border. (B) RV wall thickness (subcostal long axis view): illustrates the LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle; and RV lateral wall. (C) Proximal RVOT (parasternal short-axis view PSAX at the great vessels level): illustrates the Ao, aorta; PA, pulmonary artery; LV, left ventricle; RVOT, right ventricular outflow tract; and RV anterior wall. (D) Distal RVOT (parasternal long axis view PLAX): illustrates the Ao, aorta; LA, left atrium; LV, left ventricle; RVOT, right ventricular outflow tract, RV anterior wall.

2.5. Right ventricular outflow tract

The RVOT includes the pulmonic valve and subpulmonary infundibulum or conus that extends from the crista supraventricularis to the pulmonary valve [21, 22]. RVOT is usually imaged from the left parasternal short axis view. In patients with congenital heart disease and arrhythmogenic RV dysplasia, parasternal long axis view may be added to assess the proximal and distal diameter of RVOT. There is no standard window for measurement of RVOT size; oblique imaging may interfere in the accurate estimation of its size. The upper reference limit for the PSAX distal RVOT diameter is 27 mm and for PLAX is 33 mm (Table 5).

2.6. Interventricular septal morphology

Normally the LV has circular shape throughout the cardiac cycle. During systole, the LV protrudes into the RV. Compliance of one ventricle can modify the other through diastolic ventricular interaction. However, interventricular septum gets flattened and curved into LV cavity secondary to volume and pressure overload of the RV. The LV cavity, therefore, appears D-shaped at end-systole and end-diastole in RV pressure overload and RV volume overload (e.g., tricuspid regurgitation), respectively [17, 19] (Figure 3).

2.7. Volumetric assessment of RV

Two-dimensional TTE approximates complex RV geometry and underestimates MRI-derived RV volume. Assessment of RV volume using 3D TTE is superior and more accurate because 3D echo uses disc summation and apical rotational methods for RV volume and EF assessment [20]. However, the accuracy of RV volume assessment is less definite when the RV is dilated.

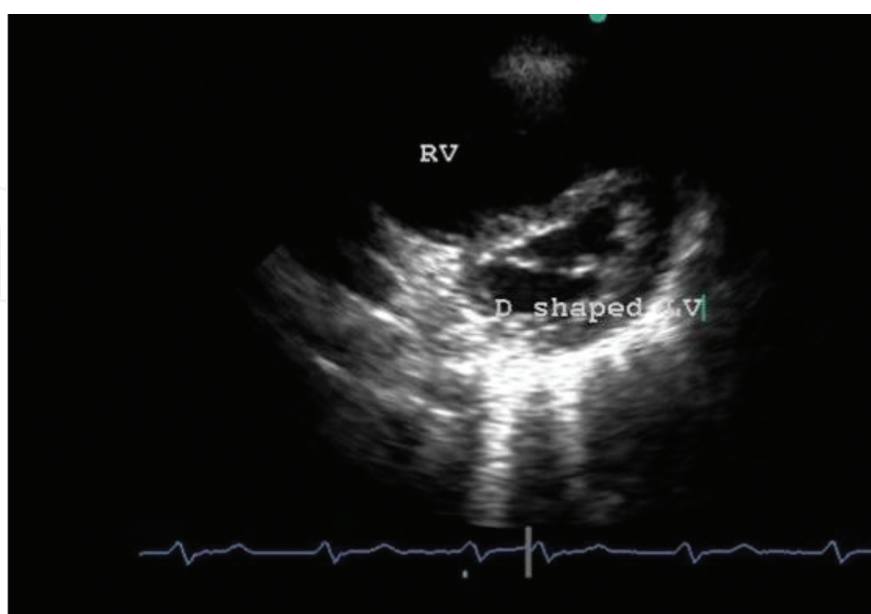


Figure 3. Example of RV with D-shaped LV cavity. RV, right ventricle; LV, left ventricle.

3. Intracardiac pressure measurement

Echocardiography can provide an estimate of right heart hemodynamics.

3.1. Estimated right atrial (RA) pressure

Estimation of right atrial pressure can be derived from the size of the inferior vena cava (IVC) and its response to changes in spontaneous respiration [23, 24]. Using a dilated IVC to assess elevated RA pressures is not accurate in mechanically ventilated patients. However, a small IVC of less than 1.2 cm in mechanically ventilated patient is 100% specific for an RA pressure of less than 10 mm Hg. Normal IVC is <2 cm in diameter, approximately 1 cm from RA-IVC junction and collapses by at least 50% with inspiration or sniff. A flat IVC indicates low RA pressure [0–5 mm Hg]. IVC <2 cm with normal inspiratory collapse indicates RA pressure of 5 mm Hg, and an IVC of >2 cm with normal inspiratory collapse suggests an RA pressure of 10 mm Hg. IVC <2 cm but without inspiratory collapse suggests 15 mm Hg RA pressure; IVC >2 cm but without inspiratory collapse suggests an RA pressure of 20 mm Hg. The normal RA pressure is 0–5 mm Hg.

3.2. Pulmonary artery systolic pressure (PASP)

Pulmonary artery (PA) systolic pressure can be determined from tricuspid regurgitation peak velocity. Provided there is no tricuspid valve obstruction, peak TR velocity depends on the pressure gradient between the right ventricle and right atrium [the difference between peak right ventricular systolic pressure (RVSP) and RA pressure] (**Table 4**). Therefore, estimated RVSP is equal to pressure difference (determined from peak TR velocity using Bernoulli equation) and estimated RA pressure [25]. (**Figure 4**) When there is no obstruction across the pulmonic valve, RVSP will be similar to PASP. $PASP = 4 \times \text{peak TR velocity}^2 + \text{estimated RA pressure}$. For example, if TR velocity is 2.5 m/sec and IVC is normal in size and collapses with inspiration the estimated PASP would be 33 mm Hg [$4(2.5)^2 \text{ mm Hg} = 25 \text{ mm Hg} + 5 \text{ mm Hg}$ (estimated RA pressure)]. If the estimated PASP is >35 to 40 mm Hg, pulmonary HTN is considered to be present.

3.3. Pulmonary artery diastolic pressure (PADP)

Pulmonary regurgitation (PR) represents the pressure difference between pulmonary artery and right ventricle. Hence, the end-diastolic pulmonary regurgitation velocity can be utilized to measure the end-diastolic pressure difference between PA and right ventricle. PA diastolic pressure can be estimated from the spectral Doppler signal of pulmonary regurgitation. The right ventricular end-diastolic pressure is the same as RA pressure; therefore, PADP can be estimated by addition of the estimated RA pressure to the end-diastolic pressure difference between PA and right ventricle. Thus, $PADP = 4 \times (\text{end-diastolic pulmonary regurgitation velocity})^2 + \text{estimated RA pressure}$.

Color flow regurgitant jet area of 30% or more of RA area
Annulus dilation (≥ 4 cm) or inadequate cusp coaptation
Late systolic concave configuration of the continuous-wave signal
Late systolic flow reversals in the hepatic vein
ERO of 0.4 cm^2 or larger
Regurgitant volume of 45 mL or more
Width of vena contracta of 6.5 mm or more

Abbreviations: ERO, effective regurgitant orifice; RA, right atrium.

Table 4. Severe TR is defined by echocardiography on the basis of the following criteria.

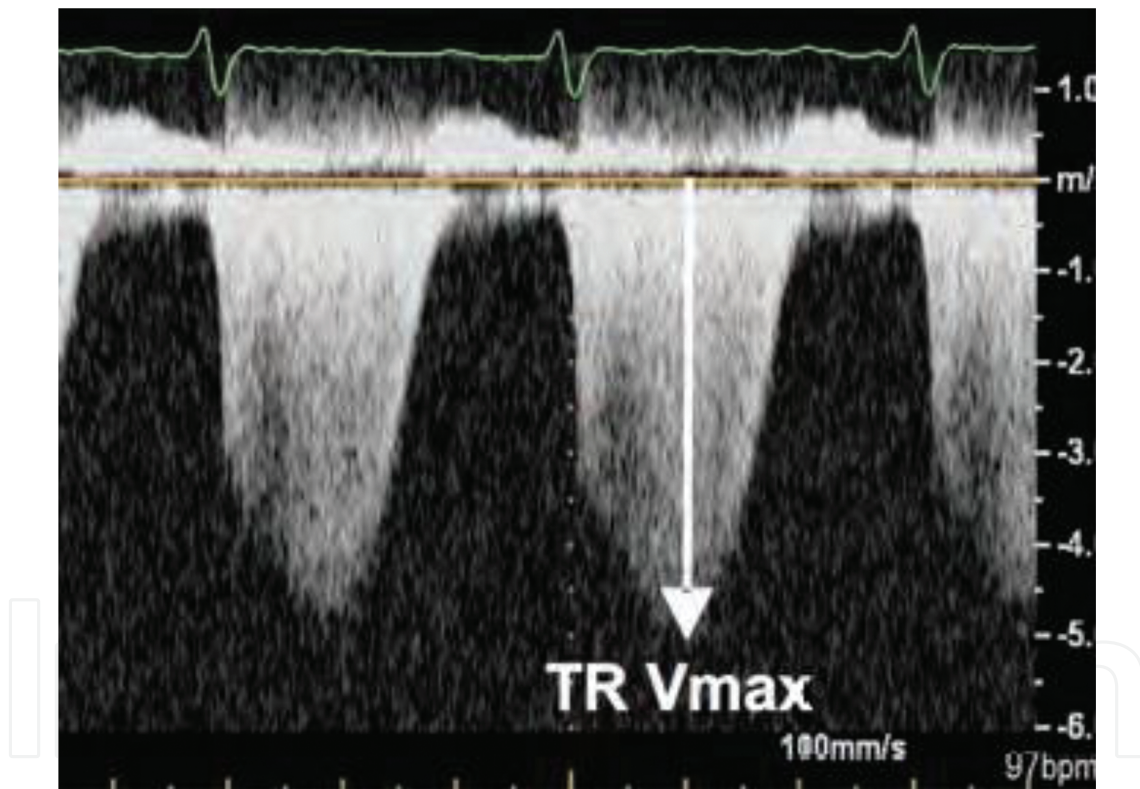


Figure 4. Peak TR velocity. $RVSP = 4(V_{max})^2 + RAP$. In the absence of pulmonic stenosis: $RVSP = PASP$. Peak TR velocity depends on pressure gradient between right ventricle and right atrium [difference between peak RVSP and RA pressure] provided there is no tricuspid valve obstruction. TR, tricuspid regurgitation; RAP, right atrium pressure; RVSP, right ventricle systolic pressure; V_{max} , peak TR velocity.

3.4. Mean pulmonary artery pressure

There are various formulae to estimate mean PA pressure [26–28]. Mean PA pressure = $1/3(PASP) + 2/3(PADP)$. Mean PA pressure can be estimated by pulmonary acceleration time

(AT) measured by pulsed Doppler of the pulmonary artery in systole. Mean PA pressure = $79 \times (0.45 \times AT)$ or if $AT < 120$ ms, mean PA pressure = $90 - (0.62 \times AT)$. Mean PA pressure = $4 \times (\text{early PR velocity})^2 + \text{estimated RA pressure}$. Mean PA pressure = estimated RA pressure + velocity-time integral of the TR jet to calculate a mean systolic pressure.

3.5. Pulmonary vascular resistance (PVR)

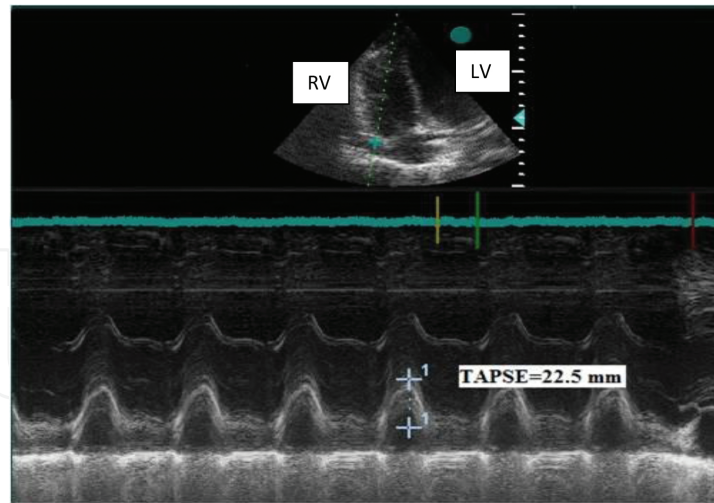
As per the formula $P = QR$, where pressure (P) equals the product of flow (Q) and resistance (R), PASP can be elevated in the setting of increased stroke volume without PVR being elevated. PVR can be calculated by the ratio of peak TR velocity (m/s) to RVOT VTI (velocity time integral) (cm) [29, 30]. $PVR = [(TR \text{ velocity}/RVOT \text{ VTI}) \times 10] + 0.16$. PVR value is in Woods units (WU) and correlates well with invasively measured PVR up to approximately 8 WU [30]. However, when PVR is >8 WU by invasive hemodynamic measurement the relationship is not reliable. This method is not validated and should not be used for routine clinical purposes in place of invasive hemodynamic measurements. It can be used when PASP is elevated from increased stroke volume or PASP is low (despite increased PVR) from decreased stroke volume.

4. Assessment of RV function

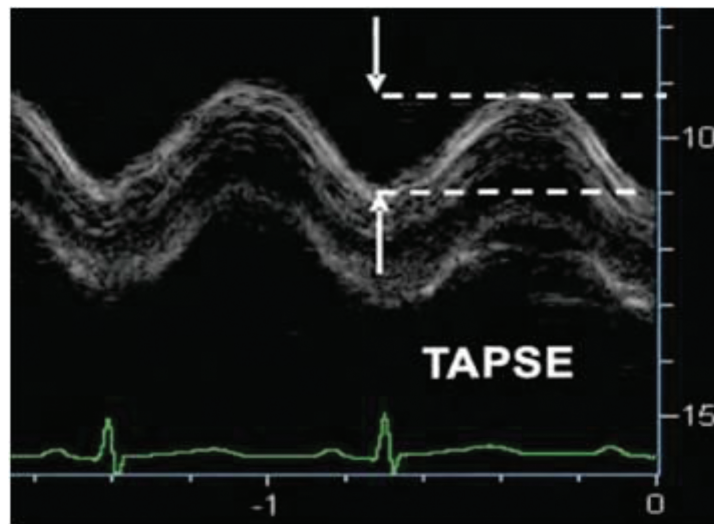
Most of the RV contraction occurs longitudinally from base to apex (contributing to most of the RV stroke volume), along with radial thickening/inward motion. The following techniques are used to assess RV function [15, 16, 31, 32].

4.1. Tricuspid annular planar systolic excursion (TAPSE)

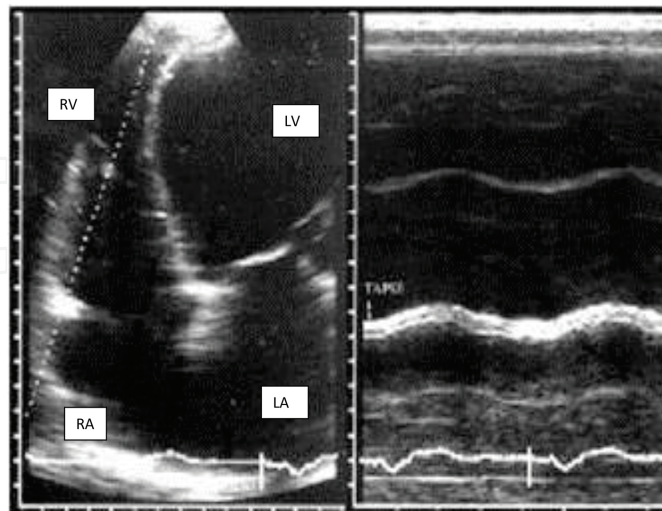
TAPSE is a diagnostic and prognostic tool of mortality and morbidity in patients with precapillary pulmonary hypertension, RV infarction associated with inferior myocardial infarction, and chronic left-sided heart failure [33, 34]. TAPSE is assessed in an apical four-chamber view by placing the M-mode on the lateral tricuspid annulus; maximum systolic excursion of the lateral annulus along its longitudinal plane toward the apex is recorded [33, 35]. The displacement of the basal segment from the reference point reflects longitudinal contraction of the RV. Normal reference limit is TAPSE of >1.6 cm [36, 37]. This is the most commonly used method as it is a simple, easily obtainable, reproducible with a low interobserver variability. For accurate estimation of TAPSE, one should place M-mode cursor parallel to the plane of longitudinal motion carefully measuring the magnitude of displacement from the M-mode image. The limitations of this method are that TAPSE estimates only the longitudinal contraction within one segment of RV and hypothesizes that the function of a single RV segment reflects the entire RV function which is not true in conditions like RV infarction and pulmonary embolism (Figure 5).



A



B



C

Figure 5. A: Example of a normal TAPSE (tricuspid annular planar systolic excursion) value >1.6 cm; B: normal TAPSE; C: reduced TAPSE.

4.2. Tricuspid annular velocity S'

The tricuspid annular velocity is also known as systolic excursion velocity S'. In an apical four-chamber view, the cursor of pulsed tissue Doppler or color-coded tissue Doppler is placed on the lateral tricuspid annulus to measure the longitudinal velocity of excursion of basal-free wall segment and tricuspid annulus in systole. Normal reference limit (**Table 5**) of S' is >9.5 cm/s. Color-coded tissue Doppler yields lower velocities and is analyzed off-line on specific platforms. The advantages and disadvantages are similar to TAPSE.

RV systolic dysfunction

TAPSE \leq 1.6 cm

Pulse Doppler peak annular velocity at tricuspid annulus S' <9.5 cm/s

2D RV FAC <35%

Tei index/RIMP >0.40 by pulsed Doppler and >0.55 by tissue Doppler

RVEF 3D \leq 44%

RV diastolic dysfunction

E/A <0.8 by tissue Doppler

E/A >2.1 by tissue Doppler

Dilated RV chamber

Basal RV diameter >4.2 cm

Mid-level diameter >3.5 cm

Longitudinal dimension >8.6 cm

Abnormal RVOT value

RVOT in PSAX distal diameter >2.7 cm

RVOT in PLAX proximal diameter >3.3 cm

Increased RV subcostal wall thickness >0.5 cm

Abbreviations: 2D, two-dimensional; 3D, three-dimensional; FAC, fractional area change; MPI, myocardial performance index; PLAX, parasternal long-axis; PSAX, parasternal short-axis; RA, right atrium; RV, right ventricle; RVOT, right ventricular outflow tract; TAPSE, tricuspid annular plane systolic excursion; RIMP, right ventricular index of myocardial performance.

Table 5. Echocardiographic parameters for assessment of right ventricle based on ASE recommendations.

4.3. Myocardial performance index (MPI)

The myocardial performance index is also denoted as the right ventricular index of myocardial performance (RIMP) or right ventricular Tei index. It is an index of global ventricular function and is independent of the geometry of the ventricle [38]. The MPI is calculated by the ratio of isovolumetric time interval over ventricular ejection time as follows: MPI = (isovolumetric

relaxation time + isovolumetric contraction time)/ventricular ejection time = (tricuspid closure to opening time – ejection time)/ejection time. Lower MPI values indicate healthy RV function as less time is utilized in isovolumetric state and more time is consumed in ejecting blood. MPI can be measured through pulsed Doppler or tissue Doppler methods (**Figure 6**).

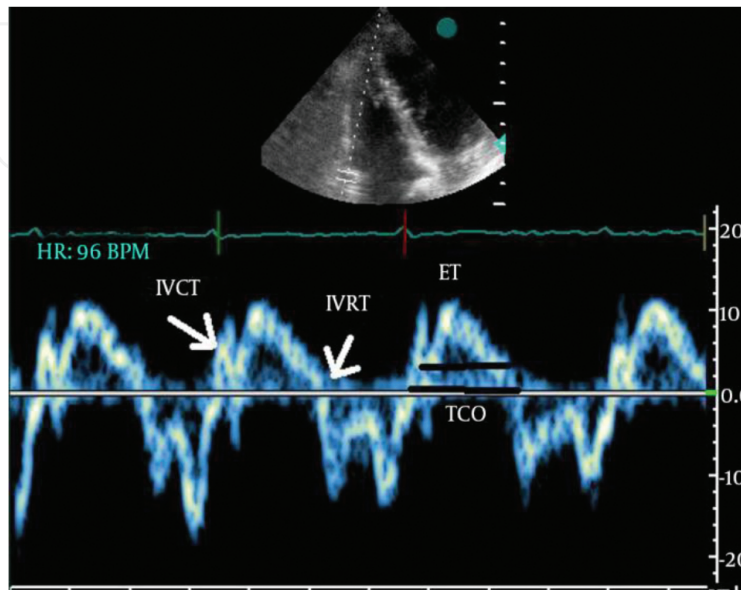


Figure 6. Pulse Doppler MPI. Calculation of RIMP by pulse tissue Doppler imaging $RIMP = (TCO - ET)/ET$ or $IVRT + IVCT/ET$. RIMP, right ventricular index of myocardial performance; TCO, tricuspid valve closure to opening time; IVCT, isovolumetric contraction time; IVRT, isovolumetric relaxation time; ET, ejection time.

4.3.1. Pulsed Doppler method

In the pulsed Doppler method, pulsed wave Doppler tracing of the distal RVOT is used to obtain ejection time, while the tricuspid-closure-opening time is calculated from the pulsed wave Doppler tracing of the tricuspid inflow (time from end of the A wave to the onset of the following E wave) or the continuous wave Doppler tracing of the tricuspid regurgitation jet. The total isovolumetric time is calculated from the difference between the tricuspid-closure-opening time and the ejection time. The normal reference limit for the pulsed Doppler MPI is <0.40.

4.3.2. Tissue Doppler method

Tissue Doppler method obtains the ejection time, tricuspid-closure-opening time, and total isovolumetric time from the pulsed tissue Doppler tracing of the lateral tricuspid annulus. The normal reference limit for the tissue Doppler MPI is <0.54.

MPI is a sensitive parameter to evaluate subclinical or early RV dysfunction even in poorly visualized RV because it depends on time intervals [39]. However, MPI is observer dependent as delineating time intervals can be challenging [40].

4.4. Fractional area change (FAC)

FAC is the percent change in RV area from diastole to systole.

FAC = [(end-diastolic RV area – end-systolic RV area)/end-diastolic RV area] × 100. FAC is best correlated with MRI-derived RVEF. RV endocardium is traced 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. The RV wall should be carefully traced under the trabeculations. FAC has prognostic value and is an independent predictor of all-cause mortality in patients with acute myocardial infarction and low left ventricular ejection fraction. The reference value for normal RV systolic function is >35%.

5. Pulse Doppler MPI

5.1. Three-dimensional echocardiogram

3D echo combined with intravenous contrast agents can improve endocardial border delineation and RV end-diastolic and end-systolic volumes.

$$\text{RVEF} = \frac{[\text{end – diastolic RV volume} - \text{end – systolic RV volume}]}{\text{end – diastolic RV volume.}} \quad (1)$$

The RV volumes measured by 3D echo use disk summation or surface modeling method. Although 3D echo-derived RVEF correlates well with MRI-derived RVEF, the method is complex, time-consuming, and very much dependent on image quality [20]. The normal reference limit for 3D-derived RVEF is >45%.

5.2. Strain imaging by 2D

The strain is the degree of myocardial deformation, while strain rate represents the rate of myocardial deformation over time [38]. In echocardiography, RV longitudinal strain can be assessed reliably from apical views, whereas the assessment of radial strain is challenging from the parasternal views because of near-field artifacts and extremely small computational distance. The crescent shape of thin-walled RV contributes to inhomogeneous strain rate and values with the highest values in the apical segments and outflow tract. One-dimensional strain is measured using a tissue Doppler (angle-dependent) [18], while 2D strain is measured by speckle tracking (non-angle-dependent). 2D strain imaging estimates global and regional RV function, reflects intrinsic contractility of the RV (with contractility defined as the less stress-strain interplay), and evaluates diastolic properties [39, 41].

Disadvantages are a dearth of normative data, challenges in the adequate image acquisition and analysis requiring high frame rates, high signal-to-noise ratio, minimal image dropout, and most notably the need for experienced observers for reproducible measurements. As it is

not highly reproducible, this technique is not recommended for routine use. Given high variability, no reference limits are available [40].

6. Clinical and prognostic significance of assessment of right ventricle

Quantitative assessment of RV size and function has prognostic value regarding exercise tolerance and outcome in various cardiac and pulmonary diseases [3, 36, 42, 43]. RV pump function depends on contractility, afterload, preload, heart rate, rhythm, and valve function. Being a thin-walled chamber, it is not suited to sustain high pressure (**Tables 1 and 2**). RV dysfunction can be acute or chronic, secondary to RV volume overload, pressure overload, or decreased contractility.

6.1. RV overload

RV overload can be related to pressure overload or volume overload. RV overload, in turn, reduces LV diastolic function and causes higher filling pressures.

6.1.1. Volume overload

Volume overload can result from tricuspid regurgitation, pulmonary regurgitation, ASD, and VSD and is assessed through the movement of IVS. Normally during systole, IVS thickens and moves into the left ventricle and during diastole, it moves into the RV cavity. In RV volume overload, RA and RV are enlarged, and IVS is pushed into the LV during end-systole and early diastole as RV pressure exceeds LV pressure. This leads to IVS flattening and a D-shaped LV only during early diastole. At the outset of systole, LV contraction increases LV pressure pushing the IVS in the direction of RV cavity [12].

6.1.2. Pressure overload

Pressure overload can be acute or chronic. Acute pressure overload can be from adult respiratory distress syndrome (ARDS) or massive pulmonary embolism. Echocardiographic findings of RV pressure overload are the same as volume overload. RA and RV are enlarged with no hypertrophy of RV-free wall; there is a flattening of the IVS in diastole. The peak RV systolic pressures rarely exceed 50 mm Hg in acute pressure overload. Chronic pressure overload is secondary to chronic lung diseases, chronic thromboembolism, or chronic pulmonary venous hypertension from left heart pathology. RV is enlarged with thickening of the RV-free wall and increased trabeculation. The RV can generate higher peak systolic pressures, usually exceeding 50 mm Hg. The IVS remains flattened into the LV cavity during the entire cardiac cycle.

6.2. Right ventricular diastolic function

RV diastolic dysfunction has prognostic value and has been associated with both acute and chronic conditions. RV diastolic function is assessed like that of the left ventricle. Techniques

used are Doppler velocities of the trans tricuspid flow (E, A, E/A), tissue Doppler velocities of the tricuspid annulus (E', A', and E'/A'), deceleration time, and isovolumetric relaxation time [20, 40]. Estimation of RA pressure by measurement of IVC diameter and collapse with inspiration is to be considered while determining the RV diastolic function.

6.3. Cardiac rhythm and the RV

RV function is dependent on cardiac rhythm. RV function is compromised by atrial fibrillation and ventricular tachycardia originating from the RV examples of which are arrhythmogenic RV dysplasia, RV myocardial infarction, idiopathic ventricular tachycardia, or ventricular tachycardia occurring after surgical repair of congenital heart disease [44].

6.4. Cardiac markers

The elevated B-type natriuretic peptide is associated with RV failure secondary to pulmonary hypertension, congenital heart disease, or pulmonary disease [45–47]. Elevated troponin levels indicate poor prognosis in pulmonary embolism and pulmonary hypertension [48].

6.5. Evaluation of pulmonary arterial hypertension

Pulmonary arterial hypertension is a clinical entity that is seen as a consequence of both left heart disease and pulmonary pathology, as well as occurring without an underlying etiology such as primary pulmonary arterial hypertension. Estimation of pulmonary artery pressure can be performed by TTE in the majority of patients [37, 49].

6.6. Evaluation of patients with pulmonary embolism

Pulmonary embolism is associated with high mortality and morbidity; hence, prompt diagnosis and treatment is imperative. When a patient has had a large pulmonary embolism, this may place acute pressure overload on the right ventricle. The right ventricle handles pressure poorly and may undergo acute dilation with decreased right ventricular systolic function as a result of an acute increase in afterload. Usually, peak systolic pressures in the pulmonary artery do not exceed 50 mm Hg unless there is a baseline chronic RV pressure overload. The size and function of the RV are among the most important factors in determining the initiation of either thrombolysis or referral for surgical embolectomy. A classic pattern of RV systolic dysfunction in acute pulmonary embolism has been described. This is known as McConnell sign and is characterized by akinesis of the free wall of the right ventricle with sparing of the apical segment. This phenomenon has 77% sensitivity and 94% specificity for the diagnosis of acute pulmonary embolism (**Figure 7**).

6.7. Evaluation of RV dyssynchrony

Echocardiographic indices of dyssynchrony are assessed by measuring time delay in mechanical activity between segments. Tissue Doppler imaging is limited to the assessment of the septum-RV free wall.

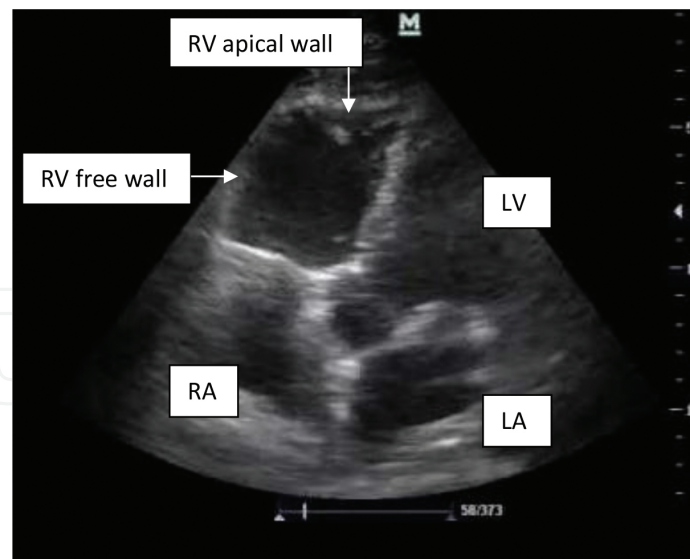


Figure 7. McConnell sign. McConnell sign: akinesis of the free wall of the right ventricle with sparing of the apical segment seen in acute pulmonary embolism causing right ventricle systolic dysfunction.

7. Conclusion

Accurate quantitative assessment of right ventricular size and function remains difficult given its unique shape despite significant advances in echocardiography. RV dysfunction is an important diagnostic and prognostic indicator in many cardiac and pulmonary diseases [42, 43, 50, 51]. Qualitative evaluation of RV systolic function is through visual assessment. For quantitative assessment of RV, FAC, TAPSE, pulsed tissue Doppler S', and MPI are available and at least one of them should be routinely performed and reported as recommended by the ASE. If more than one of these measurements is used in conjunction, RV function can be more reliably and accurately assessed [36, 40, 52].

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