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REVIEW

Non-invasive investigations of the right heart: How and why?

Explorations non invasives du cœur droit : comment et pour qui?

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MOTS CLÉS

Ventricule droit ;
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Summary The importance of right ventricular (RV) function in the clinical management of patients with cardiopulmonary disorders is now well recognized. However, due to both its shape and location and to the load dependence of its ejection fraction, accurate evaluation of its function is still a challenge. Echocardiography allows morphological, hemodynamic and functional assessment of the right heart. Displacement and deformation parameters derived from new techniques are promising tools. 3D echocardiography also has a potential interest in the quantification of RV volumes and ejection fraction. Radionuclide technique allows an easy and accurate measurement of right ventricular ejection fraction. MRI remains nowadays the technique of choice for the quantification of volumes and function of the RV. All these techniques have proven their interest in various diseases affecting the right heart. RV function is an important prognostic factor in heart failure and is a major component of functional capacity in such patients. In pulmonary arterial hypertension, echocardiography is the best tool for the routine follow-up of patients. Finally, all these non-invasive techniques of investigation of the right heart enable the diagnosis of specific right ventricular damage such as myocardial infarction or arrhythmogenic right ventricular dysplasia.

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Résumé L'étude de la fonction ventriculaire droite est un des éléments importants de l'évaluation clinique et pronostique des patients atteints de différentes pathologies cardiopulmonaires. Néanmoins, la forme du ventricule droit, sa localisation et le fait que sa fraction d'éjection soit très dépendante des conditions de charge, font que l'appréciation fine de sa fonction représente toujours un défi pour le clinicien. L'échocardiographie permet à la fois une

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myocarde ;
Dysplasie
arythmogène du
ventricule droit

évaluation morphologique, hémodynamique et fonctionnelle du cœur droit. Les paramètres de déplacement et de déformation myocardique dérivés des nouvelles techniques paraissent prometteurs et l'échocardiographie tridimensionnelle permet potentiellement de quantifier les volumes et la fraction d'éjection ventriculaire droite. Actuellement, les techniques isotopiques donnent accès facilement à une mesure de la fraction d'éjection du ventricule droit et l'IRM est considérée comme la technique de référence pour la quantification de ses volumes. Toutes ces techniques ont montré leur intérêt dans différentes pathologies. Ainsi, la fonction ventriculaire droite est un facteur pronostique très important dans l'insuffisance cardiaque, et est en outre, très liée à la capacité fonctionnelle de ces patients. Dans l'hypertension artérielle pulmonaire, l'échocardiographie permet le suivi et aide à la prise en charge thérapeutique des patients. Enfin, ces techniques non invasives permettent le diagnostic d'atteintes plus spécifiques du ventricule droit telles que nécrose myocardique et dysplasie arythmogène.

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Abbreviations

2D	two-dimensional
3D	three-dimensional
ARVD	arrhythmogenic right ventricular dysplasia
DTI	Doppler tissue imaging
IVRT	isovolumic relaxation time
LV	left ventricular
MRI	magnetic resonance imaging
PAP	pulmonary artery pressure
PVR	pulmonary vascular resistance
RAP	right atrial pressure
RV	right ventricular
RVEF	right ventricular ejection fraction
RV FAC	right ventricular fractional area change
RVOT	right ventricular outflow tract
TAPSE	tricuspid annular plane systolic excursion

Background

For many years, the study of LV morphology and function overshadowed analysis of the right ventricle. Non-invasive techniques, such as echocardiography, which were of great benefit when studying the left ventricle, were hampered by the complex shape of the right ventricle and by its poor echocardiographic visualization due to its anterior retrosternal intrathoracic position. As a result, most right heart analysis was done invasively during catheterization.

However, recognition of the importance of RV function in the clinical management of patients with cardiopulmonary disorders led to a new interest in the right ventricle. In addition, improvements in echocardiographic 2D and 3D imaging, the development of new techniques allowing the study of myocardial displacement and deformation, and the advent of MRI progressively allowed the right heart to be approached more readily.

The goal of our review is to offer a clinical perspective on non-invasive right heart functional evaluation and to underline the importance of the right heart in some specific heart diseases.

Reminders about the right heart

Anatomy

The right ventricle in the normal heart is the most anterior cardiac chamber and is located immediately behind the sternum. The cavity has a complex shape: quite triangular in long-axis views and crescent (curving around the left ventricle) in small-axis views [1].

The right ventricle is now described as a three-component cavity:

- the inlet portion with the tricuspid valvular apparatus (tricuspid valve, chordae tendineae and papillary muscles);
- the trabeculated apical myocardium;
- the outlet portion with the infundibulum or conus, which corresponds to the smooth myocardial outflow region [2].

The thin wall of the right ventricle (only 3–5 mm thick) is composed mainly of circumferential fibres in the superficial layer and longitudinal muscle fibres in the subendocardium. The continuity between the muscle fibres of the right and left ventricles binds the ventricles together functionally, contributing to ventricular interdependence.

Physiology

The performance of the right ventricle depends on intrinsic (contractility) and extrinsic factors such as preload and afterload and ventricular interdependence.

RV contraction is the result of three major components:

- inward displacement of the RV free wall toward the septum;
- shortening of the long axis with descent of the tricuspid annulus towards the apex;
- free RV wall traction caused by septal movement towards the left ventricle during systole [3].

This contraction follows a "peristaltic" pattern and the outflow tract contracts 25–50 ms after the inflow.

The right and left ventricles operate in series, both in systole and in diastole. Furthermore, they share common myocardial fibres (notably in the interventricular septum) and compete for the same space within the pericardium. As a result, their function and contraction affect each other – a phenomenon called ventricular interdependence. The

role of LV contraction (and most specifically the role of the septum [4]) is of great importance in LV function, being responsible for as much as 20–40% of the pressure and output generated by the right ventricle.

The right ventricle is very compliant and tolerates a large increase in preload (end-diastolic volume), without a corresponding increase in RV end-diastolic and right atrial mean pressures. Furthermore, the increased volume improves the subsequent contraction according to the Frank-Starling mechanism.

On the other hand, the low-pressure pump of the right ventricle is very sensitive to any increase in afterload. The pulmonary vascular bed consists of highly distensible vessels, which are able to accommodate large increases in blood volume, such as those that occur during exercise, while retaining a low pressure by distension and recruitment of the pulmonary microvascular bed [5]. In pulmonary vascular disease, normal resting PAP rises at a relatively late stage, when up to 70% of the bed is already obstructed.

Echocardiographic investigation of the right heart

The non-invasive method used most widely is echocardiography, which allows simultaneous morphological, haemodynamic and functional analysis of the right heart in a single examination.

Morphological evaluation

Because of its shape and position behind the sternum, the right heart has to be imaged from different echocardiographic windows to provide several cross-sectional planes (Fig. 1) [1]. The echocardiographer is then able to make a diagnosis and to quantify the degree of dilatation of the right heart cavities, from the inferior vena cava to the pulmonary artery trunk.

The vena cava is measured from the subcostal view, 1–2 cm before the right atrium, with the patient lying on the back to avoid the variations that can occur in the lateral supine position. This examination also includes end-inspiratory and end-expiratory measurements to assess the degree of collapsibility of the vena cava during inspiration.

Quantification of right atrial size is usually performed from the apical four-chamber view. By analogy with the left atrium, the end-systolic surface of the right atrium is considered to be normal when less than 20 cm² and the end-systolic volume is considered to be normal when greater than 21 ml/m² (measured by the single plane area-length method) [6].

2D quantitative assessment of the right ventricle is difficult due to its shape and correlates poorly with MRI measurements [7]; as a result, evaluation is mostly qualitative, obtained by comparison with the left ventricle. The right ventricle should not be more than two-thirds of the size of the left ventricle. Measurements can be made using the basal or mid-/end-diastolic diameters of both ventricles, or by using the ratio of the RV surface to the LV surface. In case of RV enlargement, the right ventricle will be “apex forming” [8].

RV enlargement can be quantified in the small-axis view by calculation of the eccentricity index [9]. This index is defined as the ratio of the length of two perpendicular minor-axis diameters of the left ventricle, one of which bisects and is perpendicular to the interventricular septum. This ratio, obtained at end-systole and end-diastole, allows the quantitative description of septal flattening and the differentiation between volume (normal end-systolic index, elevated end-diastolic index) and pressure (elevated end-systolic and end-diastolic indices) overload.

RV free wall thickness, normally less than 0.5 cm, is measured from the subcostal view using either M-mode or 2D imaging. Measurements of the RVOT and the pulmonary artery are most accurate from the parasternal short-axis view [6].

Haemodynamic analysis

Echocardiography allows the estimation of all the usual variables measured during invasive catheterization [10]. In addition to the assessment of right heart pressures, which will be described in detail, measurement of cardiac output and evaluation of LV filling pressures are of course part of the examination.

Pulmonary artery pressure

The most frequently used and highly reproducible method is Doppler, with the application of the modified Bernoulli formula ($\Delta P = 4V^2$) to calculate the systolic gradient across the tricuspid valve from the recording of tricuspid regurgitation maximal velocity from the apical four-chamber view or the parasternal short-axis view [11,12]. In the same manner, the proto- and end-diastolic gradients between the right ventricle and the pulmonary artery can be derived from the tracing of pulmonary regurgitation. From these gradients, and in the absence of pulmonary stenosis or intracardiac shunt, pulmonary artery systolic, proto-diastolic (considered to be the representative of mean PAP) and end-diastolic pressures can be calculated by adding an estimate of RAP. Care must be taken in patients with advanced lung disease in whom PAP is frequently overestimated from tricuspid regurgitation velocity [13].

An estimation of diastolic PAP can also be obtained by transposing the pulmonary opening time (from the onset of the R wave on the electrocardiogram to the beginning of pulmonary forward flow on Doppler examination) onto the tricuspid regurgitant velocity curve and calculating the diastolic PAP value as the pressure gradient between the right ventricle and the right atrium at that time [14,15].

The pattern of pulmonary valve flow varies with the level of PAP [16,17]. As PAP increases, peak velocity will occur earlier in systole, resulting in a shortening of pulmonary flow acceleration time. A value of less than 100 ms has a sensitivity of 78% with a specificity of 100% for diagnosing a mean PAP greater than 20 mmHg [18].

In the presence of severe pulmonary hypertension, a mid-systolic notch may appear in the deceleration phase of pulmonary flow due to the reflection of flow from a constricted and non-distensible pulmonary artery [19].

DTI also helps in the assessment of PAP. In the normal heart, the peak RV pressure is reached early during ejection,

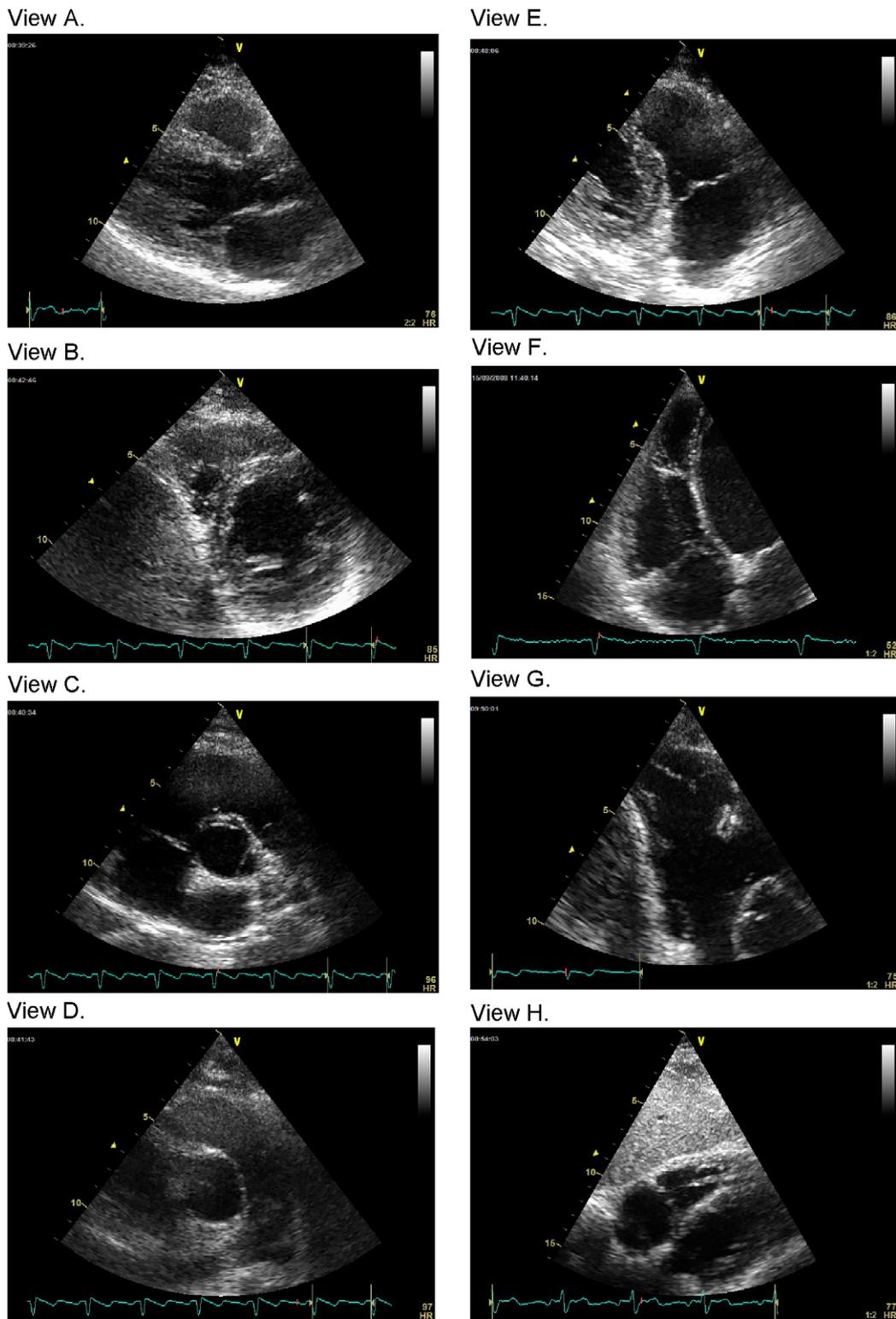


Figure 1. Echocardiographic planes for the anatomical and morphological investigation of the right heart. View A. Parasternal long axis; visualization of RV anterior wall. View B. Parasternal short axis at the ventricular level; visualization of RV anterior, lateral and inferior walls. View C. Parasternal short axis at the aortic level; visualization of the anterior wall of the outflow tract. View D. Parasternal short axis at the pulmonary level; visualization of the pulmonary trunk and its division into right and left pulmonary arteries. View E. Parasternal long axis through the right heart; visualization of RV inferior wall (left) and anterior wall (right). View F. Apical four-chamber through the right heart; visualization of RV lateral and septal walls. View G. Apical two-chamber through the right heart; visualization of RV inferior wall. View H. Subcostal; visualization of RV inferior wall.

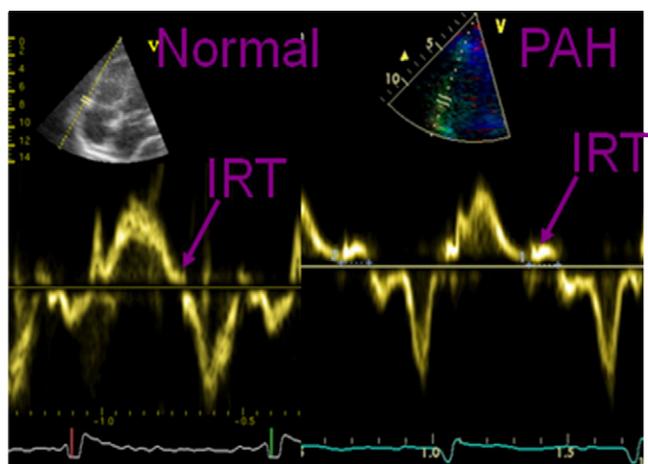


Figure 2. Increase in myocardial regional IVRT in pulmonary hypertension. Left panel: normal patient; IVRT is almost virtual. Right panel: increase of IVRT in a patient with pulmonary arterial hypertension.

which (in contrast with LV ejection) continues even during the late systolic decline of the RV pressure and after the end of the mechanical systole. Therefore, the IVRT is short and often absent. In pulmonary hypertension, the length of time for pressure to fall to the level of the right atrium is increased; furthermore, an increase in IVRT may also be due to the intrinsic alteration of RV relaxation secondary to chronically-elevated afterload.

Although not identical, the measurement of regional myocardial IVRT by DTI (measured as the time between the end of the systolic S wave and the beginning of the diastolic E wave on a DTI recording of the basal RV free wall) may be used as a surrogate measure of haemodynamic IVRT (Fig. 2). Caso et al. [20] first reported the gradual increase in regional myocardial IVRT in parallel with the increase in PAP. Further studies found variable degrees of correlation of myocardial IVRT (corrected or not corrected for heart rate) with the invasive PAP measurement [21–23]. As a significant inverse relationship was also noted with mean RAP [24], this may suggest that IVRT is an inaccurate means of estimating PAP when there is a severe increase in preload.

Right atrial pressure

RAP is usually estimated from the degree of dilatation of the inferior vena cava and from the respiratory variations of its diameter. A reduction in inferior vena cava diameter of greater than 50% is consistent with RAP less than 10 mmHg [25]. By analogy with the estimation of filling pressures in the left ventricle, an E/E' tricuspid ratio (ratio of tricuspid early diastolic velocity to early diastolic velocity of tricuspid annular displacement; Fig. 3) greater than 6 can be considered to be a marker of RAP greater than 10 mmHg, with a sensitivity of 79% and a specificity of 73% [26].

Pulmonary vascular resistance

PVR is calculated as the ratio of the transpulmonary pressure gradient ($\Delta p = \text{mean PAP} - \text{pulmonary capillary wedge pressure}$) to flow (Q_p). The ratio of peak tricuspid regurgitant velocity to the RVOT time-velocity integral obtained by Doppler echocardiography provides a clinically reliable

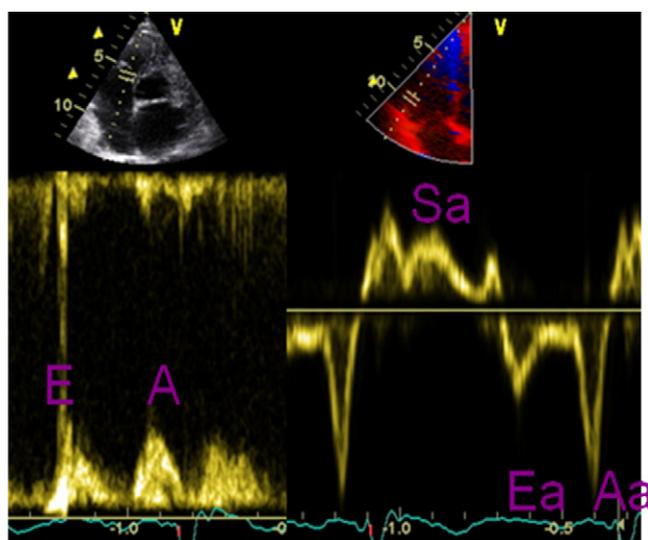


Figure 3. Ratio of tricuspid early diastolic velocity to early diastolic velocity of tricuspid annular displacement. Left panel: recording of tricuspid inflow with measurement of E velocity. Right panel: recording of lateral tricuspid annulus velocities with measurement of Ea or E' velocity.

method for estimating PVR; a cut-off value of 0.175 has a sensitivity of 77% and a specificity of 81% in determining PVR above 2 Wood's unit (WU) [27].

Evaluation of right ventricular function

By analogy with the left ventricle, the RVEF is considered to be the marker of RV function. As already mentioned, the shape of the right ventricle does not allow the use of geometric formulae to calculate RVEF. The area-length method has been used, but its results are hampered by the poor delineation of the anterior wall.

A range of echocardiographic variables has therefore been developed to evaluate RV function, and have been compared with other methods of calculating RVEF. However, as RVEF itself is highly load dependent (as shown in many studies underlining the close correlation between RVEF, mean PAP and PVR), all these echocardiographic variables are more or less load dependent. It is important, therefore, that echocardiographers remember that none of these variables is sufficiently accurate to be used alone, and that a combination of indices should be taken into account before a conclusion is made about the level of RV dysfunction [28].

Right ventricular fractional area change

RV FAC (normal value < 40%) can be derived from the end-diastolic and end-systolic areas defined by tracings of the endocardial border ($100 \times [\text{end-diastolic area} - \text{end-systolic area}] / \text{end-diastolic area}$). The major limitation is the lack of precision in endocardial delineation due to apical trabeculations, which should be included in the traces. However, the ongoing better image quality and the use of contrast agents [29] and automated border detection software [30,31] increase the diagnostic value of this variable, which has shown

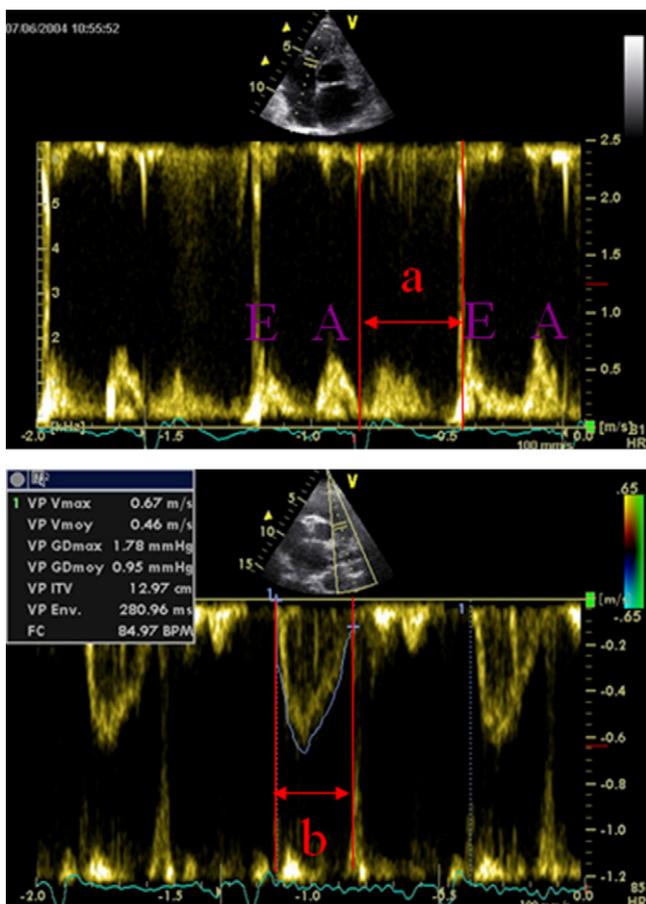


Figure 4. $MPI = (a - b)/b$ where a is the sum of isovolumic contraction time, ejection time and isovolumic relaxation time, and b is ejection time.

significant correlation with radionuclide- and MRI-derived RVEF.

Myocardial performance index

Myocardial performance index (MPI), first described by Tei et al. [32], is considered to be an index of global ventricular function, including both systolic and diastolic function. MPI is calculated as the sum of isovolumic contraction and relaxation times divided by ejection time (Fig. 4). This index increases in RV dysfunction because of lengthening isovolumic times and shortening contraction time. MPI may also be derived from DTI recordings of tricuspid annular velocities, with the advantage of being measured at the same site during the same cycle [33–35].

Recent studies confirmed correlations with both dP/dt and $-dP/dt$ [36]. MPI helps to diagnose pulmonary artery hypertension in patients with connective tissue diseases [37] and has been shown to be an independent prognostic factor in patients with pulmonary arterial hypertension [38].

Tricuspid annular motion

Tricuspid annular descent towards the apex is one of the major components of RV ejection. This probably explains why, despite being representative of a limited part of the right ventricle, variables derived from tricuspid annular

motion have been shown to be very useful in the evaluation of global RV function [39].

Tricuspid annular plane systolic excursion

TAPSE (normal value > 20 mm) is an old variable derived from M-mode analysis of the lateral tricuspid annular ring, and has shown strong correlations with radionuclide-derived RVEF [40]. TAPSE may also be derived from colour DTI images with the use of tissue tracking or from 2D speckle imaging. Many studies have confirmed the value of TAPSE in the evaluation of RV function [41] by showing correlations of TAPSE with haemodynamic- [42], radionuclide- or MRI-derived [43] RVEF. According to Urheim et al. [42], an RV displacement cut-off of 15 mm yields a sensitivity of 100% and a specificity of 41% for RV dysfunction. TAPSE is also considered to be an independent prognostic factor in patients with pulmonary arterial hypertension and in patients with heart failure [44].

Systolic velocity of tricuspid annular motion

DTI also allows analysis of tricuspid annular motion. The maximal systolic velocity of the lateral portion of the tricuspid annular ring (S wave) can be recorded from pulsed wave Doppler (Fig. 5). This variable correlates well with MRI-derived RVEF [45].

Meluzin et al. showed that an S velocity less than 11.5 cm/s was predictive of an RVEF less than 45% with a sensibility of 90% and a specificity of 85% [46] created three levels of S velocity (< 9 , 9–12 and > 12 cm/s) to distinguish three levels of RVEF (< 30 , 30–55 and $> 55\%$). In pulmonary arterial hypertension, an S value less than 9.5 cm/s yields the best diagnostic value for predicting RVEF less than 40% [47]. Furthermore, S velocity, which is independent of age [48–51], is a prognostic factor in congestive heart failure patients [52,53] and is an interesting adjunct in the diagnosis of RV myocardial infarction [54–56].

Presystolic motion of tricuspid annulus

DTI recordings of tricuspid annular velocities show a small positive wave that occurs immediately before the systolic wave during the regional isovolumic contraction time (Fig. 5). Myocardial acceleration of this wave has been validated experimentally as an RV contractility variable; it is unaffected by preload and afterload changes in the physiological range and is able to measure the force-frequency relationship [57]. In clinical studies, the maximal velocity of this wave has been shown to be independent of age [50] and to correlate well with invasive measurements of RV state of contractility and RV filling pressures [58].

The clinical value of these variables has been confirmed in patients with various congenital heart diseases [59–62] and in those who have undergone heart transplantation [63].

Measurement of right ventricular outflow tract fractional shortening

Measurement of end-diastolic and end-systolic RVOT diameters from the short-axis view allows the calculation of RVOT fractional shortening; this variable has been shown to correlate well with other echocardiographic variables such as PAP derived from tricuspid regurgitation, pulmonary acceleration time and TAPSE [64]. It was proposed by the authors as another variable to evaluate of RV systolic function.

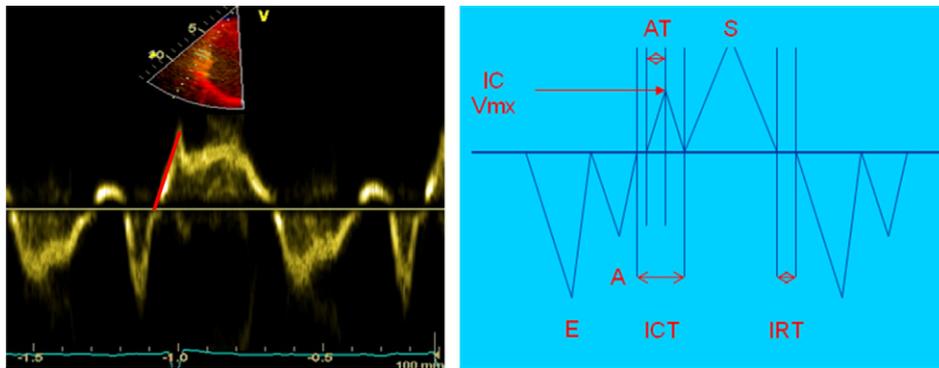


Figure 5. TDI recording of myocardial velocity at the level of tricuspid annulus. Measurement of myocardial acceleration during isovolumic contraction as the ratio of maximal velocity (ICVmax) to acceleration time (AT).

Right ventricular dP/dt

In the same manner as in the left ventricle, RV dP/dt can be extrapolated from the initial slope of the tricuspid regurgitation spectrum [65,66]. The time interval is set between 0 and 2 m/s, the difference of pressure between those two points being 16 mmHg according to the Bernoulli formula (Fig. 6). The dP/dt value is considered to be normal if it is greater than 400 mmHg/s. However, this echocardiographic index is highly correlated with PAP [67] and it has been suggested that RV dP/dt/P max (normalized by maximal tricuspid regurgitation velocity) is less sensitive to load conditions [68].

Right ventricular strain

Strain and strain rate are indices of myocardial deformation, which can be derived from DTI data or from 2D speckle imaging (Fig. 7; 2D speckle imaging calculates tissue velocities via frame-to-frame tracking of unique acoustic markers within the image and provides strain parameters in two dimensions). There is a small bias towards higher values of RV strain and strain rate using DTI [69].

DTI-derived measurement of strain in the RV anterior free wall has been correlated experimentally with regional systolic shortening measured by ultrasonomicrometry under various loading conditions [70], showing its value in the evaluation of RV contraction function.

Normal RV strain values are higher than those for the left ventricle, with a mean value around -30%. In contrast to the left ventricle, where strain is homogeneous from base to apex, strain values are higher in the median and apical segments of the right ventricle than in the basal segment, and RV longitudinal strain is higher than radial strain [71-75].

Strain measured in the apical or median portions of the right ventricle seems to be the most useful index of RV function. A RV systolic strain cut-off of 20% yielded a sensitivity

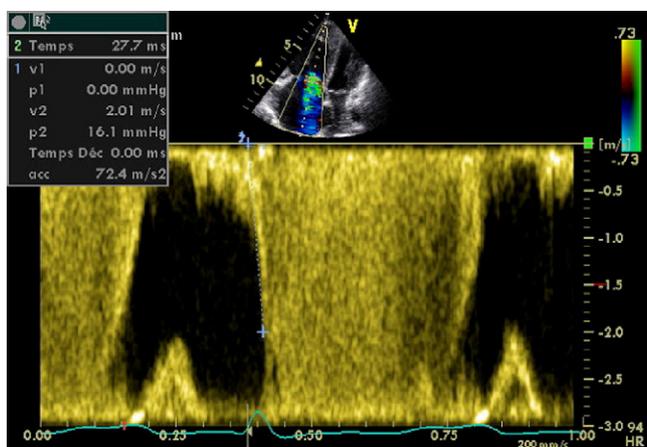


Figure 6. Measure of RV dP/dt from the tricuspid regurgitation flow (dP/dt = 16/28 = 570 mmHg). dP/dt: 575 mmHg/s.

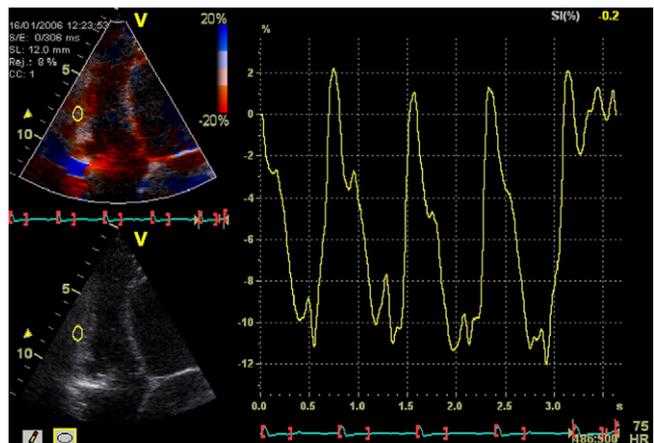


Figure 7. Upper panel: RV strain derived from 2D colour DTI. Lower panel: RV strain derived from 2D speckle imaging.

of 91% and a specificity of 63% for RV dysfunction [42]. Delayed time to peak strain is also well correlated with RV FAC [76]. RV strain also correlates with pulmonary haemodynamic indices in patients with pulmonary hypertension [77].

Many clinical studies have shown a decrease in strain and strain rate values in diseases affecting the right ventricle (congenital heart disease [78–83], pulmonary arterial hypertension [84–87], chronic obstructive pulmonary disease [88], pulmonary transplantation [89], RV myocardial infarction [90] and diabetes mellitus [91]). Furthermore, some preliminary studies have underlined the prognostic role of this variable in patients with chronic heart failure [92].

Analysis of strain curves is also interesting in terms of RV dyssynchronism, which has been shown to be prevalent in pulmonary arterial hypertension and associated with more pronounced RV dysfunction [93–95].

Three-dimensional echocardiography

In the 1990s, the first experimental studies on 3D echocardiography applied to the right ventricle concluded that this technique could reconstruct shape and quantify volume and function accurately without geometric assumptions [96,97].

The summation of disks method is usually applied, which is quite time-consuming. Images are generally analysed in short-axis view but some authors advocate the use of long-axis images to improve the results [98]. Recently new 3D transthoracic echocardiographic software adapted for right ventricle morphology was introduced and validated against MRI with excellent results [99,100].

Validation of 3D-derived RV volume and RVEF showed good results compared with MRI- or radionuclide-derived RVEF [99,101]; the results were probably better in children [102,103] than in adults where 3D echocardiography often underestimates volumes [43]. This underestimation is more important in patients with a dilated right ventricle [101].

The major limitation of 3D echocardiography applied to the right ventricle is its feasibility (around 85% with the latest software [100]), as poor detection of the RV anterior wall behind the sternum and the presence of trabeculations hamper the accuracy of endocardial tracings, as with 2D echocardiography.

Other non-invasive investigations of the right heart

Nuclear angiography

RVEF is best determined by first-pass radionuclide ventriculography after a bolus injection of a ^{99m}Tc tracer. A short sequence of cardiac cycles is acquired during transit of the bolus through the heart. The region of interest over the right ventricle and the marking of the tricuspid valve plane are defined manually and allow precise calculation of the RVEF. Quality control of bolus injection is important, as rapid bolus passage through the ventricle may reduce the counts accumulated in the RV region of interest critically, affecting the accurate determination of RVEF. Normal values of RVEF are $52 \pm 6\%$ with a lower limit of normal of 40% [104].

Equilibrium radionuclide ventriculography has been used to determine RVEF. However, the overlap between the right ventricle and the right atrium, which cannot be avoided during imaging, makes this method less accurate [105–107] and it is not recommended [108]. Tomographic equilibrium radionuclide ventriculography may be used as a less validated alternative [109].

Radionuclide-based time-activity curves are also useful in the quantification of shunts. The major limitation of radionuclide ventriculography is the lack of determination of RV volumes. Furthermore, it requires that the cardiac rhythm remains stable during acquisition.

Magnetic resonance imaging

MRI has become the technique of choice in the evaluation of RV volumes and function; it has long been shown to be accurate [110] and reproducible [111]. At present, imaging is performed on 1.5 to 3 Tesla systems, using dedicated cardiac phased-array coils with multiple elements and electrocardiogram triggering. Optimal results are obtained using fast breath-hold techniques, echoplanar or balanced fast field echo [112].

Functional (and anatomical) images of both the left and right ventricles are usually obtained in the short-axis direction. However, Alfakih et al. suggested that RV measurements made along the axial direction were more reproducible [113]. Measurement of flow velocity and volume by phase velocity mapping can be done on the right heart and allows calculation of regurgitant fractions, cardiac output, and quantification of shunt. Furthermore, contrast-enhanced MRI can be used for imaging of RV myocardial infarction [114].

MRI also has clear limitations, such as limited temporal resolution, contraindication in patients with intracardiac devices (which applies to most of our patients) and relatively limited availability. Furthermore, data acquisition and RV analysis are rather time consuming.

64-slice computed tomography scan

RVEF and RV volume can also be measured by computed tomography scan. However, the acquisition of RV data cannot be done simultaneously with that of LV data or with computed tomography coronary angiography. Examination of the right ventricle would therefore be responsible for additional radiation exposure. As it has been shown that 64-slice computed tomography coronary angiography is associated with a non-negligible lifetime attributable risk of cancer, especially in women and younger patients and for combined cardiac and aortic scans, this technique will not be used routinely in the non-invasive estimation of RV function [115].

Importance of the right heart in various pathologies

According to Bleeker et al. [112], the primary functions of the right ventricle are to maintain adequate pulmonary perfusion pressure under varying circulatory and loading conditions in order to deliver desaturated venous blood to

the gas exchange membranes of the lungs, and to maintain a low systemic venous pressure to prevent tissue and organ congestion. As long as right heart volumes and pressures stay within physiological ranges, RV function is considered to be normal.

RV dysfunction means that the right ventricle is still able to fulfil its physiological function but only by activating its reserve or compensatory mechanisms (mostly RV dilatation and/or hypertrophy). When these mechanisms are exceeded, RV failure develops and causes systemic venous congestion, underfilling of the left ventricle, low cardiac output syndrome (including reduced coronary perfusion responsible for RV myocardial ischaemia) and cardiogenic shock.

RV function may be impaired either secondary to left-sided cardiomyopathy or valvular heart disease or primary to right-sided heart or pulmonary disease. The more relevant clinical situations will be discussed. Congenital heart diseases will not be considered in this section.

Left-sided heart diseases

The most frequent cause of RV failure is left heart failure. Causes of secondary RV failure include pulmonary venous hypertension, intrinsic myocardial involvement, ventricular interdependence, neurohormonal interactions or RV myocardial ischaemia. RV failure occurs more frequently in idiopathic dilated cardiomyopathy than in ischaemic cardiomyopathy. RV function is a strong and independent prognostic factor in heart failure patients [116,117]. Many echocardiographic variables have been studied and their prognostic value has been confirmed: RV FAC in myocardial infarction [118], TAPSE [44], MPI [119], S wave and isovolumic acceleration [52,53,55,120] and RV strain [92]. The combined use of these variables increases their prognostic value [53].

RV function is also an important component of functional capacity in heart failure patients [121], and TAPSE, MPI and strain have been shown to correlate with the levels of functional variables [92,122].

Right-sided heart diseases

Right-sided heart diseases include:

- pressure overload due to pulmonary hypertension;
- volume overload due to severe pulmonary or tricuspid regurgitation or left to right shunting in atrial septal defects and anomalous pulmonary venous drainage;
- direct RV myocardial dysfunction, such as in myocardial infarction or arrhythmogenic RV dysplasia.

Pressure overload

Right heart evaluation is of major importance in all patients with pulmonary hypertension, regardless of its category according to the Evian classification [123]. RV function is an important prognostic factor in pulmonary arterial hypertension [124], along with elevated RAP and decreased cardiac output [125]. Echocardiographic factors related to prognosis include size of the right atrium, severity of tricuspid regurgitation, diastolic eccentricity index, MPI and the presence of pericardial effusion [38,126,127]. Late diastolic and systolic

velocity of the tricuspid annulus also have prognostic significance [128]. By contrast, the PAP value does not influence prognosis.

The degree of RV failure also plays an important role in acute RV pressure overload secondary to pulmonary embolism [129].

Direct right ventricular myocardial dysfunction

Right ventricular myocardial infarction

RV myocardial infarction is caused mainly by proximal right coronary artery occlusion and is associated frequently with LV dysfunction in the inferior wall. The incidence of RV myocardial infarction among patients with inferior myocardial infarction varies highly in the literature (from 20–50%) depending on the diagnostic criteria used [130]. However, the recognition of RV myocardial infarction is of significant importance as it is associated with an increased risk of death, cardiogenic shock, ventricular tachycardia or fibrillation, and high-grade atrioventricular block [131].

Usually, it is the RV free wall that is affected, and this can be imaged from the parasternal long-axis view focusing on the RV free wall, in the form of localized akinesia. Occasionally, the inferior wall may also be akinetic viewed from the RV inflow track view and subcostal projections. Furthermore, the right ventricle is often dilated in these patients [132].

Many echocardiographic variables have been found to be of value in the diagnosis of extension of inferior myocardial infarction to the right ventricle. In patients with RV involvement, the pulmonary regurgitant flow pattern is characterized by a rapid rise in flow velocity to a peak level followed by an abrupt deceleration in mid-diastole, whereas in patients without RV involvement the deceleration in mid-diastole is gradual [133]. A cut-off value of pressure half-time of pulmonary regurgitant flow less or equal to 150ms has very good diagnostic accuracy. Furthermore, this variable is also an independent predictor of in-hospital complications [134].

TAPSE [135], systolic and early-diastolic velocities of tricuspid annulus by DTI [55,56] and RV strain and strain rate [90] are decreased in patients with LV inferior wall myocardial infarction with RV infarction compared with patients without RV infarction. RV MPI is also increased significantly in these patients; however, severe RV infarction can occur with limited or no increase in the Tei index due to its pseudonormalization [136]. Repeated RV MPI measurements 5 days after thrombolysis showed a dramatic reduction in MPI [137].

Late enhancement cardiovascular MRI detects acute inferior myocardial infarction more frequently than other current standard diagnostic technique [138]. Furthermore, it shows the persistence of irreversible injury of the right ventricle at 13 months.

In survivors of RV myocardial infarction, the infarcted right ventricle recovers its function regardless of the infarcted artery patency. This suggests that the right ventricle is particularly resistant to irreversible ischaemic injury and almost always remains viable [139–141].

Arrhythmogenic right ventricular dysplasia

ARVD is a heritable cardiomyopathy characterized by the fibrofatty replacement of RV myocardium leading to RV failure and arrhythmias. The diagnosis of ARVD is based on the

presence of major and minor criteria encompassing genetic, electrocardiographic, pathophysiological and histopathological factors [142]. ARVD is responsible for RV dilatation and aneurysms, which are typical deformities located mainly in the RVOT, apex and infundibulum.

MRI is considered to be the best imaging modality for this disease [143]; it allows the visualization of adipose infiltration as a bright signal of the RV myocardium and it describes the anatomical, functional and morphological features of ARVD in a single study [144–146]. Furthermore, it has excellent correlation with histopathology and predicts inducible ventricular tachyarrhythmia on programmed electrical stimulation [147].

Cardiac multidetector computed tomography also has strong potential for detecting qualitative and quantitative abnormalities of the right ventricle in patients with ARVD [148].

Echocardiography is often difficult at the early stages of the disease; it must search for pathognomonic abnormalities that are localized aneurysmal regions in the form of end-systolic bulges, mostly found in the RVOT [149]. Other morphological features of ARVD include trabecular derangement, hyper-reflective moderator band and sacculations. In an advanced stage of ARVD, extensive areas of the RV free wall may become thin and akinetic. RV dilatation, and – more specifically – enlargement of the RVOT, occurs frequently [150]. In addition to morphological features, RV function is altered as shown by the decrease in RV FAC [150], in RV systolic velocities and strain [151,152] and in 3D RVEF [151,153].

Conclusion

The right ventricle is a pivotal chamber and its dysfunction is clinically important in the diagnosis of many diseases as it gives interesting insights into prognosis, functional capacity and therapeutic management. Despite its complex geometry and physiology, non-invasive techniques now allow the right ventricle to be evaluated accurately. Echocardiography is the most widely-used imaging technique in cardiology and must therefore include the recording of RV morphological, haemodynamic and functional variables routinely.

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