REVIEW ARTICLE

Echocardiography and Heart Failure: A Glimpse of the **Right Heart**

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The catastrophic consequences for patients in the settings of certain clinical conditions such as acute right ventricular infarction or massive pulmonary embolism with right heart failure illustrate the essential role that the right ventricle plays in sustaining life. With the development of more sophisticated diagnostic imaging technologies at the end of the last century and the dawn of this century, the importance of the right ventricle has been clearly demonstrated. The continued and evolving nature of our understanding of the right ventricle was emphasized in 2006, when the National Heart, Blood, and Lung Institute formed a working group focused on developing a better understanding of the right ventricle in both healthy and disease states. The objective of this review paper is to examine the right ventricle structure and function and describe the role of echocardiography in the evaluation of the right ventricle and right heart failure. Special focus will be on echocardiographic images and major society guidelines. (Echocardiography 2014;00:1–13)

Key words: heart failure, echocardiography, right ventricular function, right ventricle

The catastrophic consequences for patients in the settings of certain clinical conditions such as acute right ventricular (RV) infarction or massive pulmonary embolism with right heart failure illustrate the essential role that the RV plays in sustaining life. Historically, however, the RV has been dismissed as a less than useful component of the circulatory system and evolution appears to have given the right heart a second-place status, at least initially: the right heart only appears in mammals and birds, while the evolution of most reptiles, amphibians, fish, and invertebrates did not require a RV.¹ During fetal growth, the RV derives from the neural crest cells that travel to the developing heart, rather than originating from the fundamental mesodermal heart tube that the left ventricle and atria arise from. With the development of more sophisticated diagnostic imaging technologies at the end of the last century and the dawn of this century, the importance of the RV has been clearly demonstrated. The continued and evolving nature of our

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understanding of the RV was emphasized in 2006, when the National Heart, Blood, and Lung Institute formed a working group focused on developing a better understanding of the RV in both healthy and disease states.² In addition, the medical literature has developed a significant interest in this area.^{1,2} The objective of this review paper is to examine the RV structure and function and describe the role of echocardiography in the evaluation of the RV.

RV Structure and Function:

Background:

The RV functions as a low-pressure, high-volume pump, as opposed to the left ventricle, which may be defined as a high-pressure and high-volume pump.³ Experiments in the 1940s and 1950s suggested that the RV was an organ of minor physiologic consequence.^{4,5} As diagnostic technologies advanced, an improved understanding developed in regards to RV function in disease states: determining RV systolic and diastolic function is important in the management of many cardiac conditions, including acute decompensated heart failure, chronic heart failure in the setting ventricular dyssynchrony requiring biventricular pacing, chronic heart failure requiring ventricular assist devices (VAD), pulmonary

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hypertension, dysrhythmias requiring permanent pacemakers, cardiac transplantation, and congenital heart disease (especially in the growing adult population with surgical correction in childhood).

Anatomy of the RV:

The anatomy of the RV is distinctly different from that of the left ventricle, although they are functionally interdependent.³ While the shape of the left ventricle may be described as ellipsoid and concentric, the RV may be described as bow- or crescent-shaped. In addition, the overall muscle mass of the RV is only about one-sixth of the left ventricle. However, during the course of one cardiac cycle, the RV usually contains a somewhat greater volume of blood compared to the left ventricle. Also, the RV appears to have an increased distensibility compared to the left ventricle. Nonetheless, these two anatomically different chambers are connected by a complex network of muscle fibers resulting in functional interdependence.

In general, there are two regions of the RV used anatomic description and imaging reporting (in addition to the interventricular septum): the body of the RV (or sinus) and the RV outflow tract (also described as the conus or infundibulum). During the embryonic development of the vertebrae heart, the RV outflow tract arises from the bulbus cordis (a chamber distal to and separate from the common ventricle).⁶ While it appears that the RV body and outflow tract have the same wall thickness, the outflow tract has a functional superiority given its lack of arching or curvature in comparison to the body.

Function of the RV:

The RV functions and pumps in synchrony with the left ventricle, with the RV attached to the high-compliance pulmonary vasculature and the left ventricle connected to the less compliant systemic circulation. The entire heart is enveloped within the pericardium, which itself does not significantly change in size due to acute changes in volume or pressure in any of the four cardiac chambers. This is due to the structural matrix of the pericardium, comprised of collagen and elastin fibers. Therefore, in this relatively "closed" system, acute clinical alterations in volume or pressure in either ventricle can affect the other ventricle. This concept was initially proposed by Bernheim in 1910, who suggested that hypertrophy and dilation of the left ventricle could compress the RV and result in RV dysfunction. Further studies in the 1900s on a feline model of heart failure provided evidence for Bernheim's hypothesis.⁸ Furthermore, observation of human patients with heart failure demonstrated that initial RV dysfunction (such as volume and pressure overload in the setting of atrial septal defects) led to a subsequent left ventricular dysfunction, due to the ventricular septum being forced leftward.⁹ Even in the absence of the pericardium, it appears that the functional co-dependence of the two ventricles persists. In experiments with an isolated beating heart in the absence of a pericardium, two groups demonstrated that load of one ventricle shifted the diastolic pressure-volume relation of the other ventricle, and that a shift of the interventricular septum to the left via RV loading decreased left ventricular chamber dimensions.^{10,11} Clearly, however, the presence of an intact pericardium will affect the co-dependent function of the ventricles, which has been demonstrated in isolated heart preparations in the presence and, by way of comparison, absence of a pericardium.¹²

Right Heart Dysfunction and Ventricular Interdependence:

In the clinical setting, certain disease states have clearly demonstrated the functional interdependence of the two ventricles. In particular, this has been documented in RV myocardial infarction, cardiac transplantation and subsequent acute RV failure, left ventricle mechanical assist device implantation, and acute pulmonary embolus.³ All of these severe deteriorations of cardiac function result in low cardiac output and require critical care unit management. The etiology in each arises from distention of the RV in the setting of a stiff pericardium and resulting decreased left ventricle preload.

In the setting of pulmonary embolism, pulmonary artery and RV volumes are increased, with subsequent decreases in systemic blood pressure and cardiac output. With a large, acute pulmonary embolism, this hemodynamic com-promise can lead to death.¹³ Experimental models of produced pulmonary embolus in canines vielded significant decreases in dimensions of the left ventricle (from the interventricular septum to the left ventricular free wall).¹⁴ Thus, with increased RV pressure in the setting of pulmonary embolus, functional ventricular interdependence results in underfilling of the left ventricle. In the setting of acute RV myocardial infarction, elevated right-sided heart pressures result. One study showed that opening the pericardium helped to increase cardiac output by approximately one-third as well as to increase the left ventricle size.¹⁵ This again demonstrates how the pericardium resists acute changes in ventricular hemodynamics; in particular, increased RV afterload can result in a shift of the interventricular septum to the left with resultant decreased cardiac output.

Some question exists as to which anatomic structure and its functional consequences play the more dominant role in the functional interdependence of the ventricles: the interventricular septum or the restraining effect of the pericardium.³ A human study showed that an increase in the systemic peripheral resistance results in increased afterload of both the right and left ventricles and increased RV volumes, which suggested that pericardial restraint, rather interventricular septum shift, may play a more dominant role in ventricular interdependence.¹⁶

Cellular and Biochemical Implications:

The right and left ventricles also differ in their biochemistry and cellular biology properties. In the porcine heart, the mitochondrial density of the RV is decreased compared to that of the left ventricle.¹⁷ In addition, the mitochondrial to myofibril ratio of the left ventricle and the interventricular septum are similar to each other and both much greater than in the RV. This ratio is a marker for myocardial oxygen consumption and workload, indicating comparatively decreased values in the RV. The decreased oxygen demand of RV myocardial tissue results in a protective effect from ischemia and resultant necrosis due to right coronary artery occlusion. This protective effect can be explained by increased oxygen extraction during stress and a greater systolic to diastolic coronary blood flow ratio as compared to the left ventricle, resulting in increased oxygen delivery during ischemic events.³ A porcine model of right coronary artery occlusion revealed decreased RV necrosis in the absence of previous left-to-right collateral vessel development.¹⁸ In the same animal model, previous RV damage and hypertrophy caused by pulmonary artery banding did not spare the RV from necrosis; in this model both normal and hypertrophic RV had a similar degrees of left-to-right collateral coronary vessels.

Right Ventricular Ejection Fraction:

The RV's inherent protection from ischemia likely plays a role in its importance in predicting mortality and morbidity in heart failure. In those patients with advanced systolic heart failure, a right ventricular ejection fraction (RVEF) less than 40% suggests a greater likelihood of hospitalization and death, while a value below 20% qualifies as an independent predictor of increased risk of hospitalization and death.¹⁹

À normal, healthy RVEF (greater than 40%) requires a synchronized physiologic interaction between the two defined anatomic regions of the RV, the body and outflow tract, and also the interventricular septum. The outflow tract contracts about 30-50 msec after the body of the RV.²⁰ Studies have shown that increased sympathetic tone can eliminate this contraction delay, while increased vagal tone can lengthen the delay.²¹ Experimental models in canines suggest that normal RV ejection relies on active shortening of the free wall surface area in the early phase and septal to free wall distance in the late phase.³ During the late phase, the blood located in the outflow tract flows into the pulmonary artery due to blood momentum.²² Furthermore, the interventricular septum likely plays a significant role in the late phase of RV contraction, as demonstrated in canine studies which showed that RV free wall shortening stops before the free wall to septal dimension reaches maximal excursion.^{3,23}

Impact of the Left Ventricle:

A significant contributor to RV ejection is the left ventricle. This was demonstrated in experiments in canines which demonstrated that significant damages to the RV free wall resulted a minimal decrease in RV function.⁴ Implied from this finding is that left ventricular contraction directly impacts RV systolic function.^{4,24} Additional studies in humans with ventricular dyssynchrony from left or right bundle branch block demonstrated ventricular interdependence via transmission of left ventricle developed pressure to the RV.²⁵ In addition, a laprine animal model demonstrated that increased left ventricular volume resulted in increased RV pressure.¹¹ Further studies in the laprine model showed that coronary artery ligation with resultant left ventricle free wall ischemia yielded a rapid decrease in RV pressures.¹¹ As noted previously, these animal models were not considered physiologic due to the absence of an intact pericardium and loss of diastolic function.³ In the clinical setting, the importance of left ventricle contractile function to RV systolic function has been observed for several decades, and was demonstrated in a small series of patients with postcardiac surgery heart failure requiring vasopressor and intra-aortic balloon pump and then subsequent mechanical VAD placement.²⁶ Preoperative assessment of ventricular function did not predict which patients would require VAD placement; however, those patients with perioperative myocardial infarction predicted those patients who would eventually require VAD placement. Eighty-seven percent of the patients developed biventricular failure, and of these patients, those that received a biventricular VAD had a better chance of being weaned from intensive care unit support and eventually surviving than those patients who received a left-side only VAD. Of note, those patients with left-sided only ventricular failure did well with left-side only VAD.

Echocardiographic Imaging of the Right Heart:

Background:

The widespread use of echocardiography in diagnostic medicine continues to develop with technological improvements and the advancement of clinical knowledge. An echocardiogram is often the first test of choice in patients who present with dyspnea. The analysis of the RV has posed a definite challenge, although the aforementioned increased appreciation of RV dysfunction on morbidity and mortality has created an increased need for improved diagnostic methods for evaluating the RV. The development of two-dimensional echocardiography (2DE) allowed for improved evaluation of RV size and function. Standardized approaches to RV evaluation have made it easier for routine evaluation and comparison of RV function over time.²⁷ Given its availability, portability, relatively low cost (compared to other imaging modalities), and lack of risk or radiation exposure, transthoracic echocardiogram remains the test of choice for initial evaluation of suspected right heart dysfunction.

Other Imaging Modalities:

A brief review of other imaging modalities will aid to understand the advantages (and disadvantages) of echocardiography in the evaluation of the right heart. Chest x-ray is often used as an initial step for patients with suspected right heart disease as it is widely available and relatively inexpensive. The RV is best viewed in the lateral view, with RV enlargement noted when the cardiac silhouette involves more than 40% of the lower retrosternal space. Enlargement of the RV can cause the heart to rotate in a posterior direction and also can push the RV outflow tract in a lateral direction. Of course, a routine chest x-ray can also visualize other disease processes, including acute infection processes, pulmonary edema, intrinsic pulmonary disease, enlargement of the right atria, and left ventricular enlargement. If any cardiac abnormality is noted on a routine chest x-ray, the next diagnostic step is most often the echocardiogram.

Nuclear imaging plays a role in the evaluation of RV function, particularly in the assessment of RV volumes and function and also myocardial characterization. First-pass radionuclide ventriculography, with bolus injection of technetium-99m with RV counts of at least 40%, allows for one imaging plane and is the test of choice for nuclear evaluation of RV evaluation. Also, gated equilibrium blood pool imaging can be used. Due to the advantages of other imaging modalities relative to these techniques, nuclear scans are not routinely used for the evaluation of RV function and volume. Positron emission tomography has been applied to characterize RV myocardial metabolism with fluorodeoxyglucose-18F (FDG) in patients with known left heart dysfunction.²⁸ A negative correlation exists between RVEF and the RV-to-left ventricle FDG ratio.^{28,29} This imaging is best obtained in the setting of RV hypertrophy and in particular with pulmonary hypertension, and can allow for evaluation of RV ischemia.³⁰

Cardiac catheterization with cine angiograms of the RV can obtain several views of the RV, although no more than two are usually performed and recorded due to a desire to limit iodinated contrast dye. With these projections, RV volume and function can be quantitated using Simpson's rule³¹ or other approaches.²⁸ The advantage of invasive diagnostic right heart catheterization is that other valuable testing can be done at that time, including recording of a variety of right heart and pulmonary hemodynamic measurements and also pulmonary angiography, if indicated. Given other imaging techniques available and the risk of an invasive procedure with significant dye load, RV angiography is not often used in routine evaluation of the RV size and function.

Computed tomography (CT scans) are widely used for the evaluation of pulmonary embolus, and nongated CT scans have been used in the setting of acute pulmonary embolus to examine the RV as a predictor of thirty-day mortality.³ Other quantified measures, including threedimensional (3D) imaging and interventricular septal displacement, have been developed as mortality predictors in this clinical setting.²⁸ Helical scans of the heart with electrocardiographic gating has been shown to provide accurate RV function and volume quantification.³³ However, the need for radiation and contrast (compared to other modalities which require neither) has limited the use of CT scanning in this setting; most often CT is used in patients with suspected right heart dysfunction or congenital heart disease with a permanent device (such as mechanical VAD or permanent pacemaker).²⁸

Cardiac magnetic resonance imaging (CMR) has recently developed as a valuable tool in the evaluation of the RV. CMR is currently considered the "gold standard" in the assessment of RV volumes and ejection fraction.³⁴ The ability to obtain unlimited, highly reproducible images in high resolution without radiation exposure, without contrast administration requirement (in the setting of function assessment only), and without the acoustic limitations of ultrasound has made it one of the diagnostic tests of choice in the evaluation of patients with suspected right heart dysfunction.²⁸ CMR is more expensive, less broadly

available, and more time-consuming compared to echocardiography, however, and patients with medical devices (see CT section above) are unable to undergo scanning due to the magnetic field. CMR is often applied to the evaluation of patients with pulmonary hypertension due to the ability to quantify RV hypertrophy, enlargement, and systolic function.²⁸ In adult patients with complex congenital heart disease with involvement of the RV (such as tetralogy of Fallot or transposition of the great arteries), CMR is the test of choice as other imaging modalities are more limited in this setting.³⁵ ČMR is also well suited for the evaluation of suspected arrhythmogenic cardiomyopathy, given its ability to evaluate RV dysfunction and enlargement and to detect (with use of a contrast agent) fatty deposits and/or fibrosis in the myocardium.³⁶

Echocardiographic Methods in RV Evalution:

Despite the various advantages of the other imaging modalities detailed above, ultrasound of the heart is still the most common diagnostic modality used in the evaluation of patients with suspected or known right heart dysfunction, with a significant amount of information available from standard echocardiographic techniques, as detailed in Table I.²⁸ Current clinical guidelines endorse the use of echocardiography in this setting.³⁷ Advantages of echocardiography include low comparative cost, widespread availability, portability (currently handheld echocardiograms are being developed for widespread application),

TABLE I

Echocardiographic Techniques to Assess the Right Ventricle (RV)

M-mode
RV wall thickness
RV outflow tract shortening
Tricuspid annular plane systolic excursion (TAPSE)
(Fig. 1)
2D echo (Fig. 2)
Linear dimensions
Visual assessment of RV volumes/ejection fraction
Ventricular eccentricity index
Fractional area change
Conventional Doppler
RV systolic pressures (Fig. 3)
Myocardial performance index
Doppler tissue imaging
Myocardial performance index
Isovolumic acceleration (IVA)
Strain and strain rate
Speckle tracking
Strain and strain rate
3D echo (Figs. 4 and 5)
RV volumes
RV ejection fraction



Figure 1. A. Normal TAPSE: M-mode and apical four-chamber view measurement of systolic tricuspid annular plane excursion which represents the systolic right ventricular (RV) function (normal >1.6 cm). **B.** Abnormal TAPSE: M-mode and apical four-chamber view measurement of systolic tricuspid annular plane excursion which represents the systolic RV function (abnormal <1.6 cm).

and safety (no radiation exposure and newer contrast agents, although not required for an exam, appear nontoxic and well tolerated). As echocardiography has been used for decades, a significant amount of clinical trial data and evidence-based medicine in the management of heart failure and heart disease in general are founded on echocardiography data. Also, the use of Doppler allows for the evaluation of certain hemodynamic parameters. Limitations exist specifically in the setting of echocardiographic evaluation of the RV: the complex shape of the chamber does not allow for the use of geometric models for volume and function quantification, the retrosternal position of the RV creates a barrier for ultrasound waves, and the high-volume, low-pressure system of the RV with its thinner walls and prominent trabeculations (in comparison to the left ventricle) confound data analysis.²⁸



Figure 2. A. Normal right ventricular dimensions. **B.** Abnormal right ventricular dimensions. **C.** Right ventricular (RV) dimensions in a patient with pulmonary embolism. **D.** RV pressure overload causing flattening of septum or "D-sign." **E.** RV pressure overload causing flattening of septum or "D-sign." **i** left panel (right panel normal and middle panel suggest slightly increased pressure). **F.** "D-sign" with enlarged RV cavity seen.

RV Volume and Morphology:

RV size and morphology are assessed via echocardiography via 2D imaging.³⁸ RV pressure overload can be evaluated with standardized image acquisition and may reveal RV hypertrophy, dilatation, and abnormal interventricular septum motion.²⁸ Hypertrophy is characterized by an end-diastolic free wall thickness of greater than 5 mm and correlates with increased afterload.³⁹ With chronic elevated pressure, the RV will dilate

and function will become impaired. Given the curved shape and complicated structure of the RV, volumes are difficult to evaluate in an objective manner, and the 2D approaches used routinely and accurately in left ventricle that employ Simpson's formula are often imprecise. Measurements of transverse and longitudinal lengths are used instead, along with subjective comparison of relative size of the RV versus the left ventricle [Table II].²⁸ Common practice dictates that a RV diastolic area that visually appears to be equal to or greater than that of the left ventricle (in a standard four-chamber image) indicates RV enlargement (Fig. 2).⁴⁰

An important marker of RV pressure overload is flattening of the interventricular septum (Fig. 2D,E) with motion of the septum toward the left ventricle during end-systole. In addition, volume overload of the RV can also cause flattening of the septum (Fig. 2F) and movement toward the left ventricle, in this case during enddiastole.²⁸ As noted previously, this septal motion causes under filling of the left ventricle and decreased stroke volume, and can cause critical illness in certain settings. The ratio of the left ventricle anteroposterior to septal-lateral short-axis cavity dimension at end-systole or end-diastole is termed the left ventricle eccentricity index, and values above one indicate RV overload. 28,40 Furthermore, the relation of septal curvature to left ventricle free wall curvature as well as the degree of septal curvature can be employed to estimate RV systolic pressures and trans-septal pressure gradients.^{28,41}

RV Function:

Several techniques are used to measure RV systolic function with several emerging techniques in continued development, as detailed in Table I.⁴¹ Routine practice involves the use of visual estimation of function from multiple imag-



Figure 3. Enlarged right atrium on 3D echocardiography.



Figure 4. A. Doppler of tricuspid regurgitant jet estimating peak right ventricular systolic pressure as high as ~51 mmHg. **B.** Doppler of tricuspid regurgitant jet estimating peak right ventricular systolic pressure at ~39 mmHg. **C.** Doppler of tricuspid regurgitant jet estimating peak right ventricular systolic pressure at ~31 mmHg

ing planes. Other more objective techniques will be discussed below.

RV fractional area change is the percent change in ventricle chamber area in the apical four-chamber view throughout the course of the cardiac cycle; it correlates well with CMR data



Figure 5. A. Speckle tracking of RV. **B.** Speckle tracking of RV. RV = Right ventricle.

and can predict death, heart failure, and stroke after myocardial infarction. $^{\rm 41-43}$

RV index of myocardial performance (RIMP) is a calculation used to evaluate RV systolic and diastolic function; it is defined as a ratio of total RV isovolumic time divided by RV ejection time.⁴⁴ The RIMP value can be correlated with RVEF using nuclear ventriculography and appears to be less affected by heart rate and loading conditions.^{28,44,45} RIMP values greater than 0.55 by tissue Doppler and 0.40 by pulsed-wave Doppler signify ventricular dysfunction.⁴⁰

Tricuspid annular plane systolic excursion (TAPSE) uses M-mode apical four-chamber views to evaluate longitudinal RV function, with a value less than 1.6 cm suggestive of RV dysfunction. It is predictive of mortality in advanced systolic heart failure⁴⁶ and pulmonary hypertension.^{28,40,47} Correlation with other standardized imaging modalities is under continued investigation, although initial studies suggested close correlation with radionuclide angiography.^{42,48} Drawbacks of TAPSE are that only one-dimensional (1D) images representing the basal RV are used,²⁸ however, it is currently recommended for routine use in the evaluation of RV function.⁴⁰

Pulsed Doppler tissue imaging (DTI) objectively measures peak velocities with a tricuspid lateral annular systolic velocity value below 9.7 cm/sec indicating abnormal contractility and possible early RV dysfunction,^{28,34,49} with this

TABLE II								
Normal Adult Right Ventricular (RV) Echocardiographic Dimensions (See Fig. 2)								
Dimension	RV Length (Base-to-Apex)	Basal RV	Mid RV	RV Outflow Tract	Pulmonary Artery			
Normal range, cm	7.1–7.9	2.0–2.8	2.7–3.3	2.5–2.9	1.5–2.1			

TABLE III											
Measures of Right Ventricular (RV) Function											
Method	RVEF on 2D	Fractional Area Change (FAC)	TAPSE (Fig. 1)	RIMP	Isovolumic Acceleration (IVA)	S' (Pulsed) (Fig. 6)	RVEF on 3D (Fig. 4)				
Abnormal	<44%	<35%	<1.6 cm	Pulsed>0.44 Tissue>0.55	Not defined	<10 cm/sec	<44%				
Limitations	Unreliable	Endocardial border needs to be accurately traced	Load and angle dependent	Not reliable in elevated RA pressures or irregular heart rates like atrial fib	Load and angle dependent; varies with age and heart rate	Angle dependent, assumes all segments have similar function; limited data in elderly	Not readily available				

RVEF, right ventricular ejection fraction.







Figure 6. A. Determining RVEF with 3D echo. **B.** Normal RVEF on 3D echocardiography (normal >44%). **C.** Abnormal RVEF on 3D echocardiography (abnormal <44%). RVEF, right ventricular ejection fraction.

measure correlating well with radionuclide imaging assessment of RVEF.⁵⁰ Color tissue Doppler has been utilized to obtain S' (Table III and Fig. 7) as a measure of RV function.

Increasingly, evaluation of strain and strain rate of the RV are being used to assist in the evaluation of RV function and can be obtained with DTI.^{28,51} Strain measures myocardial tissue deformation, with strain rate measuring this



Figure 7. A. Color tissue Doppler demonstrating normal S' (>10 cm/sec). **B.** Color tissue Doppler demonstrating abnormal S' (<10 cm/sec). **C.** Abnormal S' in a patient with acute PE.

deformation over time. In routine practice, stain is only measured in the longitudinal, apical four-chamber view. Speckle tracking (Fig. 5) measures motion in the myocardium by following speckle location and tracking them with continued image acquisition via a complex algorithm, with derivation of strain values.²⁸ Advantages include decreased dependence on 2D image quality, angle, and frame rate and

TABLE IV

Inferior Vena Cava Diameter to Assess Volume Status

Inferior vena cava (IVC) normally collapses more than 50% with inspiration or sniffing: Collapse <50% suggests volume overload

- Correlation between RA pressure (CVP) and IVC appearance:
 - 1 CVP 0–5 cm: IVC totally collapses on inspiration and is <1.5 cm in diameter
 - 2 CVP 5–10 cm: IVC collapses >50% on inspiration and is 1.5–2.5 cm in diameter
 - 3 CVP 11–15 cm: IVC collapses <50% on inspiration and is 1.5–2.5 cm in diameter
 - 4 CVP 16–20 cm: IVC collapses <50% on inspiration and is >2.5 cm in diameter
 - 5 CVP >20 cm: No change in IVC on inspiration and is >2.5 cm in diameter

measurement of RV strain in both the short and long axis. Clinical studies confirm correlation with both global and regional RV function.^{28,52}

The measurement of RV diastolic dysfunction is under continued development. Pulsed wave Doppler measurement of tricuspid inflow and myocardial DTI provides one method of diastolic analysis and yields objective data for E/A, E/E', and E'/A' ratios. Also, measurement of the isovolumic relaxation time can provide information regarding RV diastolic dysfunction.^{28,43,53}

Advanced Echocardiographic Analysis of the RV:

Evaluation of RV hemodynamics can be obtained via several methods, including objective analysis of RV systolic pressures via application of the modified Bernoulli equation to the tricuspid regurgitant jet. Other modalities include Doppler imaging of the RV outflow tract and estimation of right atrial pressure via measurement of inferior vena cava diameter and inferior vena cava change with respiratory variation (Table IV and Fig. 8).^{28,40,54}

The development of 3D echocardiography has occurred as a result of improved technology in the setting of advanced transducers, improved image analysis, and high-level software development. Using full-volume image acquisition, the entire RV may be visualized (Fig. 6) and does not require geometric assumptions to measure cardiac chambers, which is often a problem with traditional methods and the complex structure of the RV.²⁸ Real time 3D techniques have been shown to accurately provide objective measurement of RV volumes.⁵⁵



Figure 8. A. Normal inferior vena cava diameter. B. Dilated inferior vena cava.

Practical Assessment of the RV:

Heart Failure:

The first step is to evaluate pulmonary artery pressure (PAP): several studies have established the prognostic value of pulmonary hypertension in cohorts of ambulatory patients with heart failure.⁵⁶ The cutoffs used were quite different and somewhat arbitrary as there is no definition of pulmonary hypertension based on the systolic pulmonary pressure estimated at echocardiography. Guidelines in fact recommend that pulmonary hypertension is diagnosed on the basis of mean PAP at right heart catheterization. Pulmonary regurgitation velocity can be used to assess pulmonary artery diastolic pressure; however, caution is required as it is usually difficult to correctly align the ultrasound beam with the pulmonary regurgitant jet to obtain accurate values. Mathematical formulae have been published which allow for the calculation of pulmonary vascular resistance; however, this measurement is necessary only when patients have to be screened for heart transplant and in this situation a precise hemodynamic determination must be obtained.

The simplest echocardiographic indicator of RV function, i.e. the TAPSE has proved to be a robust prognostic indicator in heart failure; a TAPSE \leq 14 mm is associated with a high risk of death or hospitalization or urgent heart transplantation during follow-up.⁴⁶ Importantly, risk stratification in heart failure may be greatly enhanced if TAPSE and systolic PAP are considered together.⁵⁷

To summarize, pulmonary artery systolic pressure and TAPSE represent the minimum dataset in heart failure patients.

Pulmonary Hypertension:

In patients with pulmonary hypertension, survival and progression of the disease are strongly dependent on the ability of the RV to adapt to the chronically elevated PAP. Although several echocardiographic variables have been associated with outcome, in recent years the simple TAPSE has drawn the attention of researchers.47,58,59 Importantly, TAPSE is only the first step of a pathophysiological approach to the RV function with echocardiography. Patients with normal or relatively preserved TAPSE can be further stratified according to the degree of tricuspid regurgitation, since TAPSE might be overestimated in the presence of significant tricuspid regurgitation. Patients having a reduced TAPSE can be further stratified according to the left ventricular eccentricity index in diastole, since this is a good indicator of RV diastolic pressures.47

Finally, the visual inspection of the shape of the Doppler flow velocity envelope of the RV outflow tract may provide useful insight into the hemodynamic basis of pulmonary hypertension: the presence of mid-systolic notching is associated with the most severe pulmonary vascular disease and right heart dysfunction.⁶⁰

In summary, TAPSE, the degree of tricuspid regurgitation, the end-systolic left ventricular eccentricity index, and the envelope of the RV outflow tract at pulsed Doppler represent the minimum dataset in pulmonary hypertension patients.

Guidelines from Major Societies:

The American College of Cardiology and American Heart Association 2013 Guideline for the Management of Heart Failure recommends 2D echocardiogram with Doppler for the initial evaluation of heart failure.⁶¹ In addition, they recommend repeat measurements of ejection fraction in patients with heart failure who have had a significant change in clinical status or received treatment that might affect cardiac function or for consideration of device therapy. Both of these are I Class of Recommendations with C Level of Evidence. Specifically, they state that RV size and function should be measured and that all valves, including the tricuspid and pulmonic valves, should be evaluated for anatomic and flow abnormalities. In addition, secondary changes (in particular the severity of tricuspid valve insufficiency) should be determined. The tricuspid valve regurgitant gradient, measurement of inferior vena cava diameter, and the inferior vena cava diameter response during respiration provides estimates of PAP and central venous pressure, and therefore give insight to right heart function during heart failure.

The Heart Failure Association of the European Society of Cardiology 2012 guidelines for the diagnosis and treatment of acute and chronic heart failure recommend transthoracic echocardiogram, along with electrocardiogram, as the two most useful tests in patients with suspected heart failure.⁶² The data obtained from the echocardiogram provide measurements of chamber volumes, wall thickness, valve function, and systolic and diastolic function, which are crucial in deciding appropriate therapies. Specifically, the echocardiogram can provide data in regards to RV function and PAP, in addition to left ventricular function. Isolated RV failure may occur in the setting of acute coronary syndrome or after massive pulmonary embolism and treatment approaches contrast those of treatment for left ventricular dysfunction. Specifically, vasodilators and diuretics should be avoided or used cautiously, as they may cause reduction in RV filling, in the setting of isolated right heart failure. Furthermore, progressive isolated RV failure may occur in patients with pulmonary hypertension, and specific agents are indicated in pulmonary hypertension that are not part of the standard therapy for left ventricular dysfunction. Medications which help pulmonary arterial decrease to resistance include type V phosphodiesterase inhibitors, endothelin antagonists, and prostacyclin analoques.

Conclusions:

Accurate determination of RV function is important in the management and prognostication of heart failure. Echocardiography has emerged as important modality in assessment of RV function and the suspicion that RV function is impaired should be considered when there is reduced (TAPSE <16 mm), increased tricuspid regurgitation peak velocity (when>3.4 m/sec), increased systolic PAP (increased when >50 mmHg), or dilated inferior vena cava (>2.5 cm).

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