Chapter 8
The Right Heart

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Abstract Historically, clinical and research efforts have primarily focused on the left ventricle. However, relatively recent identification that evidence of right heart dysfunction increases the perioperative morbidity and mortality of patients undergoing both cardiac and noncardiac surgery has brought the echocardiographic evaluation of the right heart into the spotlight. This chapter serves to identify the unique concerns when evaluating echocardiographically the complex geometry and physiology of the right heart as well as its unique adaptive properties in response to acute and chronic afterload increases. Both anatomical assessments and quantitative methods of evaluating right ventricular function will be discussed. Transesophageal echocardiography therefore plays a key role in the identification and management of those patients at risk for right heart dysfunction.

Keywords Right ventricle · Right atrium · Pulmonary hypertension · Pulmonary embolism · Tricuspid regurgitation · Pulmonary artery

Introduction

The right ventricle (RV) historically has been overlooked and viewed as less important than the left heart. However, recently there has been considerable interest in understanding the function of the right ventricle. Right ventricular dysfunction has been associated with increased morbidity and mortality within both cardiac and

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noncardiac surgical arenas. The main function of the RV is to enhance venous return by accommodating systemic return into a low-pressure chamber and subsequently transit blood to the left heart via the low-pressure pulmonary circuit. Recognition of right heart dysfunction due to acute pathology such as pulmonary embolism or chronic pathology such as pulmonary hypertension is key to perioperative management. Transesophageal echocardiography (TEE) plays a significant role both in the identification and quantification of perioperative right heart dysfunction.

Right Ventricle Structure and Function

The RV forms the major portion of the anterior surface of the heart immediately behind the sternum, while the inferior border of the RV abuts the diaphragm. The right ventricle is an asymmetric crescent-shaped chamber that is wrapped around the right and anteroseptal side of the left ventricle (LV). In healthy individuals, RV dimensions are roughly two-thirds the size of LV dimensions. The RV is generally separated into three portions: inflow, apical, and outflow. These distinct regions are separated by a series of encircling muscular bands. The inflow portion is trabeculated and consists of the tricuspid valve, chordae tendinae, and papillary muscles. The apical portion is also a muscular portion connecting the inflow to the smooth-surfaced outflow tract. The outflow region therefore contains the smooth myocardial outflow tract and pulmonic valve [1, 2].

The encircling muscular bands include four individual bands, however the moderator band is most important to the echocardiographer. The moderator band extends from the base of the anterior papillary muscle near the ventricular free wall to the ventricular septum. When it becomes prominent from right ventricular hypertrophy, the moderator band may be mistaken for a thrombus or intracavitary mass [3, 4].

The RV wall motion is complex, characterized by a peristaltic-like motion. Contraction is sequential, beginning with the inlet portion contracting toward the apex and ending with the infundibulum. RV longitudinal shortening occurs mainly during the ejection phase of the cardiac cycle, while circumferential motion occurs mainly during the isovolumic contraction phase [5, 6].

The RV is mostly supplied by the right coronary artery (RCA) with the posterior descending artery (PDA) supplying one-third to two-third of the RV septum. The left anterior descending artery (LAD) may supply a portion of the apex, while branches off of the RCA, the acute marginal arteries, supply the RV free wall. Lastly, in the majority of patients, the RCA supplies perfusion to the atrioventricular (AV) node and sinoatrial (SA) node.

The RV provides a similar stroke volume as the LV, however with 25 % of the stroke work due to the low-pressure, high-compliance circuit in the pulmonary circulation [7]. As a result, the wall thickness of the RV is half the size of LV wall, making the RV a much more compliant chamber. The right and left ventricles share

the interventricular septum (IVS) and are enclosed within the same pericardial sac. Therefore, ventricular interdependence exists. The IVS contributes to both LV and RV function, is responsible for approximately one-third of the RV stroke work under normal conditions, and is a major determinant of overall RV performance [8–10]. Normally the IVS is shifted toward the RV free wall (concave toward the LV) during systole and diastole, contributing to RV ejection. However, this relationship is changed in conditions that lead to an increase in RV pressure or volume overload, shifting the septum paradoxically toward the LV, altering LV geometry, resulting in decreased LV preload and low cardiac output [11].

Transesophageal Echocardiographic Views for Evaluation of the Right Ventricle

Qualitative evaluation of the RV should allow the examiner to answer five questions as illustrated in Table 8.1. As described in Chap. 2, there are several views that focus on evaluation of the right heart. They are briefly outlined below.

Midesophageal four-chamber—A midesophageal (ME) view with a multiplane angle of 0–20° allows for evaluation of RV chamber size, function, and anatomical variants [12, 13]. From a standard ME four-chamber view, turning the probe to the right (clockwise) allows imaging of the right atrium (RA), right ventricle, tricuspid valve (TV), as well as the interatrial and interventricular septums. This view is the most utilized view to assess the right heart.

ME RV inflow–outflow view—Placing the right heart in the center of the imaging sector and increasing the multiplane angle to 60–90° will develop the ME RV inflow–outflow view. This view demonstrates the "wrapping" nature of the RA, RV, and pulmonary artery (PA) in relation to the left heart. Assessment of the RA, TV, RV free wall, RV outflow tract, pulmonic valve (PV), and main PA can be accomplished in this view. With an image of both the TV and PV, this view is used to help guide placement of the pulmonary artery catheter as well as estimate RV systolic pressure.

ME bicaval view—From the ME RV inflow–outflow view, increasing the multiplane angle to 90–110° and turning the probe to the right develops the ME bicaval view. This view displays the LA, RA, and the interatrial septum. In the

Table 8.1 Outline of echocardiographic evaluation of the right heart

Echocardiographic evaluation of the RV should allow the examiner to answer the following five questions
1. What is the RV Shape?
2. What is the RV area relative to LV area?
3. What is the RV free wall thickness?
4. How is the RV free wall motion?
5. What is the interventricular septal motion?

standard bicaval view, the SVC and IVC, as well as the right atrial appendage, are in view. Turning of the probe slightly left (counterclockwise) or increasing the multiplane angle by 10–20° will bring the tricuspid valve and coronary sinus in view. This adjustment results in the modified bicaval view [2, 12].

ME ascending aortic short axis view—This view demonstrates the relation of the main and right PA to the ascending aorta and SVC, and proves useful in identifying PA dilatation and PA thrombus, as well as confirming the position of a properly placed PA catheter.

Transgastric (TG) midpapillary short axis view—With a rightward probe rotation from a standard TG midpapillary short axis view, an image of the crescent-shaped RV is developed. Again, the anterolateral position of the RV in relation to the left ventricle (LV) is noted.

TG RV inflow view—Increasing the multiplane angle to 90° from the prior view yields the TG RV inflow view. This view shows a "two-chamber" view of the right heart with a focus on the tricuspid valve and the subvalvular apparatus, as well as the right ventricular free wall.

Physiology of the Right Heart: Adaptations to Pulmonary Hypertension

The pulmonary circulation is normally a low-pressure and low-resistance circuit in comparison to the systemic circulation. The normal systolic, diastolic, and mean pulmonary artery pressure (PAP) are 22, 10, and 15 mmHg, respectively. The pulmonary vascular resistance (PVR) is normally 0.9–1.4 Wood units, or about 90–120 dynes sec cm^{-5}. Pulmonary hypertension (PH) is generally defined as a mean PAP of greater than 25 mmHg, or a PVR greater than 300 dynes sec cm^{-5}. A mean PAP greater than 50 mmHg or a PVR greater than 600 dynes sec cm^{-5} is considered severe PH. The PVR is important because it represents the afterload of the right ventricle (the pressure encountered during ejection), and therefore, affects RV function and cardiac output.

Emphasis on PH classification began in 1973 at the World Health Organization conference and since then, has undergone multiple changes as the appreciation of the disease and treatment of PH has evolved, resulting in five distinct subgroups of patients sharing specific features [13–15] (Table 8.2). The effects on the RV in response to pulmonary hypertension are readily identified on echocardiography. The adaptations are generally categorized as RV dilation in response to volume overload (inability to properly move the volume through the pulmonary circuit) and RV hypertrophy in response to the increase afterload (inability to eject a proper stroke volume due to increased resistance). Both of these adaptations, when progressive, lead to RV dysfunction. Additionally, septal wall motion may become abnormal with a deviation toward the left heart, further contributing to RV dysfunction. An echocardiographic evaluation with particular focus on the right side of the heart allows both the identification as well as quantification of RV dysfunction.

Table 8.2 Classification of pulmonary hypertension

Definition pulmonary hypertension (PH)	Clinical group(s)
Precapillary: MPAP > 25 mmHg PCWP < 15 mmHg	**Group I**: Pulmonary arterial Hypertension (PAH) and other subtypes of PAH **Group III**: World Respiratory disease and hypoxemia **Group IV**: Chronic thromboembolic pulmonary hypertension (CTEPH) **Group V**: Miscellaneous causes
Postcapillary: MPAP > 25 mmHg PCWP > 15 mmHg	**Group II**: Left heart disease

Adapted from McLaughlin et al. [16], Bossone et al. [17]. Mean pulmonary artery pressure (MPAP); Pulmonary capillary wedge pressure (PCWP)

Right Ventricular Enlargement

The normally thin-walled, crescent-shaped RV allows the RV to be highly compliant, accommodating a large increase in preload to maintain a normal stroke volume in the early stages of RV dysfunction. Consequently, the initial primary compensatory mechanism of RV dysfunction often is dilatation that is usually well tolerated. With progressive dilation, the ability to maintain a normal stroke volume is lost. Qualitative visual measures are typically used to assess RV chamber size in the ME four-chamber view. RV size normally occupies two-thirds of the cross-sectional area in comparison to LV area. Additionally, the LV typically forms the cardiac apex in this view. With mild enlargement, the RV increases in size to greater than two-thirds of the LV cross-sectional area; with moderate enlargement, the RV apex includes both the RV and LV and the cross-sectional areas are equal; while with severe enlargement, the RV apex completely forms the cardiac apex, and the cross-sectional area of the RV exceeds that of the LV (Fig. 8.1; Video 8.1).

Additionally two-dimensional measurements of the RV in the ME four-chamber view can be utilized to assess RV size. The RV basal dimension [measure of the width of the TV annulus in the ME four-chamber view] ranges from 2 to 2.8 cm with 4.0 cm as the reference limit of a severely dilated annulus [18]. Longitudinal measurements from the tricuspid valve plane to the RV apex range from 7.1 to 7.9 cm, with a 9.2 cm measurement indicating a severely dilated RV [19, 20].

Right Ventricular Hypertrophy

Echocardiographic evaluation of RV wall thickness may also serve to evaluate global RV performance because conditions of RV pressure overload cause

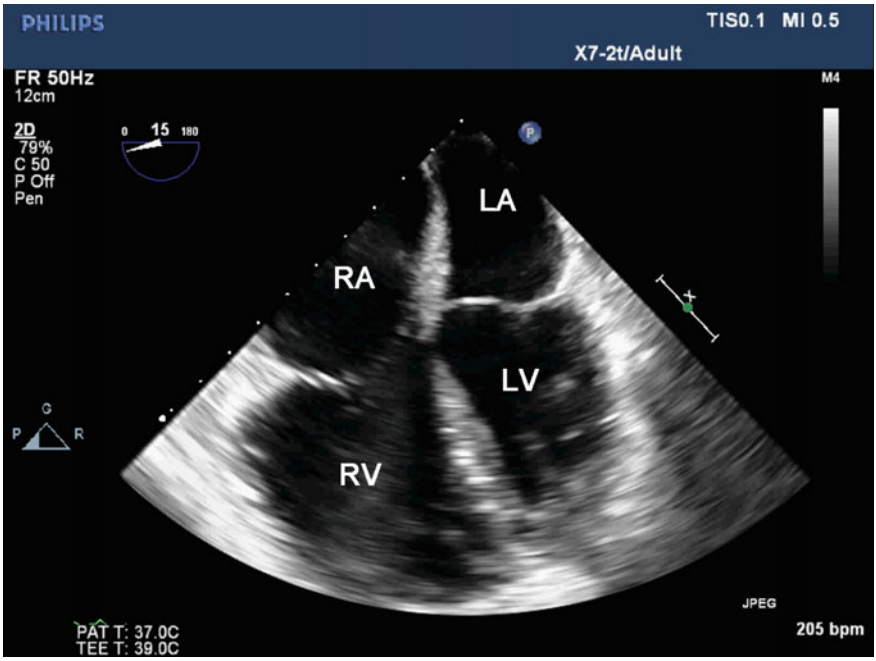

Fig. 8.1 A midesophageal four-chamber view of a patient with pulmonary hypertension and severe right ventricular enlargement. Note that the size of the right ventricle (RV) exceeds that of the left ventricle (LV) and the apex of the heart appears to be formed by the RV. *RA* right atrium; *LA* left atrium

compensatory right ventricular hypertrophy (RVH). This increase in RV myocardial mass is an effort to maintain cardiac output in the presence of increased PVR.

TEE evaluation of RV wall thickness typically assesses the lateral RV free wall thickness. Normally, the RV is thin-walled with RV free wall thickness (RVWT) approximately half the wall thickness of the LV, measuring <5 mm at end-diastole. RVH may be diagnosed when the RVWT is >5 mm, while RVWT exceeding 10 mm is considered severe hypertrophy (Fig. 8.2). The best views to measure the RV free wall are in the ME four-chamber, ME RV inflow–outflow, or TG RV inflow views. Measurements of RVWT occur during myocardial relaxation (diastole) and must exclude the epicardial fat layer. Additionally, the moderator band is usually more prominent in patients with RV hypertrophy and is often visualized near the RV apex (Fig. 8.3; Video 8.2).

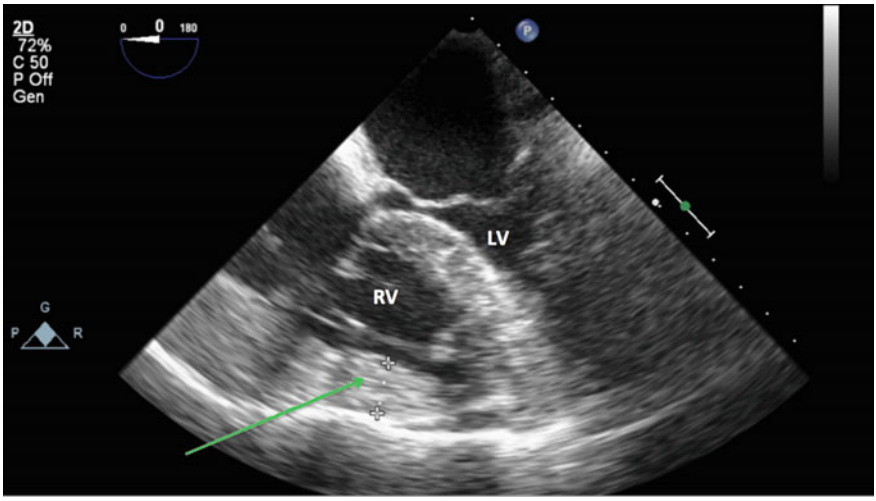

Fig. 8.2 A midesophageal four-chamber view of a patient with chronic pulmonary hypertension and resultant right ventricular hypertrophy (*green arrow*)

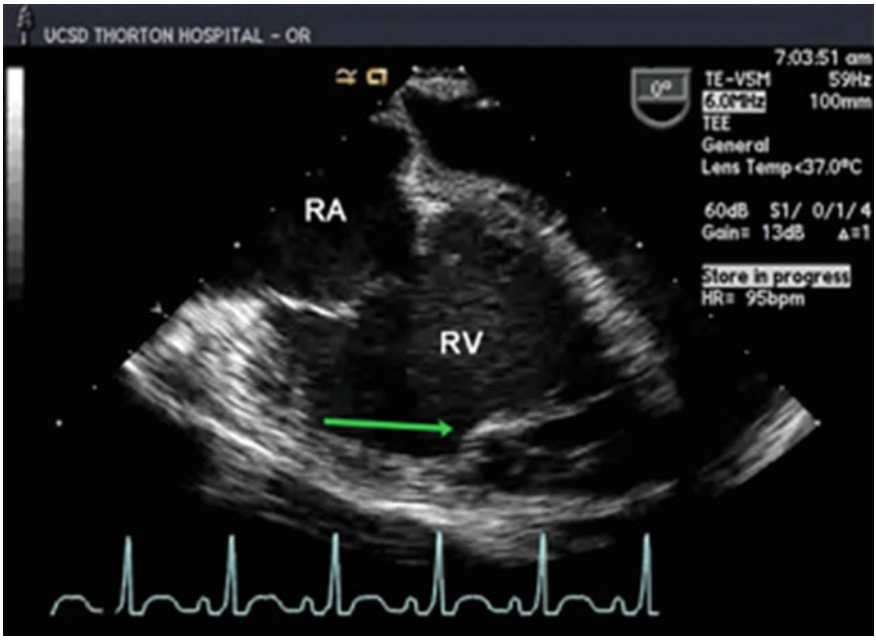

Fig. 8.3 A midesophageal four-chamber view of a patient with severe right ventricular and right atrial enlargement. A prominent moderator band (*green arrow*) is present

Quantitative Assessment of Right Ventricular Function

Right ventricular dysfunction	
2D	• Right Atrial Enlargement • Right Ventricular Enlargement and/or hypertrophy • IAS/IVS shifted toward the left heart • Underfilled LA and LV • Decreased TAPSE (M-mode) • Decreased FAC
CFD	• Tricuspid regurgitation
Spectral	• TDI: Tricuspid Annular Velocity (S') • PASP Estimation • Hepatic Vein confirmation of TR

IAS Interatrial septum; *IVS* Interventricular septum; *LA* Left atrium; *LV* Left ventricle; *TAPSE* Tricuspid annular plane systolic excursion; *FAC* Fractional area of change; *TDI* Tissue Doppler imaging; *PASP* Pulmonary artery systolic pressure; *TR* Tricuspid regurgitation

A quantitative evaluation of RV global function is more difficult to achieve owing to its more complex shape [19–21]. Unlike the shape of the LV which allows some geometrical assumptions (e.g., Simpson's method of discs), the shape of the RV is complicated as evidenced by its triangular shape in the ME four-chamber view, "wrap around" nature in the ME RV inflow–outflow view, and crescent shape in the TG midpapillary short axis view. Additionally, contraction of the RV differs from the LV's concentric and "piston-like" longitudinal contraction. The RV contracts in a peristaltic-like manner from the base toward the apex and subsequently the outflow tract. The major contributor to RV ejection is the basal contraction, and therefore constitutes a basis for several methods of quantitative RV analysis. This complex shape and differing contraction has necessitated surrogate parameters to be developed and subsequently validated for RV systolic function.

Tricuspid Annular Plane Systolic Excursion (TAPSE)

TAPSE measures the longitudinal movement of the tricuspid annulus toward the apex during systole. Again the basal contraction of the RV is a large contributor to RV ejection; therefore TAPSE is a single measurement that extrapolates and estimates global RV function. Normal TAPSE measurement is greater than 17 mm, therefore, TAPSE values <17 mm are suggestive of RV systolic dysfunction [18]. While often obtained as a visual estimate in noncardiac surgery, measurement of

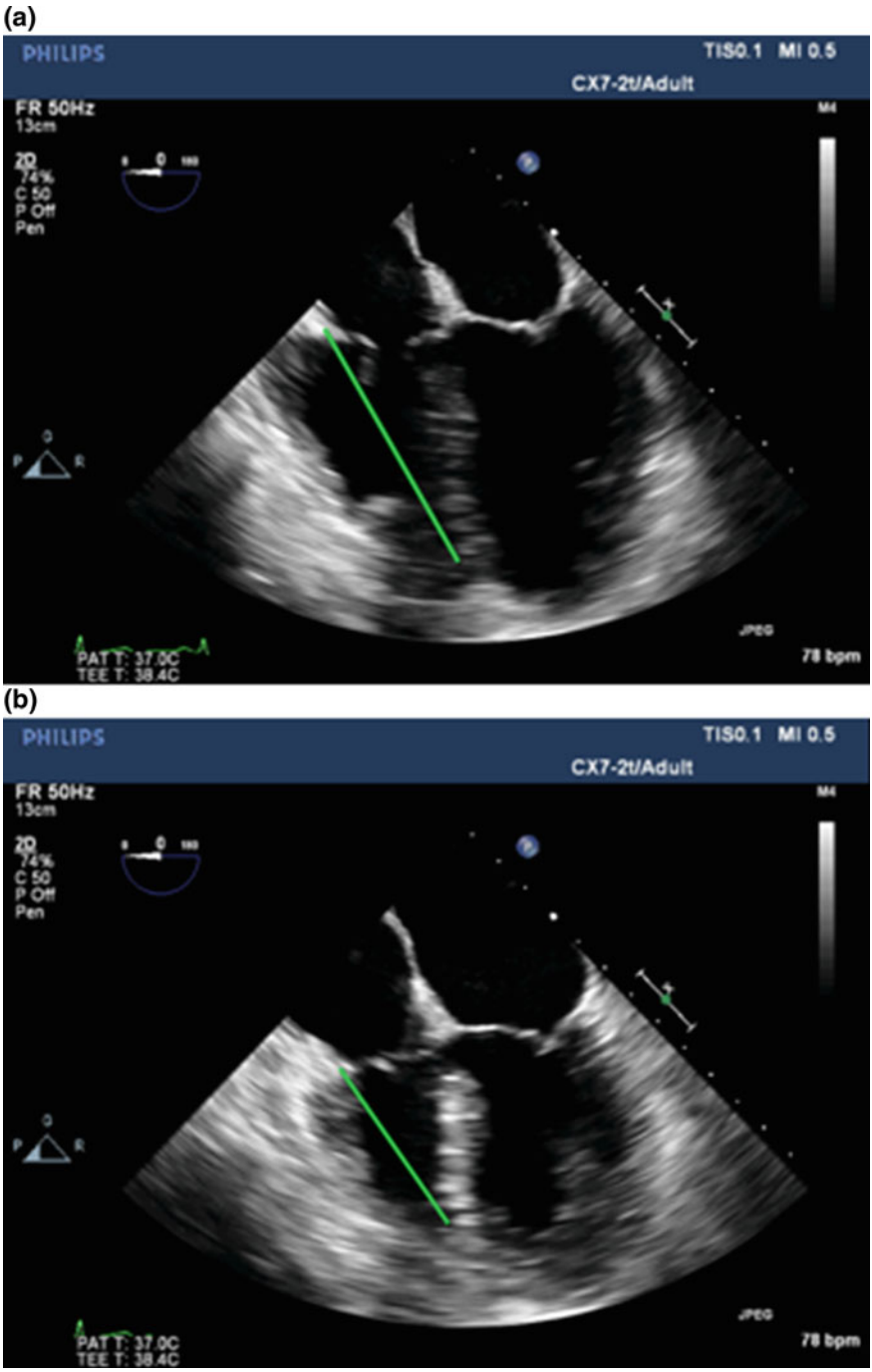

Fig. 8.4 **a** A midesophageal four-chamber view with a *green line* depicting measurement of the distance from the tricuspid annulus to the apex in diastole. **b** A systolic frame from the same patient with a *green line* depicting the distance from the tricuspid annulus to the apex. TAPSE is calculated as the difference of these two measurements

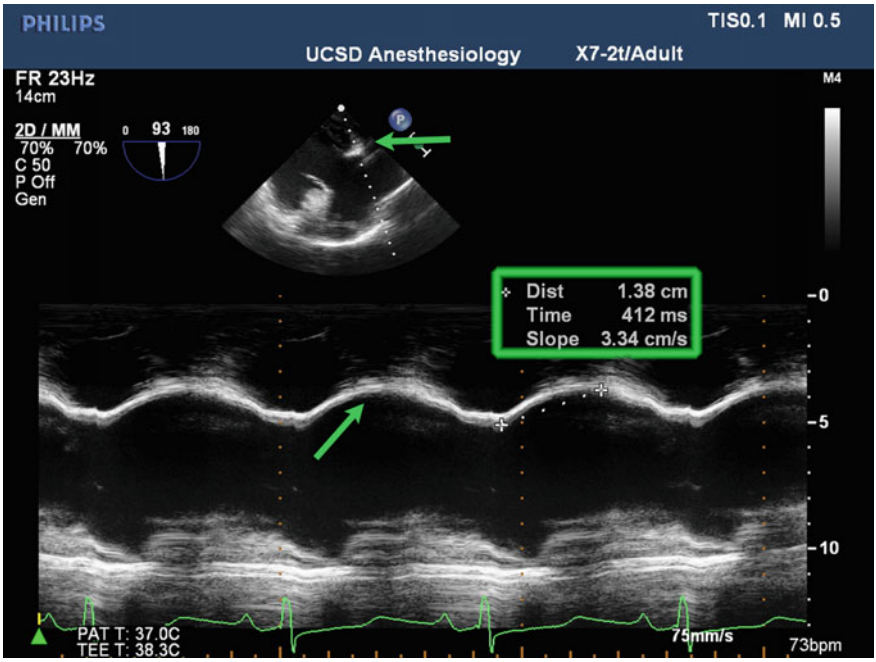

Fig. 8.5 An M-mode analysis of the lateral tricuspid annular motion in a modified transgastric right ventricular inflow view. The *green arrow* on the 2D image depicts the tricuspid annular motion on the M-mode display. TAPSE is easily measured by the movement of the annulus between diastole and systole

TAPSE involves measuring along the RV long axis from the lateral tricuspid annulus to the apex of the RV at end-diastole and end-systole [2] (Fig. 8.4a, b). The difference in measurements constitutes the TAPSE.

Some of the major limitations of TAPSE include oblique imaging of the RV (TV motion is often not aligned to the TEE ultrasound signal) and poor endocardial delineation; both can yield inaccurate measurements. Transthoracic echocardiography, in an apical four-chamber view, allows the alignment of the TV annulus to the echo plane and the use of M-mode to quantify TV motion. The use of a modified TG RV inflow view (deeper insertion of the TEE probe and an increase of the multiplane angle by 10–20°) can provide a less oblique angle of TV motion to the ultrasound probe, allowing the use of M-mode echocardiography (Fig. 8.5).

RV Fractional Area of Change (RV FAC)

RV fractional area of change is analogous to LV fractional area of change (see Chap. 4), and also serves as a surrogate measure of right ventricular ejection fraction (RV EF). TEE assessment of RV FAC is commonly performed in the ME four-chamber view and is the percentage change in ventricular area between systole and diastole with a normal value of >35 %. While a RV FAC <35 % indicates RV dysfunction, values lower than 17 % for RV FAC are suggestive of severe RV dysfunction [18]. Right ventricular 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 IVS. Care must be taken to exclude trabeculations while tracing the RV area (Fig. 8.6a, b). This measurement correlates well with RV EF by cardiac magnetic resonance imaging (MRI) [22] Fractional area change (FAC) is calculated as follows:

$$\text{FAC} = (\text{End Diastolic Area} - \text{End Systolic Area})/\text{End Diastolic Area} \times 100$$

Tissue Doppler Imaging

Tissue Doppler Imaging (TDI) is a mode of Doppler imaging that utilizes a filter that removes high velocity, low intensity signals (i.e., blood) and focuses on low velocity, high intensity signals (i.e., myocardium). When applied to the RV, measurement of the velocity of the basal RV free wall allows a determination of peak systolic velocity (S'). After initiation of TDI, a pulsed wave Doppler beam is placed at the basal RV free wall (near the TV annulus) in either a ME four-chamber view or a modified TG RV inflow view [23]. Attention to alignment of the motion of the myocardium to the ultrasound probe is the key to prevent an underestimation (Fig. 8.7) An RV S' measurement less than 9.5 cm/s is suggestive of abnormal RV function [2]. Peak systolic velocity has been demonstrated to correlate well to RV EF as determined by cardiac MRI [21, 23].

Right Ventricular Myocardial Performance Index (MPI)

The myocardial performance index, also known as the Tei Index, *is a global estimate* of both systolic and diastolic cardiac performance of the RV during both ejecting and nonejecting periods. While not routinely utilized in noncardiac surgery, MPI remains a validated method for assessing RV function. Right ventricular MPI is defined as the ratio of the total isovolumic time (isovolumetric relaxation time and isovolumetric contraction time) divided by ejection time (ET) [24, 25].

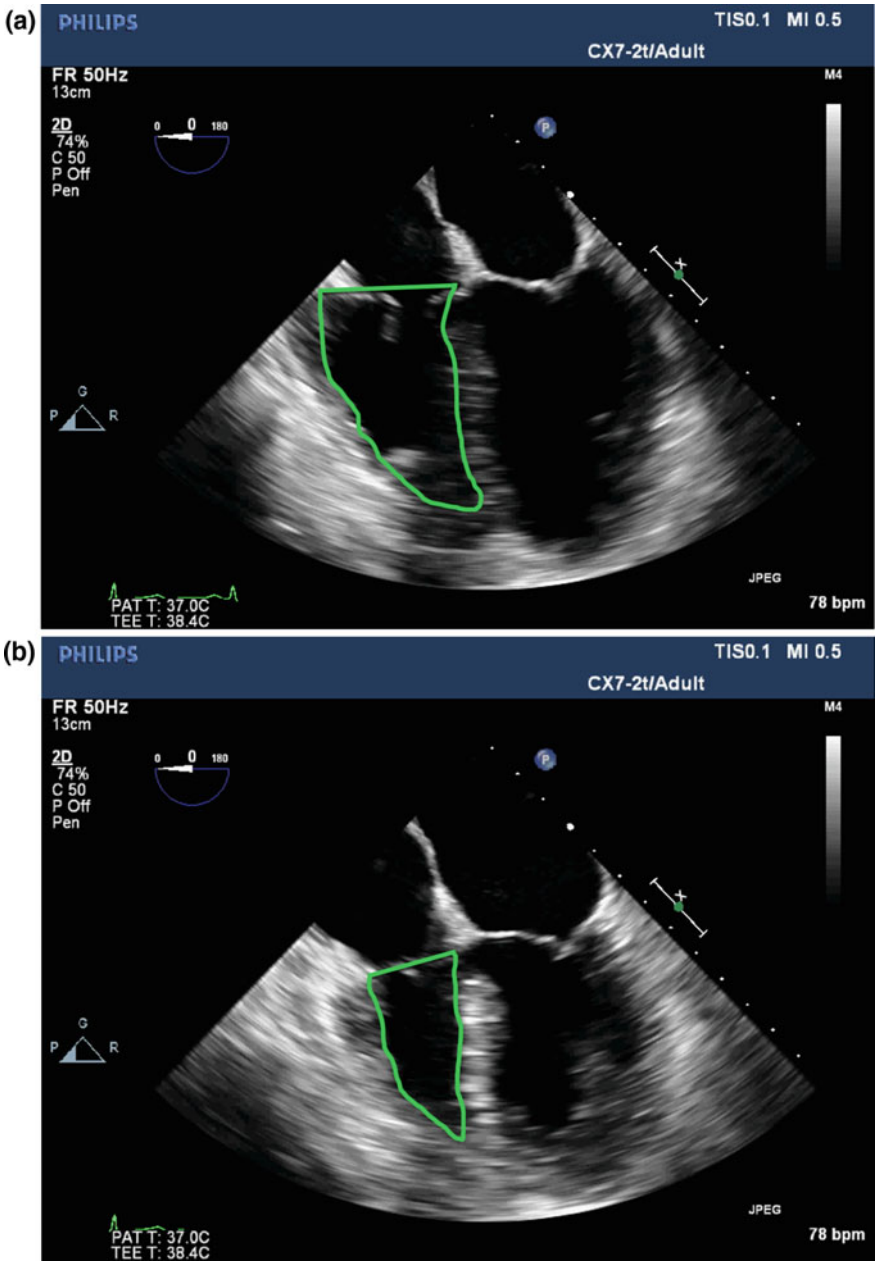

Fig. 8.6 **a** A midesophageal four-chamber view with a *green* tracing outlining the right ventricular area in a diastolic frame. **b** Systolic frame from the same patient with a *green* tracing outlining the right ventricular area. FAC is calculated as (RV Diastolic Area—RV Systolic Area)/ RV Diastolic Area × 100

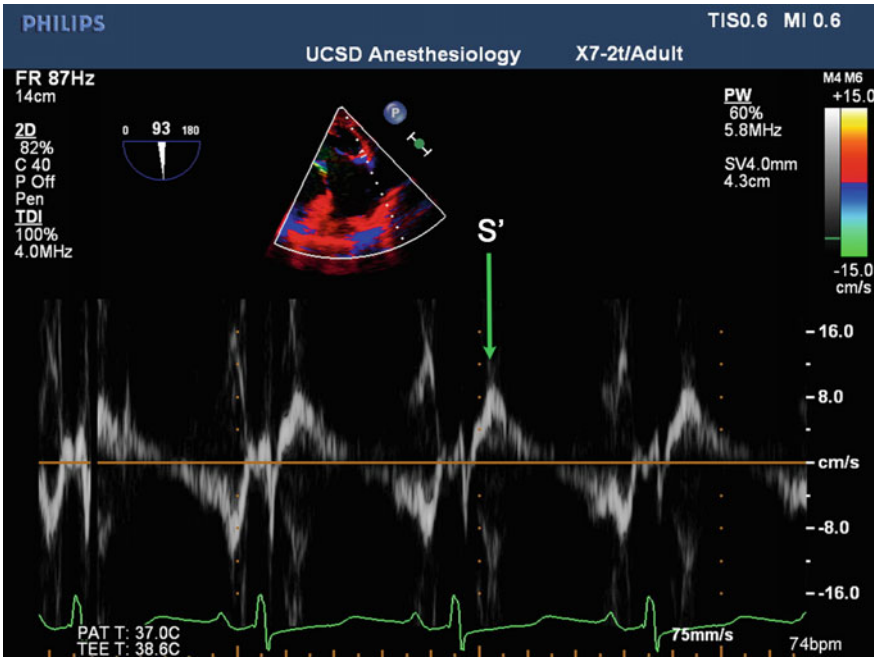

Fig. 8.7 Pulsed wave tissue Doppler imaging of the basal right ventricular myocardium in a modified transgastric right ventricular inflow view. Peak tricuspid annular velocity (S') is identified by the *green arrow*

$$\text{MPI} = (\text{IVRT} + \text{IVCT})/\text{ET}$$

The index has been shown to correlate with symptoms and survival and has a prognostic value in patients with primary pulmonary hypertension [26]. Two methods may be utilized to obtain RV-MPI including one based on pulsed wave Doppler of the transtricuspid flow and pulmonic ejection and another method based on tissue Doppler imaging of the tricuspid annular motion.

Evaluation of the Shape and Motion of the IVS to Assess Global RV Function

Evaluation of RV geometry serves as a qualitative method to assess for global RV dysfunction. In the TG midpapillary short axis view, the RV often appears crescent shaped due to the high LV pressure, causing the IVS to bulge into the lower pressured RV. However, when RV dysfunction ensues, the compensatory RV dilation results in flattening of septum and loss of the natural crescent shape of the RV, yielding a "D-shaped" left ventricular chamber [21] (Fig. 8.8; Video 8.3).

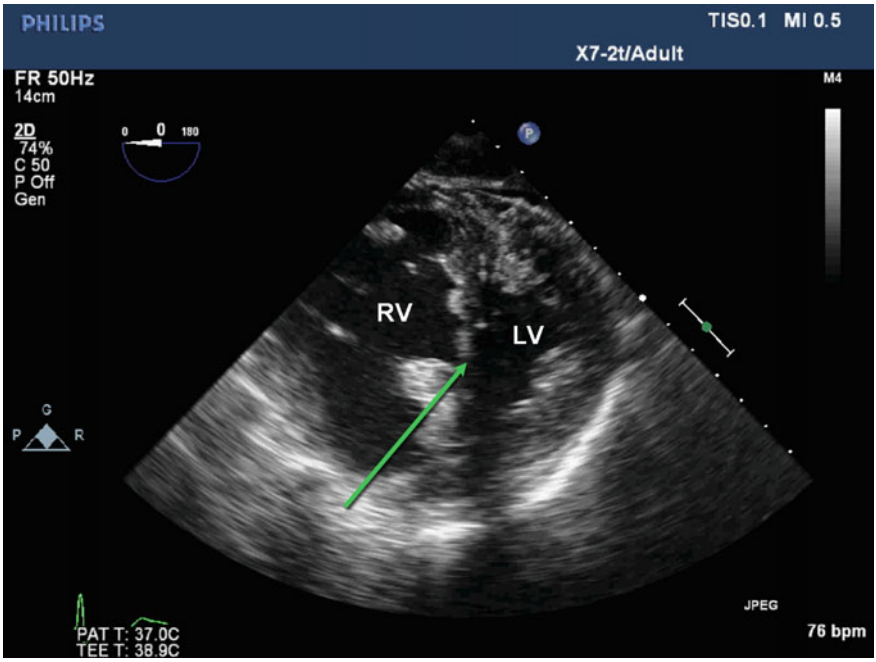

Fig. 8.8 Transgastric midpapillary short axis view in a patient with right ventricular failure. Note the shifted interventricular septum (*green arrow*), which results in a "D-shaped" left ventricle. The presence of the shifted septum at end-diastole indicates right ventricle (RV) volume overload whereas at end-systole indicates RV pressure overload

Although RV volume overload and RV pressure overload may occur concomitantly, they may also occur separately. The timing of the septal flattening can allude to the presence of RV volume overload versus pressure overload. With RV volume overload, the RV volume is largest at end-diastole, which corresponds to the time of peak diastolic overfilling (septal flattening at end-diastole). This is opposed to RV pressure overload where septal flattening occurs at end-systole corresponding to when RV systolic afterload is at its peak (i.e., the time when RV pressure is the highest).

Eccentricity index (EI) is an echocardiographic index for measuring the LV dimensions to differentiate RV volume overload from RV pressure overload [27]. It is the ratio of LV minor axis (anterior-to-inferior) to its perpendicular axis (septal-to-lateral) using the TG midpapillary short axis view. In normal individuals, the LV is round in systole and diastole and the EI has a value of 1. EI value is greater than 1 when the LV is D-shaped during end-systole in pressure overload, whereas in volume overload it is greater than 1 during end-diastole. A high EI is an important echocardiographic predictor of mortality in pulmonary arterial hypertension.

Right Atrium Adaptation to Chronic Right Ventricular Pressure Overload

In chronic right ventricular pressure overload, the right atrium adapts to support active atrial contribution to RV filling, leading to increased atrial distensibility, and functioning as a reservoir to support the cardiac output. These compensatory changes play an important role in maintaining cardiac output in the face of increased RV diastolic stiffness during chronic RV pressure overload [28, 29]. The normal RA upper reference limit is 4.4 cm and 5.3 cm for major (base to TV annulus) and minor (septal-to-lateral) axis dimensions, respectively. With chronic pulmonary hypertension, the RV dilation results in a dilated tricuspid valve annulus with significant tricuspid regurgitation leading to further RA dilatation.

Interatrial Septum Position also serves as an indicator of right heart function. With RV failure, the high right-sided pressure is transmitted to the RA, leading to increasing RA pressure and shifting of the interatrial septum toward the LA. This is usually more apparent in the ME four-chamber, ME RV inflow–outflow, or ME bicaval views as septal bowing toward the left atrium throughout the cardiac cycle can be appreciated (Fig. 8.9; Video 8.4). In patient populations where RA pressures exceed left atrial pressures, there is an increased incidence of patent foramen ovale

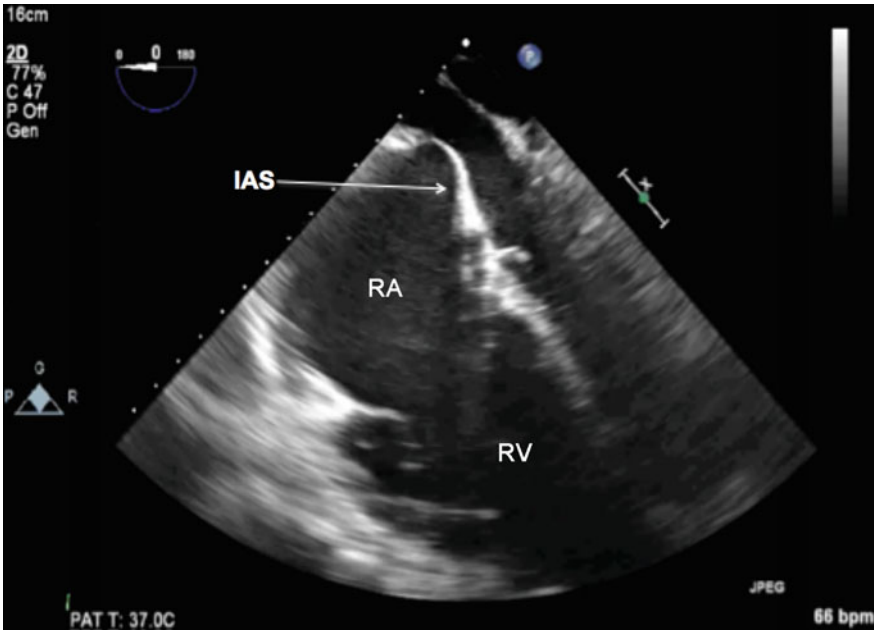

Fig. 8.9 Midesophageal four-chamber view in a patient with significant right atrial (RA) dilation and high right atrial pressure, noted by a shifted interatrial septum (IAS) toward the left atrium. *RV* right ventricle

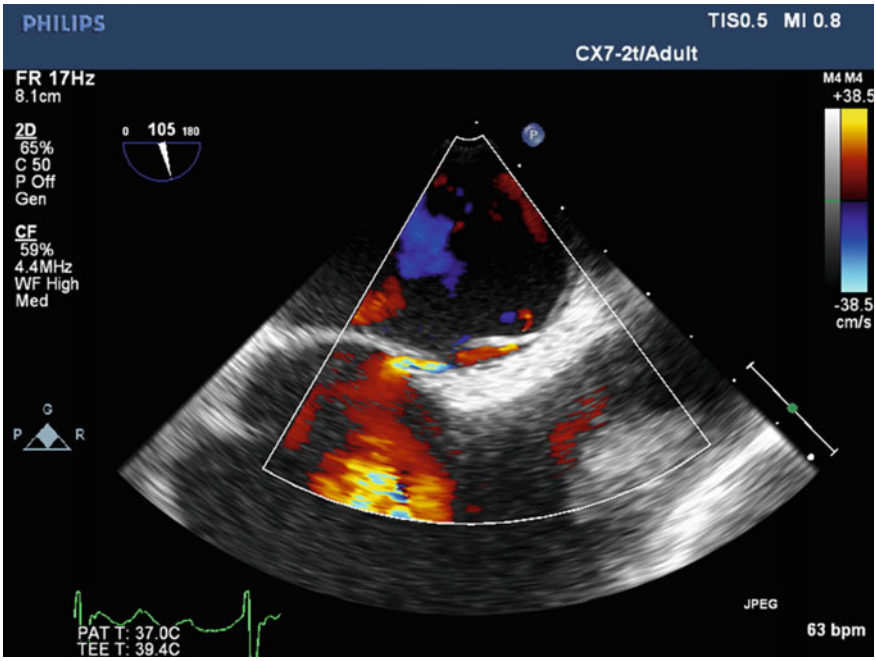

Fig. 8.10 Midesophageal bicaval view in a patient with a patent foramen ovale and right-to-left flow on color flow Doppler

(PFO) compared to the estimated 25 % of the adult population. This can lead to clinically significant right-to-left intracardiac shunting [18]. The shunting may be detected with echocardiography using color flow Doppler or agitated saline, most commonly in the ME bicaval view (Fig. 8.10; Video 8.5).

Tricuspid Regurgitation

For many years, the tricuspid valve (TV) was mostly ignored, and only in recent years has the TV claimed deserved attention. In the majority of patients who have "functional" TR, increased pulmonary and right ventricular pressures lead to RV dilation and subsequent TV annular dilatation and leaflet tenting. The result is worsening regurgitation as the valve leaflets are unable to coapt during systole [30]. In this scenario, the tricuspid leaflets are morphologically without any pathology but do not coapt adequately. With RV systolic failure, the diastolic pressure rises and the interventricular septum shifts toward the LV during diastole, which in turn will raise left ventricular diastolic pressure, further aggravating the TR [31]. Therefore, any patient with a history of PH or evidence of right heart dysfunction

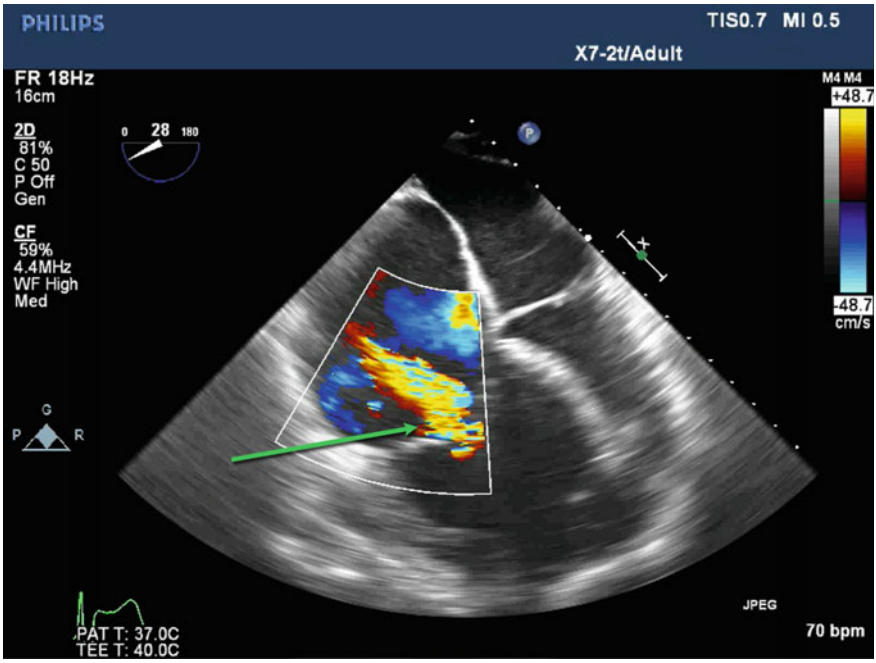

Fig. 8.11 Midesophageal four-chamber view demonstrating severe tricuspid regurgitation (*green arrow*) in a patient with a severely dilated right atrium and right ventricle

deserves an echocardiographic evaluation of the tricuspid valve morphology and evaluation for the causes of TR.

The trileaflet tricuspid valve is composed of the anterior leaflet, which is the largest, the posterior leaflet, and the septal leaflet. Several echocardiographic views are used to visualize the tricuspid valve, including the ME four-chamber, ME RV inflow–outflow, and modified ME bicaval views, as well as TG midpapillary short axis view of the RV and TG RV inflow view. There are several methods to assess the severity of TR; however, the most frequently used modality in the assessment of TR is the vena contracta measurement, with a value greater than >0.7 cm indicative of severe TR (Fig. 8.11; Video 8.6). Additionally, a confirmatory method is pulsed wave Doppler evaluation of the hepatic vein flow pattern. Evaluation of hepatic vein flow when evaluating TR is analogous to evaluating pulmonary vein flow when evaluating mitral regurgitation (MR). Similarly, systolic hepatic flow reversal is specific to severe TR, while blunting of forward systolic flow indicates moderate TR [18] (Fig. 8.12). To image the hepatic veins, perform a rightward probe rotation from the transgastric midpapillary short axis view until the liver is centered; subsequent omniplane rotation may be necessary to obtain a hepatic vein with a parallel orientation to the probe.

The presence of tricuspid regurgitation opens the opportunity to estimate the degree of pulmonary hypertension. As described in Chap. 3, utilizing the simplified

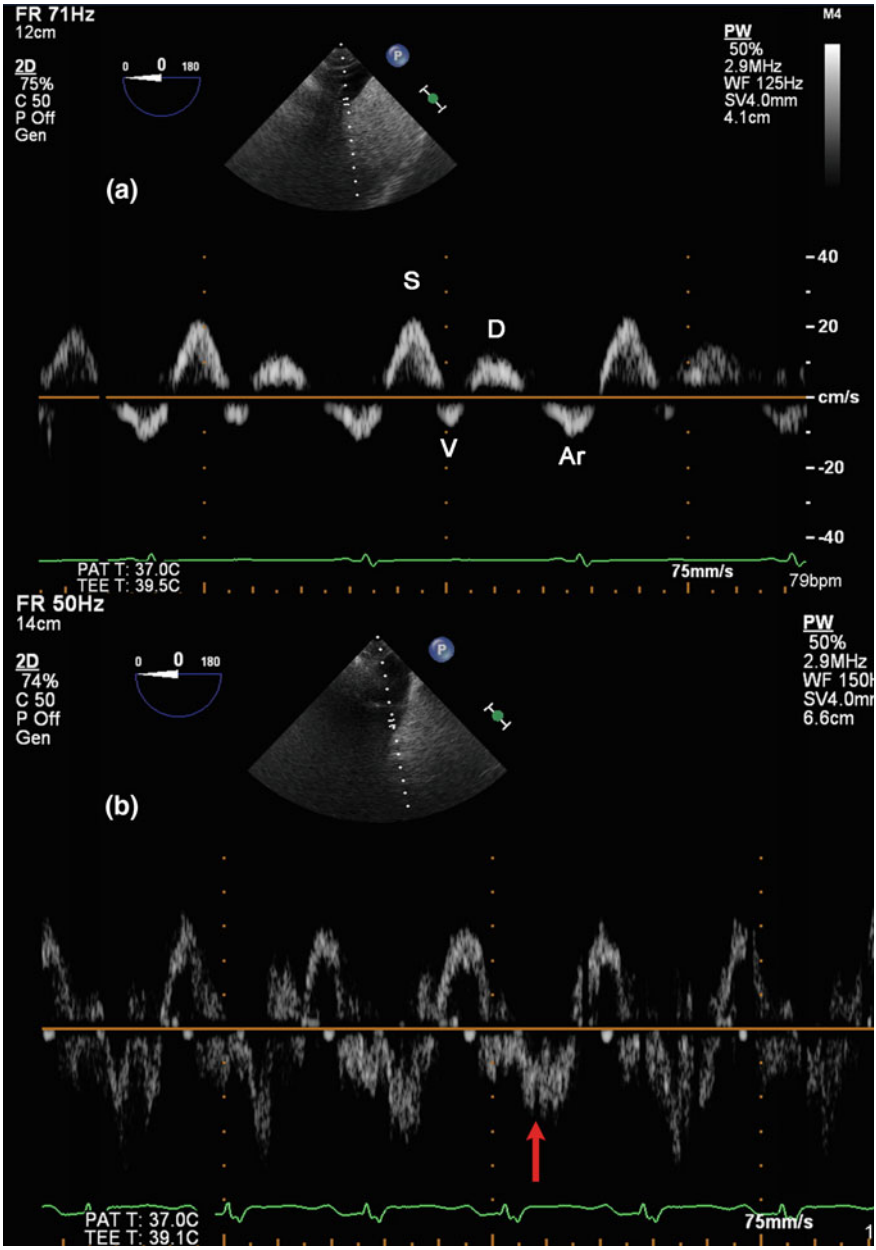

Fig. 8.12 Pulsed wave Doppler of hepatic vein flow. The *top panel* (**a**) demonstrates normal hepatic vein flow with positive systolic (S) and diastolic (D) waves, and brief negative atrial (Ar) wave and variably present V wave. The *bottom panel* (**b**) demonstrates systolic flow reversal confirming severe tricuspid regurgitation

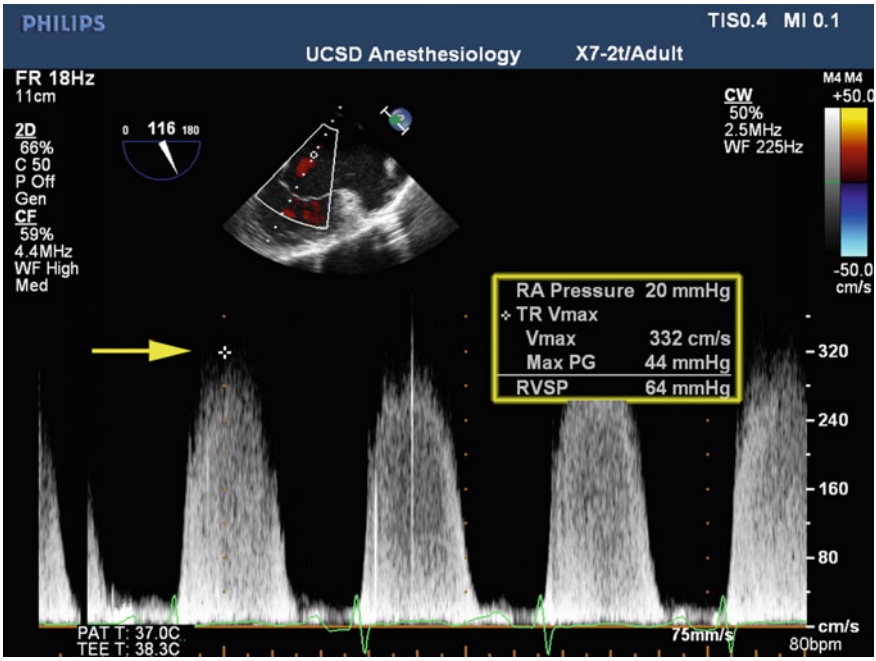

Fig. 8.13 Continuous wave Doppler of the tricuspid regurgitation jet in a modified bicaval view. Applying the modified Bernoulli equation to the peak TR jet (*yellow arrow*) yields an estimation of right ventricular and pulmonary arterial systolic pressure

Bernoulli equation ($\Delta P = 4v^2$) the *pulmonary artery systolic pressure* (*PASP*) can be calculated from the peak velocity of the TR jet using continuous wave Doppler. The peak TR velocity reflects the pressure gradient between the RV and the RA, and when added to central venous pressure provides an estimate of right ventricular systolic pressure and PASP. Commonly utilized views for measurement of PASP are the ME RV inflow–outflow view and ME modified bicaval view (Fig. 8.13).

$$\text{PASP} = 4\left(\text{TR Jet Velocity}^2\right) + \text{estimated RAP}$$

Pulmonary Artery

With long-standing pulmonary hypertension, the chronically increased pressure within the pulmonary artery will cause dilation beyond the normal diameter of 2.1 cm. Commonly utilized views to evaluate the main pulmonary artery (PA) are the ME ascending aortic short axis or UE aortic arch short axis views (Fig. 8.14). A view of the main pulmonary artery allows measurement of RV *cardiac output* (CO) which serves as a load-dependent index of global RV systolic function. The

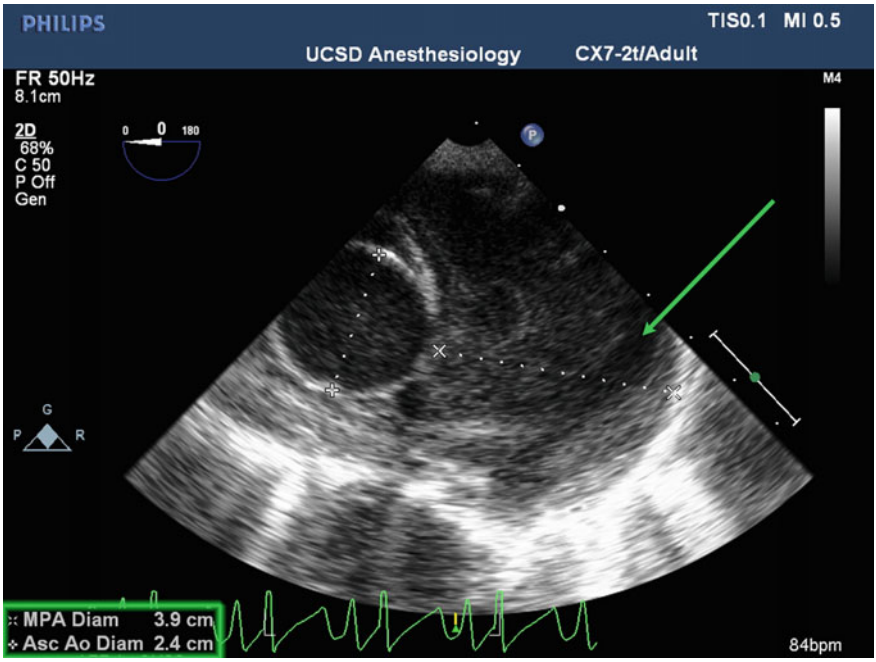

Fig. 8.14 Midesophageal ascending aortic short axis view with slight leftward probe rotation demonstrates significant pulmonary artery dilation (*green arrow*)

right-sided stroke volume can be calculated using pulsed wave Doppler in the ME ascending aortic short axis or UE aortic arch short axis views to obtain the right ventricular outflow tract (RVOT) or PA velocity time integral and the RVOT or PA diameter, respectively [32]. Doppler-derived RV CO provides a method to detect right-sided cardiac output despite the presence of significant TR.

$$\text{RV CO} = \left[0.785 \times (\text{RVOT}_{\text{diameter}})^2 \times \text{VTI}_{\text{RVOT}}\right] \times \text{HR}$$

Echocardiographic Findings in Acute Pulmonary Embolism

Pulmonary embolism (PE) is a potentially fatal condition with mortality ranging from 40 to 80 % within 2 h of onset [33]. An acute PE causes a sudden increase in RV afterload, resulting in RV dilation and dysfunction. The acute pressure increase also displaces the IVS, further contributing to RV failure. Both the decreased right-sided cardiac output and the septal shift lead to underfilling of the LV. Massive PE should be considered with the onset of unexplainable severe and sudden hypoxia or hypotension.

The most common echocardiographic findings in acute pulmonary embolism are RV dilatation and RV dysfunction, with preservation of the motility of the apex in some cases (McConnell's sign) (see Chap. 11). 90 % of patients with a large pulmonary embolism will develop ventricular hypokinesis [34]. Other signs are dilatation of the inferior vena cava with lack of collapse during inspiration, IVS flattening, and paradoxical systolic motion suggesting right ventricular pressure overload, and pulmonary artery dilatation together with tricuspid or pulmonary regurgitation. Patients with an acute PE will have marked prolongation of the isovolumic contraction time and isovolumic relaxation time, resulting in an increased RV-MPI over LV-MPI, which helps to differentiate chronic versus acute pulmonary hypertension of PE. With an acute PE, there is no time for the LV to adapt and therefore, the LV-MPI does not increase [35]. While observing an acutely dilated and dysfunctional right heart is suggestive of a pulmonary embolism, it is not entirely a specific or diagnostic finding. Observation of thromboembolic material in the right heart (thrombus-in-transit), either in the RA, RV, or PA, is a diagnostic finding (Fig. 8.15; Video 8.7). However, the contrary holds true: observation of a normal-sized RA and RV with normal RV function renders the diagnosis of pulmonary embolism unlikely. A thorough echocardiography exam is

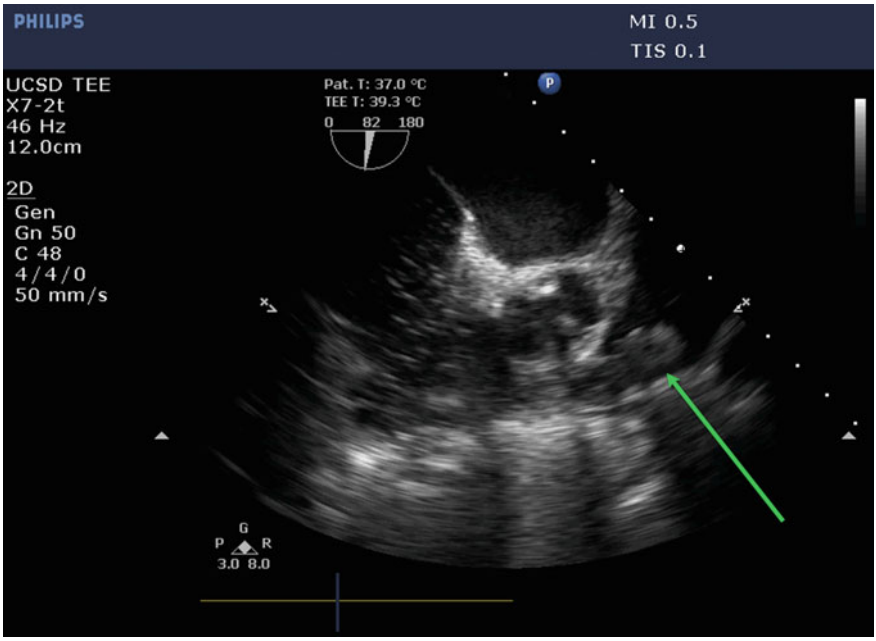

Fig. 8.15 Midesophageal RV inflow–outflow view demonstrating a thrombus-in-transit (*green arrow*) in a patient experiencing a pulmonary embolism

therefore imperative to observe the effects of acutely increased pulmonary afterload as well as to directly visualize embolized thrombi within the right heart chambers and the pulmonary arteries [36].

Conclusion

For many years, the left ventricular physiology overshadowed the study of the right ventricle. Only recently physicians have recognized the importance of right-sided function. With advances in echocardiography, new opportunities have emerged for studying the RV and its effects on patient outcomes. TEE is a helpful tool for the clinician who faces acutely ill patients and can help in the treatment of these patients. In the perioperative setting, echocardiography is able to determine which patient is at highest risk of an adverse outcome by means of evaluation and management of RV failure.

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