We are IntechOpen,
the world's leading publisher of
Open Access books
Built by scientists, for scientists

6,000
Open access books available

148,000
International authors and editors

185M
Downloads

Our authors are among the

154
Countries delivered to

TOP 1%
most cited scientists

12.2%
Contributors from top 500 universities

CLARIVATE ANALYTICS
BOOK CITATION INDEX
INDEXED

WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index
in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Chapter

Echocardiographic Prognostic Factors in Pulmonary Hypertension

Gabriela Silvia Gheorghe, Andrei Cristian Dan Gheorghe, Ana Ciobanu and Andreea Simona Hodorogea

Abstract

Pulmonary hypertension (PH) is defined as an increase in mean pulmonary arterial pressure of $\geq$25 mmHg at rest by right heart catheterization. Echocardiography estimates systolic pulmonary arterial pressure on the tricuspid regurgitation jet velocity, mean and diastolic pressure based on the pulmonary regurgitation jet, and data regarding the function of the right ventricle. ESC guidelines propose an echocardiographic risk assessment in PH according to right atrial area > 26 cm^2 and pericardial effusion. Other risk factors correlated with the severity of the PH include right atrial pressure > 15 mmHg, tricuspid regurgitation more than moderate, TAPSE <18 mm, tricuspid S′ < 11.5 cm/s assessed by TDI, right ventricle ejection fraction <45% using 3D imaging, fractional area change of the right ventricle <35%, dP/dt < 400 mmHg/s on the tricuspid regurgitation flow, reduced strain of the right ventricle, diastolic dysfunction. Left ventricular eccentricity index (EI) >1.7 combined with TAPSE <15 mm was associated with a higher death rate compared to patients with normal values. However, each of these parameters used in the assessment of the right ventricle has technical limitations, and it is necessary to use multiple tests for a correct evaluation of the prognosis of PH.

Keywords: pulmonary hypertension, tricuspid regurgitation, right ventricle, right atrium, global strain

1. Introduction

Pulmonary hypertension (PH) is defined as an increase in mean pulmonary arterial pressure (mPAP) of $\geq$25 mmHg at rest as assessed by right heart catheterization. Current data have shown that the normal mPAP at rest is 14 $\pm$ 3 mmHg with the upper limit of normal of approximately 20 mmHg. The clinical significance of mPAP between 21 and 24 mmHg is unclear [1]. There are also unclear data regarding the normal versus exaggerated elevation of mPAP at physical effort, making it difficult to put the diagnosis of exercise-induced PH. Pulmonary vascular resistance (PVR) with a cut-off of $\geq$3 Wood units has been included in the hemodynamic diagnosis of PH, and pulmonary artery wedge pressure (PAWP) with a cut-off of $\geq$15 mmHg is used for the classification of PH in pre-capillary, post-capillary, or combined pre- and post-capillary PH [1]. PH is classified into five groups, depending on the underlying disease [1].

IntechOpen

Not all patients with PH perform right heart catheterization, and, in practice, the diagnosis of PH is based on the echocardiographic evaluation of the pulmonary artery pressure. Also, echocardiography can detect the underlying disease and the consequences of the PH on the right and left ventricles (**Table 1**), [2–8]. Nevertheless, the echocardiographic evaluation of the right heart is more difficult than that of the left heart because of the complex shape of the right ventricle (RV) and its load-dependent physiology. Its shape in apical 4-chamber view is more triangular, in contrast to the left ventricle more conical. In the parasternal short axis view, it is like a crescent. The cavity of the RV has three parts: inlet, apical trabeculae, and outlet segments. The outlet segment is not trabeculated and is separated from the inlet segment by the supraventricular crest. The subepicardial myofibrils have a circumferential orientation, and the subendocardial myofibrils have predominantly a longitudinal orientation [9, 10]. The interventricular septum separates the right and left ventricles and bulges into RV during the ventricular systole. From the three leaflets of the tricuspid valve, the septal leaflet is usually visible by echocardiography. The 2D echography has a low sensitivity for defining the RV endocardial border contour of the free wall and of the apex. The complex morphology of the RV makes the correct echocardiographic evaluation difficult and implies great variation in the 2D measurement results of the RV diameters and area. In a study that included 900 patients, Tamborini G. et al. [11] demonstrated high inter- and intraobserver variabilities in the measurements of RV fractional area change (FAC), a parameter of RV systolic function. There are also difficulties in the echocardiographic examination of the right atria (RA). RA has an ellipsoid shape and includes crista terminalis, RA appendage, cavotricuspid isthmus, Eustachian valve, the orifice of the coronary sinus, and Thebesian valve. Echographic data include RA indexed major and minor axis length and systolic RA volume. The values are different in men and women, and the indexed RA volume using the single-plane method of disks has lower values than those obtained by the area-length method [12]. RA becomes dilated, and a process of RA remodeling occurs in longstanding PH. The RA pressure (RAP) increases, and its evaluation according to the diameter and inspiratory changes of the inferior vena cava (IVC) is an important parameter for the evaluation of RV systolic pressure in the absence of significant RV outflow tract obstruction. The RAP estimated non-invasively is the main source of errors in the assessment of both sPAP and mPAP. sPAP is calculated using the maximal velocity of tricuspid regurgitation flow (**Figure 1**) and mPAP by formulas that use pulmonary regurgitation flow velocity at the beginning of the diastole or pulmonary velocity acceleration time. Both can underestimate the PH in case of RV failure. RV failure can impede the left ventricle (LV) function by many mechanisms. So assessing the RV and LV morphology and function is essential for the correct diagnosis and prognostic of PH. The echocardiographic evaluation of the right heart can be improved through other methods, such as agitated saline study, echocardiographic contrast agents, speckle tracking, and 3D echocardiography [13]. 2D RV longitudinal strain of the free wall (RV-FWS) and 2D RV global longitudinal strain (RV-GLS), which includes in the analysis of the interventricular septum, can demonstrate subclinical impairment of the longitudinal contraction in patients with PH. The method is less angle and load-dependent and less influenced by the complex geometry of RV, but it depends on image quality that can be poor because of many artifacts [14, 15]. The analysis of segmental contractility of the RV walls by strain technique offers information about the pattern of RV remodeling in PH. The cut-off value proposed for RV-FWS is −23% and −20% for RV-GLS. For now, there is no consensus on which method to use, but it seems that RV-FWS is more useful. The 3D technique is more accurate than the 2D technique in the evaluation of RV strain.

Parameter	Method	Formula	Normal range	Abnormal values
Pulmonary artery flow evaluation				
Pulmonary velocity acceleration time (PAT)	Time to peak velocity of the pulmonary artery flow in parasternal short axis view (PW)		>130 ms	<105 ms and/or midsystolic notching
Systolic pulmonary arterial pressure	Maximum velocity of tricuspid regurgitation jet (CW)	$4V^2_{\ maxTRjet} + RAP$	18–25 mmHg; values of 40 mmHg can be normal after 60 years	≤35 mm Hg
Diastolic pulmonary arterial pressure (**Figure 2**)	End diastolic pulmonary regurgitant flow velocity (CW)	$4V^2_{\ PRenddiastolic} + RAP$	1. mm Hg	> 15 mm Hg
Mean pulmonary arterial pressure	Pulmonary regurgitant flow velocity at the beginning of the diastole (CW)	$4V^2_{\ PR\ early\ diastolic} + RAP$	10–20 mm Hg	>25 mmHg
	Pulmonary velocity acceleration time (PW)	$79 - 0.45xPAT$		
Pulmonary vascular resistance	Maximum velocity of tricuspid regurgitation (CW)	$0.16 + \frac{Vmax\ of\ TRjet}{VTIRVOT}x10^3$	<3 Wood units	>3 Wood units
Pulmonary velocity acceleration time (PW)	Time to peak the pulmonary flow		>130 ms	<100 ms
Right ventricle and RA evaluation				
RV and RA diameters				
RV diameters	2D	Basal, mid RV, longitudinal diameter, at the end-diastole RV/LV ratio, focused apical 4-chamber view	RV basal diameter 25–41 mm RV mid diameter 19–35 mm RV longitudinal diameter 59–83 mm	RV basal diameter > 41 mm RV mid diameter > 35 mm RV longitudinal diameter > 86 mm
RVOT dimensions	2D	Supra aortic valve diameter in end-systole (RVOT proximal diameter); basal short axis view, Supra pulmonary valve diameter, in end-systole (RVOT distal diameter); basal short axis view	RVOT proximal diameter 21–35 mm RVOT distal diameter 17–27 mm	RVOT proximal diameter > 35 mm RVOT distal diameter > 27 mm

Parameter	Method	Formula	Normal range	Abnormal values
RA dimensions	2D	Minor and major axis, area, volume, apical 4-chamber view, at end-systole	RA minor axis 1.9 ± 0.3 cm/m^2 RA major axis 2.5 ± 0.3 cm/m^2 RA volume 21 ± 6 mL/m^2 in women, 25 ± 7 mL/m^2 in men	RA minor axis > 4.4 cm RA major axis > 5.3 cm RA area > 18 cm^2 RA volume > 34 mL/m^2
RV free wall thickness	2D	Subcostal 4-chamber view, at end-diastole	1–5 mm	>5 mm
Interventricular septum	2D	Flattening, paradoxical movement		In the case of RV pressure overload, "the flattening" of IVS happens in both systole and diastole, in contrast to RV volume overload, with septal flattening predominantly during diastole.
LV maximal and end-systolic of eccentricity index	2D parasternal short-axis view at the mid-papillary level	The ratio of the diameters (from compact myocardium) parallel (D2) and perpendicular (D1) to the ventricular septum, as measured at end-diastole, end-systole, and maximum septal displacement		> 1.1
RV systolic function				
RV fractional area change (RVFAC)	2D, 3D	$\frac{End\ distolic\ area - End\ systolic\ area}{End\ diastolic\ area(cm^2)}$ X100	49% $\pm$ 7%	< 35%
RV ejection fraction (**Figure 4**)	2D or 3D; only 3D method has been validated	Must be a good image quality	58% $\pm$ 6.5%	< 45%
Tricuspid annular plane systolic excursion (TAPSE)	M mode	Apical 4-chamber view	24 $\pm$ 3.5 mm	≤16 mm
Tricuspid annular systolic longitudinal velocity by tissue Doppler (S′)	Pulsed TDI	Apical 4-chamber view	14.1 $\pm$ 12.3 cm/s	<9.5 cm/s

Parameter	Method	Formula	Normal range	Abnormal values
RV myocardial performance index (RVMPI)	Measurement of isovolumic contraction time (IVCT$_{RV}$), isovolumic relaxation time (IVRT$_{RV}$), and ejection time (ET) from the same heartbeat using pulsed Doppler or TDI of tricuspid annulus	(IVCT$_{RV}$ + IVRT$_{RV}$)/ET		>0.4 for pulsed Doppler RVMPI > 0.55 for TDI RVMPI
Free-wall RV longitudinal strain (RV-FWS) and global RV longitudinal strain (RV – GLS)	Speckle tracking imaging	RV-focused apical 4-chamber view; because the interventricular septum is an integral part of the LV also, RV-GLS might be influenced by LV dysfunction.	RV - FWS 29% ± 4.5% RV-GLS 22.3% ± 2.4%	RV - FWS < 20% RV-GLS <17%
Rate of RV pressure rise during early systole (dP/dt)	TR jet	The value is calculated from the slope of the line between 1 and 2 m/s (4 to 16 mmHg) of the TR spectral envelope		<400 mmHg/s
RV diastolic evaluation				
Tricuspid inflow E/A, TDE	PW Doppler	Apical 4-chamber view	E/A 1.4 ± 0.3 TDE 180 ± 31 ms	E/A < 0.8 or > 2 TDE <119 ms or > 242 ms
RV lateral wall TDI	TDI	Apical 4-chamber view	E/e′ 4 ± 1	E/e' > 6
Inferior vena cava (IVC) diameter and inspiratory collapse are used for the estimation of RA pressure (RAP)		IVC ≤ 2.1 cm, collapses >50% during sniff—RAP 0–5 mm Hg IVC > 2.1 cm, collapses >50% during sniff—RAP 5–10 mm Hg IVC > 2.1 cm, collapses <50% during sniff—RAP 10–20 mm Hg		

RVOT = right ventricle outflow tract; VTIRVOT = velocity time integral in the right ventricle outflow tract; PR = pulmonary regurgitation; TDE = E wave deceleration time; V = velocity; TDI = tissue Doppler imaging; LV = left ventricle; PW = pulse wave; CW = continuous wave; and IVC = inferior vena cava.

Table 1.
Echocardiographic parameters used for the diagnosis of pulmonary hypertension.

Also, right ventricular ejection fraction (RVEF) calculated by the 3D technique (3D-RVEF) is far more accurate than that calculated by the 2D technique (2D-RVEF) because it is independent of geometric assumptions. 3D-RVEF but not 2D-RVEF is validated in relation to cardiac magnetic resonance (CMR) imaging, the "gold standard" for assessing RVEF.

Also, 3D speckle–tracking technique provides a more accurate assessment of ventricular myocardial dynamics than 2D speckle-tracking, which is limited by the out-of-plane motion of different frames. At the same time, measurement of RVEF by 3D echocardiography is not possible in all patients because it requires good image quality [15]. Evaluation of RV shape by 3D technique is not currently used in clinical practice. In patients with a good image of tricuspid regurgitation Doppler signal, one can calculate other parameters with prognostic values, such as right ventricle-pulmonary artery coupling (RV-PA) and myocardial work.

Each technique has its limits, and one must use a multimodal evaluation of the anatomy and function of RV. Furthermore, these new echographic techniques are not standardized between different vendors. Indeed, the gold standard for RVEF evaluation remains for now cardiac magnetic resonance imaging. **Table 1** includes the echocardiographic parameters used in the evaluation of PH and RV.

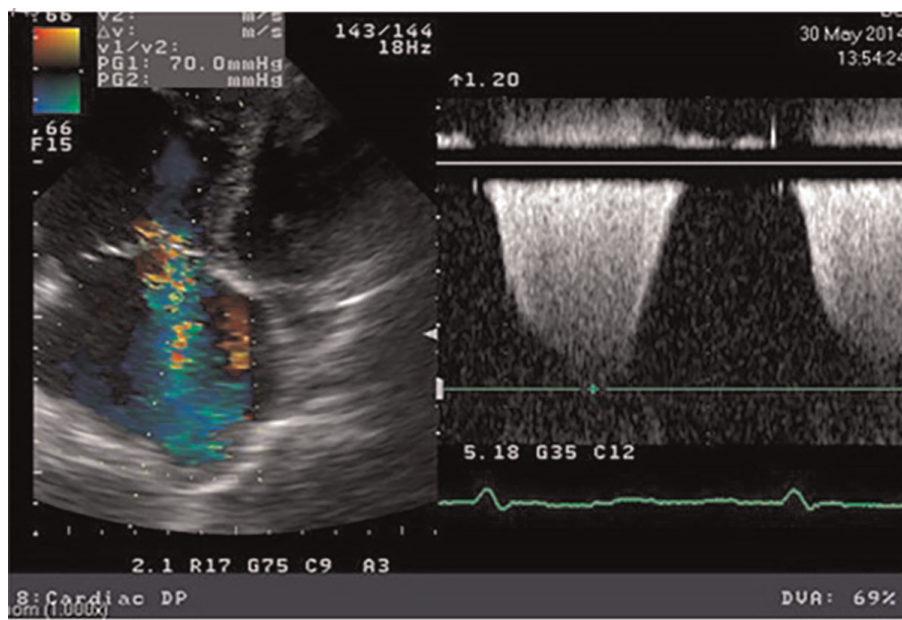**Table 1** includes the echocardiographic parameters used in the evaluation of PH and RV (**Figures 1**–**4**).

Figure 1.
Parasternal four chamber view. Tricuspid regurgitation (continuous Doppler examination). Right atrial dilation.
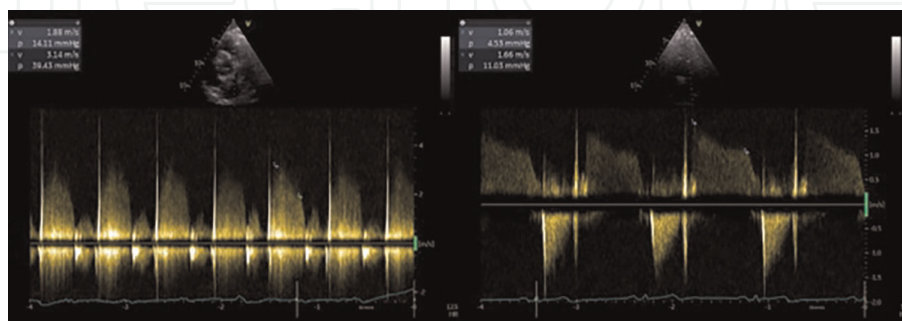

Figure 2.
Example of measurement of mPAP and dPAP in a case with pulmonary hypertension (left) versus a case without pulmonary hypertension (right). Left image: In this case, there was a dilated inferior vena cava with a diameter of 36 mm without respiratory variations. mPAP = 4 V^2 PR early diastolic + RAP = 4x3.14^2 + 20 = 54.43 mm Hg; dPAP = 4 V^2 PRenddiastolic + RAP = 4x1.88^2 + 20 = 34.13 mm Hg. Right image: In this case, there was an inferior vena cava with a diameter of 16 mm with inspiratory collapse. mPAP = 4 V^2 PR early diastolic + RAP = 4x1.66^2 + 3 = 14 mm Hg; dPAP = 4 V^2 PRenddiastolic + RAP = 4x1.06^2 + 3 = 7.5 mm Hg.
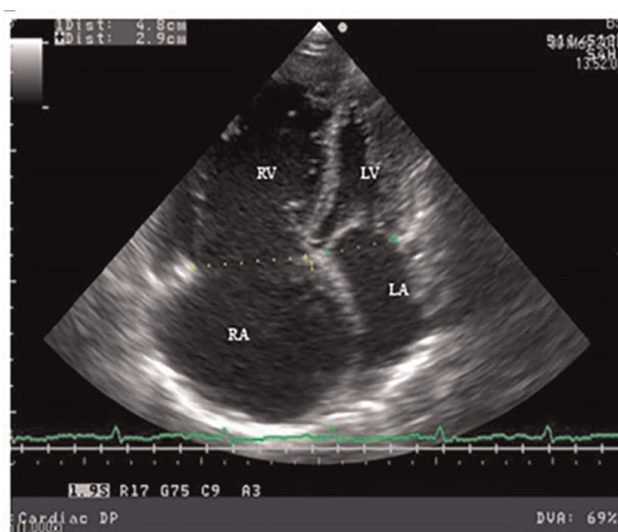
Figure 3.
Apical 4-chamber view. Dilation of the right ventricle (RV) and right atrium (RA). Displacement of the interventricular septum toward the left ventricle (LV) and interatrial septum toward the left atrium (LA).
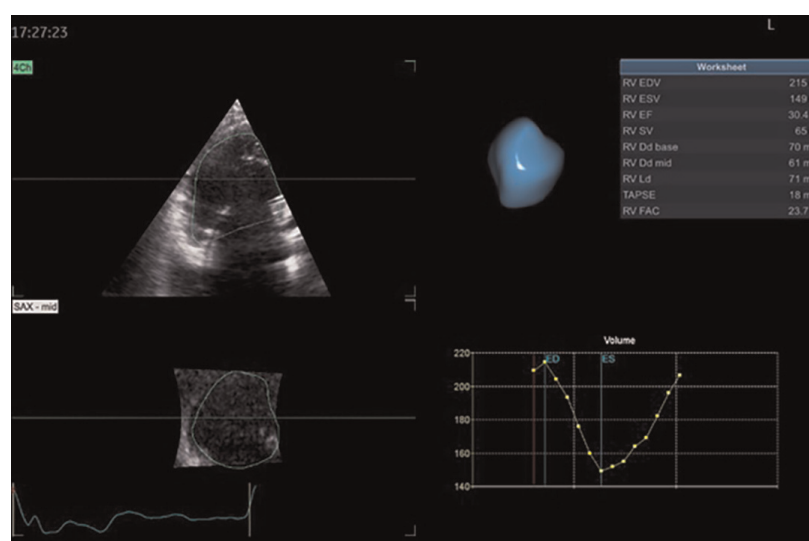

Figure 4.
3D quantification of the right ventricular ejection fraction.

2. Prognostic value of the echocardiographic data in PH

According to the clinical, biochemical, echocardiographic, or other imaging data, patients with PH can be classified as low, intermediate, and high risk of clinical worsening or death [1]. Patients categorized as low risk have estimated 1-year mortality of <5%, those categorized as intermediate risk have estimated 1-year mortality of 5–10%, and those in the high-risk category >10% [1].

The ESC guidelines on pulmonary hypertension propose an echocardiographic risk assessment in PH according to the right atrial area (area over 26 cm^2) (**Figure 1**) and the presence of pericardial effusion (**Figure 2**) [1]. However, many studies demonstrated the usefulness of other echocardiographic parameters as prognostic factors in PH. Patients, with PH and EI above 1.7 (**Figure 2**) combined with TAPSE below 15 mm, have a higher death rate than patients with normal values. The diastolic

dysfunction of the RV, expressed by the changes in tricuspid flow E/A ratio and RV relaxation abnormalities by TDI, is associated with a poor prognosis. The systolic dysfunction of the RV is related to a poor prognosis of the PH: TAPSE less than 18 mm, tricuspid S′ < 9.5 cm/s assessed by TDI, 3D - RVEF below 45%, RVFAC less than 35%, dP/dt below 400 mmHg/s on the TR flow, reduced strain of the RV using speckle tracking echocardiography (**Figure 5**).

Many studies support the prognostic role of classical echocardiographic data in PH.

- Raynold RM et al. [16] evaluated the relationships between echocardiographic findings and clinical outcomes in 81 patients with severe primary PH during a mean follow-up period of 36.9–15.4 months. Pericardial effusion and enlarged indexed RA area were independent predictors of a composite end point of death or pulmonary transplantation. They found that septal shift in diastole toward LV is also a predictor of death or pulmonary transplantation. A dilated RA can be a sign of volume and/or pressure overload, and elevated RA pressure is a sign of poor RV ejection fraction [17].

- Austin C et al. [18] performed a retrospective analysis of 121 consecutive patients with pulmonary arterial hypertension during 3 years of follow-up. They demonstrated in a univariate analysis that RA pressure > 15 mmHg, calculated by inferior *vena cava* diameter and collapsibility, RA area > 18 cm^2, the presence of pericardial effusion, RVFAC < 35% and at least moderate TR were predictive of poor survival. However, in the multivariate analysis, RA pressure > 15 mm Hg was the only echocardiographic risk factor predictive of mortality.

- Liu K et al. [19] performed a meta-analysis that included 12 studies totalizing 1085 patients with pulmonary arterial hypertension followed up 9.2 months to 5.0 years. The risk of all-cause mortality and the composite endpoint of death and other PH events was increased in patients with enlarged RA area and RA area index.

- TAPSE less than 1.8 cm was associated with lower RVFAC, lower cardiac index, and reduced survival in 63 patients with PH [20]. The 2 years survival rate was 50% in patients with TAPSE less than 1.8 cm and 88% in those with greater TAPSE. However, TAPSE assessed by M mode cannot discriminate an active contraction from passive entrainment. On the other side, the assessment of TAPSE by RV strain can obtain greater values because TAPSE measures the maximum displacement, and strain measures the peak systolic contraction.
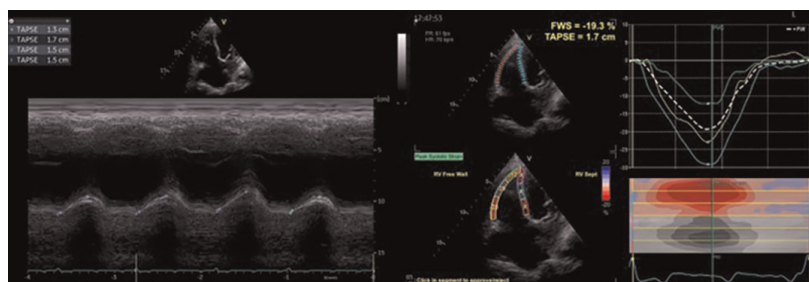

Figure 5.
Example of measurement of free wall right ventricular strain (FWS). Strain-based TAPSE is an approximated M-mode TAPSE, by calculation of the excursion relative to the image apex.

- In a prospective cohort study that included 777 patients with precapillary PH with a follow-up period of 7 years, the multivariable analysis demonstrated that moderate or severe tricuspid regurgitation, RVMPI, presence of pericardial effusion, but not TAPSE were independent predictors of mortality. The authors explained this fact by the presence of severe tricuspid regurgitation in most patients, which can induce a pseudo-normal TAPSE [21].

- Ghio S. et al. [22] examined data from 517 patients, mean age 52 ± 15 years, 64.8% females, included in seven observational studies. They divided patients into three groups according to TAPSE, tricuspid regurgitation, the diameter of inferior *vena cava*, and noted the 5 years cumulative survival. High-risk patients had impaired TAPSE and dilated inferior *vena cava*, and their 5 years cumulative survival rate was 43%, versus 82% in low-risk patients, with normal TAPSE and no tricuspid regurgitation. The intermediate-risk group had normal TAPSE and significant tricuspid regurgitation or impaired TAPSE and non-dilated inferior *vena cava*. Their 5 years cumulative survival rate was 63%. Data from this analysis suggested that the inclusion of the RA area and pericardial effusion did not provide added prognostic value.

- Barket D et al. [23] demonstrated in 78 pediatric patients with PH that an end-systolic EI > 1. 16 identifies the presence of PH, and an end-systolic EI > 1.27 correlates with a higher number of hospitalization and escalation of the therapy.

- RVMPI and the rate of RV pressure rise during early systole (dP/dt) proved to correlate with pulmonary vascular resistance and to predict a reduced RVEF [17].

- Elevated RAP is a sign of poor right ventricular failure [17].

- Blanchard D. et al. [24] demonstrated in 93 patients with chronic thromboembolic pulmonary hypertension that elevated RVMPI measured by TDI correlated with pulmonary vascular resistance but not with mPAP and cardiac output.

- Habbad et al. [25] elaborated a right heart score for the outcome of patients with familial, idiopathic, drug-induced, and toxic PH based on the examination of 95 patients, 43 ± 11 years old, with mean pulmonary arterial pressure of 54 ± 14 mm Hg, and pulmonary vascular resistance index 25 ± 12 Wood units, followed for 5 years. The severity of RV systolic dysfunction, RA enlargement, and systolic blood pressure < 110 mmHg were independently associated with death and lung transplantation.

- Kamimura Y. et al. [26] proposed a prognostic score in chronic thromboembolic pulmonary hypertension calculated as the summation of each point awarded for the presence of four parameters: TAPSE < 16 mm, 1 point, TDI-derived tricuspid lateral annular systolic velocity (S′) < 10 cm/sec, 1 point, RVFAC < 35% 1 point and RVMPI > 0.4, 1 point. An elevated RV dysfunction prognostic score was associated with an advanced functional class of heart failure, elevated mPAP, low cardiac index, high pulmonary vascular resistance, reduced mixed venous oxygen saturation, reduced 6-min walk distance, low maximal O_2 consumption,

prolonged slope of minute ventilation/CO_2 production, and elevated plasma brain natriuretic peptide level.

- REVEAL study (US Registry to Evaluate Early and Long-Term PAH Disease Management) [16] enrolled 2716 patients with PAH and assessed predictors of 1-year survival. In the multivariable analysis, variables independently associated with increased mortality included PVR > 32 Wood units, PAH associated with portal hypertension, New York Heart Association functional class IV, men > 60 years of age, and family history of PAH.

- Badagliacca R et al. [27] investigated during 528 ± 304 days 130 patients with idiopathic pulmonary hypertension. They demonstrated that clinical, functional class of heart failure, cardiac index, and RVFAC were the independent predictors of the clinical worsening of the patients.

- The most frequently used parameters for the assessment of prognostic in PH in real-world practice are presented in **Table 2** [28, 29].

Speckle tracking echocardiography applied to the RV has demonstrated its utility in the prognostic stratification of patients with PH:

- Park JH et al. [30] determined RV-GLS in 81 patients with PAH with a follow-up period of 45 ± 15 months. They showed a significant correlation between RV-GLS and RVFAC, TAPSE, RVMPI, pulmonary vascular resistance, and B-natriuretic peptide concentration. In the multivariate analysis, RV-GLS ≥ −15.5% and age were the independent predictor factors for death, lung transplantation, and heart failure hospitalization. Accepted normal values for RV-GLS were − 28%.

- Badagliacca R et al. [31] analyzed RV strain patterns using the speckle tracking technique and identified three post-systolic strain patterns derived from the mid-

Echocardiographic parameters	Abnormal values predicting a poor prognosis of PH
TAPSE	<16 mm
S´	<10 cm/s
RV FAC	<35%
Peak longitudinal RV strain	≥−19%
Isovolumetric contraction velocity (IVCv) by TDI	<9 cm/s
Main PA diameter	>29 mm
LV eccentricity index	>1.4
Pericardial effusion	presence
IVC	>21 mm, inspiratory collapse <50%

TAPSE = tricuspid annular plane systolic excursion; S´ = right ventricle free wall tissue Doppler systolic velocity during ejection period (Lateral tricuspid annulus peak systolic velocity); RVFAC = right ventricle fractional area changes PA = pulmonary artery; LV = left ventricle; and TDI = tissue Doppler imaging.

Table 2.
Echocardiographic parameters recommended in clinical practice for the assessment of PH prognosis.

basal RV free wall segments. Pattern 1 was characterized by a prompt return of strain-time curves to baseline after peak systolic negativity, like in normal control subjects, and corresponded to mild PH. Pattern 2 was characterized by persisting negativity of strain-time curves well into diastole before an end-diastolic returning to baseline and corresponded to more advanced PH with preserved RV function. Pattern 3 was characterized by a slow return of strain-time curves to baseline during diastole corresponded to PH with end-stage RV failure. 60% and respectively 33% of patients with Pattern 3 had a faster-worsening disease assessed at 1 and 2 years

- RV longitudinal strain is superior to TAPSE as a prognostic factor in PH [32]

- RV-arterial coupling reflects both RV after-load and contractility and has prognostic value in PH. It can be assessed using various parameters, such as TAPSE, FAC, 3D-RVEF, which are divided to sPAP. [15]. Serkan Unlu et al. [33] determined RA-arterial coupling by the ratio between RV-FWS and sPAP in 65 patients with precapillary PH and found a predictive value for death or heart/lung transplantation with a cut-off of 0.19.

- RV myocardial work enables the evaluation of the contractility of RV independent of the load and a more precise estimate of RV systolic function. It is a promised parameter but not in clinical practice.

3. 3D echocardiography has become increasingly important in the evaluation of RV

- Murata M et al. [34] highlighted the importance of evaluating RV ejection fraction by 3D technique in 85 patients with PH. They showed that patients with 3DRVEF of less than 38% had significantly shorter event-free survival than those with 3DRVEF greater than 38%. A total of 36 patients had mPAP >35 mmHg and a theoretical event-free survival in 2.5 years. Those with greater 3DRVEF had an event-free survival rate of 70% at 2.5 years. This suggests that 3DRVEF is an independent prognosis factor in patients with PH.

- Vitarelli A. et al. [35] studied 73 patients, mean age 53 ± 13 years with chronic PH of different etiologies, and 30 healthy subjects as a control group. They determined RVFAC, TAPSE, mitral and tricuspid TDI annular velocities, 3D RV volumes, 3D RV EF, and RV strains by 2D RV and 3D RV-speckle-tracking echocardiography. RV 3D global-free-wall longitudinal strain (3DGFW-RVLS), 2D global-free-wall longitudinal strain (2DGFW-RVLS), apical-free-wall longitudinal strain, basal-free-wall longitudinal strain, and 3D-RVEF were lower in patients with PH. Also, patients with precapillary PH had lower global and regional peak systolic RV free-wall strain than those with postcapillary PH. 3D-GFW-RVLS and 3D-RVEF were the independent predictors of mortality. 3D RVEF, 2D-STE, and 3D-STE parameters indicate global and regional RV dysfunction associated with RV failure hemodynamics better than conventional echo indices.

- The validity and prognostic importance of 3D-RVEF were recently demonstrated in 446 unselected patients with various cardiac diseases with a follow-up period

of 4.1 ± 1.2 years. Patients with 3DRVEF > 45% had the best prognosis, and those with 3DRVEF under 30% had the worst prognosis [36].

4. RV dysfunction as a prognostic factor in patients with cardiac resynchronization therapy (CRT) and cardiac surgery

The evaluation of the RV dysfunction has a prognostic role in the short and long-term outcomes of patients after cardiac resynchronization therapy (CRT). Nagy VC. et al. [37] studied 93 patients with heart failure and low basal value of RV global longitudinal strain and RV free wall strain.

- RV global longitudinal strain below 10.04% before CRT was associated with high 24-month mortality.

- Preoperative RV dysfunction is a prognostic factor for perioperative complications in cardiac surgery [38]. There are studies that demonstrate preoperatory RVFAC <32% and RVMPI >0.5 are prognostic factors for postoperative circulatory failure, higher incidence of postoperative inotropic support, and longer stay in the intensive care unit. Preoperative RVFAC <20% is associated with late postoperative death [39]. The prognostic role of preoperative RV dysfunction is important in both coronary bypass and valvular surgery. Echocardiographic parameters of RV function proposed as preoperative poor prognostic factors included RVFAC<25%, bowing end-systole or end-diastole interventricular septum into left ventricle (flattening or paradoxical movement), TAPSE<1.4 cm, RV longitudinal strain $\leq -15\%$, TAPSE/sPAP [40].

5. Conclusions

The evaluation of the function of RV in PH is essential in establishing the prognosis of PH. In addition to the size of the RA and the presence of pericardial effusion proposed by the guidelines, there are many echocardiographic parameters with prognostic value in PH. They are related to the systolic and diastolic dysfunction of the right ventricle explored by new echocardiographical techniques such as speckle tracking and 3D imaging. These promising techniques investigate in depth the function of the RV and the correlation between RV and pulmonary artery and allow an early diagnosis of the impairment of the RV in PH. Also, the new echographic techniques are useful in the prognostic evaluation of CRT and cardiac surgery. However, each of the echocardiographic parameters used in the assessment of the right ventricle has technical limitations, and it is necessary to use multiple clinical, biological, and echocardiographic tests for a correct evaluation of the prognosis of PH. The complexity of the RV makes useful a multiparametric examination, and modern techniques are increasingly useful. The modern multimodal evaluation of the RV includes not only echocardiography but also cardiac magnetic resonance, nuclear imaging techniques, metabolic imaging techniques, and cardiac scanners. Last but not the least, the influence of RV dysfunction on the LV can be systematically assessed.

Author details

Gabriela Silvia Gheorghe[1], Andrei Cristian Dan Gheorghe[2*], Ana Ciobanu[1,2]
and Andreea Simona Hodorogea[1,2]

1 Cardiology and Internal Medicine Department, Theodor Burghele Clinical Hospital,
Bucharest, Romania

2 Carol Davila University of Medicine and Pharmacy, Bucharest, Romania

*Address all correspondence to: acd2403@yahoo.com

IntechOpen

References

[1] Galie N, Humbert M, Vachiery J-L, Gibbs S, Lang I, Torbicki A, et al. 2015 ESC/ERS guidelines for the diagnosis and treatment of pulmonary hypertension the joint task force for the diagnosis and treatment of pulmonary hypertension of the european society of cardiology (ESC) and the european respiratory society (ERS). European Heart Journal. 2016;**37**(1):67-119. DOI: 10.1093/ eurheartj/ehv317

[2] Armstrong WF. Thomas Ryan Feigenbaum's Echocardiography. eight ed. Wolters-Kluwer; 2019. ISBN-13 978-1-4511-9427-2

[3] Lang RM, Goldstein SA, Kronzon I, Khanderhia BA, Saric M, Mor-Avi V. ASE's Comprehensive Echocardiography. third ed. Philadelphia, PA: Elsevier; 2022. pp. 19103-2899. ISBN 9780323698306 Library of Congress Control Number 2020945518, printed in Canada

[4] Ginghină C, Popescu B. Ruxandra Jurcuț Esențialul în ECOCARDIOGRAFIE. Editura Medicala Antaeus. 2nd ed. 2013. pp. 259-274. www.antaeus.ro. ISBN 24 978-606-8470-02-3

[5] Grünig E, Henn P, D'Andrea A, Claussen M, Ehlken N, Maier F, et al. Reference values for and determinants of right atrial area in healthy adults by 2-dimensional echocardiography. Circulation: Cardiovascular Imaging. 2013;**6**:117-124. DOI: 10.1161/ CIRCIMAGING.112.978031

[6] Parasuraman S, Walker S, Loudon BL, Gollop ND, Wilson AM, Lowery C, et al. Assessment of pulmonary artery pressure by echocardiography—A comprehensive review. IJC Heart & Vasculature. 2016; **6**(12):45-51

[7] DiLorenzo MP, Bhatt SM. Laura Mercer-Rosa How best to assess right ventricular function by echocardiography. Cardiology in the Young. 2015;**25**(8):1473-1481. DOI: 10.1017/S1047951115002255

[8] Benza RL, Miller DP, Gomberg-Maitland M, Frantz RP, Foreman AJ, Coffey CS, et al. Predicting survival in pulmonary arterial hypertension insights from the registry to evaluate early and long-term pulmonary arterial hypertension disease management (REVEAL). Circulation. 2010;**122**:164-172

[9] Ho SY, Nihoyannopoulos P. Anatomy, echocardiography, and normal right ventricular dimensions. Heart. 2006;**92**(Suppl. 1):i2-i13. DOI: 10.1136/hrt.2005.077875

[10] Zaidi A, Knight DS, Augustine DX, Harkness A, Oxborough D, Pearce K, et al., editors. Vishal Sharma echocardiographic assessment of the right heart in adults: A practical guideline from the British Society of Echocardiography, 2020 British Society of Echocardiography. Echo Research & Practice. 2020;**7**:G19-G41. DOI: 10.1530/ ERP-19-0051

[11] Tamborini G, Pepi M, Galli CA, Maltagliati A, Celeste F, Muratori M, et al. Feasibility and accuracy of a routine echocardiographic assessment of right ventricular function. International Journal of Cardiology. 2007;**115**(1): 86-89. DOI: 10.1016/j.ijcard.2006.01.017

[12] Lang RM, Cameli M, Sade LE, Faletra F, Fortuni F, Rossi A, et al. Imaging assessment of the right atrium: anatomy and function. European Heart Journal-Cardiovascular Imaging. 2022;**23**:867-884. DOI: 10.1093/ehjci/ jeac011

[13] Jones N, Burns AT, Prior DL. Echocardiographic assessment of the right ventricle–state of the art. Heart, Lung and Circulation. 2019;**28**: 1339-1350. DOI: 10.1016/j. hlc.2019.04.016

[14] Gripari P, Muratori M, Fusini L, Tamborini G, Ali SG, Brusoni D, et al. Right ventricular dimensions and function: Why do we need a more accurate and quantitative imaging? Journal of Cardiovascular Echography. 2015;**25**(1):19-25. DOI: 10.4103/2211-4122.158420

[15] Surkova E, Cosyns B, Gerber B, Gimelli A, La Gerche A, Marsan NA. The dysfunctional right ventricle: The importance of multi-modality Imaging. European Heart Journal - Cardiovascular Imaging. 2022;**23**:885-897. DOI: 10.1093/ehjci/jeac037)

[16] Raymond RJ, Hinderliter AL, Willis PW, IV DR, Caldwell EJ, Williams W, et al. Echocardiographic Predictors of Adverse Outcomes in Primary Pulmonary Hypertension. Journal of the American College of Cardiology. 2002;**39**(7):1214-1219

[17] Schneider OM, Binder T. Echocardiographic evaluation of the right heart. Wiener Klinische Wochenschrift. 2018;**130**:413-420. DOI: 10.1007/s00508-018-1330-3

[18] Austin C, Alassas K, Burger C, Safford R, Pagan R, Duello K, et al. Echocardiographic assessment of estimated right atrial pressure and size predicts mortality in pulmonary arterial hypertension. Chest. 2015;**147**(1): 198-208. DOI: 10.1378/chest.13-3035

[19] Liu K, Zhang C, Chen B, Li M, Zhang P. Association between right atrial area measured by echocardiography and prognosis among pulmonary arterial hypertension: A systematic review and meta-analysis. BMJ Open. 2020;**10**: e031316. DOI: 10.1136/bmjopen-2019-031316

[20] Forfia PR, Fisher MR, Thai SC, Housten-Harris T, Hemnes AR, Borlaug BA, et al. Tricuspid annular displacement predicts survival in pulmonary hypertension. American Journal of Respiratory and Critical Care Medicine. 2006;**174**:1034-1041

[21] Grapsa J, Nunes MCP, Tan TC, Cabrita IZ, Coulter T, Smith BCF, et al. Echocardiographic and hemodynamic predictors of survival in precapillary pulmonary hypertension seven-year follow-up. Circulation: Cardiovascular Imaging. 2015;**8**:45-54. DOI: 10.1161/CIRCIMAGING.114.002107

[22] Ghio S, Mercurio V, Fortuni F, Forfia PR, Gall H, Ghofrani A, et al. A comprehensive echocardiographic method for risk stratification in pulmonary arterial hypertension. European Respiratory Journal. 2020;**56** (3):2000513. DOI: 10.1183/13993003.00513-2020

[23] Burkett DA, Patel SS, Mertens L, Friedberg MK, Ivy D. Relationship between left ventricular geometry and invasive hemodynamics in pediatric pulmonary hypertension. Circulation: Cardiovascular Imaging. 2020;**13**: e009825. DOI: 10.1161/CIRCIMAGING.119.009825

[24] Blanchard DG, Malouf PJ, Gurudevan SV, Auger WR, Madani MM, Thistlethwaite P, et al. Utility of right ventricular tei index in the noninvasive evaluation of chronic thromboembolic pulmonary hypertension before and after pulmonary thromboendarterectomy A C. Cardiovascular Imaging. 2009;**2**(2): 143-149

[25] Haddad F, Spruijt OA, Denault AY, Mercier O, Brunner N, Furman D, et al. Right heart score for predicting outcome in idiopathic, familial, or drug- and toxin-associated pulmonary arterial hypertension JACC. Cardiovascular Imaging. 2015;**8**(6):627-638

[26] Kamimura Y, Okumura N, Adachi S, Shimokata S, Tajima F, Nakano Y, et al. Usefulness of scoring right ventricular function for assessment of prognostic factors in patients with chronic thromboembolic pulmonary hypertension. Heart and Vessels. 2018;**33**:1220-1228. DOI: 10.1007/s00380-018-1168-7

[27] Badagliacca R, Papa S, Valli G, Pezzuto B, Poscia R, Manzi G, et al. Echocardiography combined with cardiopulmonary exercise testing for the prediction of outcome in idiopathic pulmonary arterial hypertension. Chest. 2016;**150**(6):1313-1322. DOI: 10.1016/j.chest.2016.07.036

[28] Moceri P, Baudouy D, Chiche O, Cerboni P, Bouvier P, Chaussade C, et al. Imaging in pulmonary hypertension: Focus on the role of echocardiography. Archives of Cardiovascular Disease. 2014;**107**:261-271

[29] Frost A, Badesch D, Simon J, Gibbs R, Gopalan D, Khanna D, et al. Diagnosis of pulmonary hypertension. European Respiratory Journal. 2019;**53**:1801904. DOI: 101183/13993003.01904-20180

[30] Park J-H, Park MM, Farha S, Sharp J, Lundgrin E, Comhair S, et al. Impaired global right ventricular longitudinal strain predicts long-term adverse outcomes in patients with pulmonary arterial hypertension. Journal of Cardiovascular Ultrasound. 2015;**23**(2):91-99

[31] Badagliacca R, Pezzuto B, Papa S, Poscia R, Manzi G, Pascaretta A, et al. Right ventricular strain curve morphology and outcome in idiopathic pulmonary arterial hypertension. Cardiovascular Imaging. 2021;**14**(1):162-172

[32] Muraru D, Haugaa K, Donal E, Stankovic I, Voigt JU, Petersen SE, et al. Right ventricular longitudinal strain in the clinical routine: A state-of-the-art review. European Heart Journal - Cardiovascular Imaging. 2022;**23**(7):898-912. DOI: 10.1093/ehjci/jeac022

[33] Ünlü S, Bézy S, Cvijic M, Duchenne J, Delcroix M, Voigt JU. Right ventricular strain related to pulmonary artery pressure predicts clinical outcome in patients with pulmonary arterial hypertension. European Heart Journal – Cardiovascular Imaging. 2022;jeac136. DOI: 10.1093/ehjci/jeac136

[34] Murata M, Tsugu T, Kawakami T, Kataoka M, Minakata Y, Endo J, et al. Prognostic value of three-dimensional echocardiographic right ventricular ejection fraction in patients with pulmonary arterial hypertension. Oncotarget. 2016;7(52):86781-86790

[35] Vitarelli A, Mangieri E, Terzano C, Gaudio C, Salsano F, Rosato E, et al. Three-dimensional echocardiography and 2D-3D speckle-tracking imaging in chronic pulmonary hypertension: Diagnostic accuracy in detecting hemodynamic signs of right ventricular (RV) failure. Journal of the American Heart Association. 2015;**4**:e001584

[36] Muraru D, Badano LP, Nagata Y, Surkova E, Nabeshima Y, Genovese D, et al. Development and prognostic validation of partition values to grade right ventricular dysfunction severity using 3D echocardiography. European

Heart Journal - Cardiovascular Imaging. 2020;**21**:10-21

[37] Nagy VK, Széplaki G, Apor A, Kutyifa V, Kovács A, Kosztin A, et al. Role of right ventricular global longitudinal strain in predicting early and long-term mortality in cardiac resynchronization therapy patients. PlosOne. 2015. DOI: 10.1371/journal. pone.0143907

[38] Haddad F, Couture P, Tousignant C, Denault AY. The right ventricle in cardiac surgery, a perioperative perspective: II. Pathophysiology, clinical importance, and management. Anesthesia and Analgesia. 2009;**108**(2): 422-433

[39] Wencker D, Borer JS, Hochreiter C, Devereux RB, Roman MJ, Kligfield P, et al. Preoperative predictors of late postoperative outcome among patients with nonischemic mitral regurgitation with 'high risk' descriptors and comparison with unoperated patients. Cardiology. 2000;**93**:37-42

[40] Navaratnam M, DiNardo JA. Perioperative right ventricular dysfunction - The anesthesiologist's view. Cardiovascular Diagnosis and Therapy. 2020;**10**(5):1725-1734. DOI: 10.21037/cdt-20-426)