Echocardiographic estimation of right ventricular wall tension: haemodynamic comparison and long-term follow-up

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Abstract
Prognosis in pulmonary hypertension is strictly linked to right ventricle failure, which results from uncoupling between right ventricle function and its afterload. This study sought to describe how to estimate with echocardiography right ventricular wall tension, its correlation with right ventricle haemodynamics and its prognostic role. A total of 190 patients without overt right ventricle failure but with suspected pulmonary hypertension on a previous echocardiogram underwent right heart catheterization and nearly-simultaneous echocardiography. Right ventricular wall tension was estimated according to Laplace’s law as right ventricle length \(/\) tricuspid regurgitation peak gradient and it was correlated with right ventricle haemodynamic profile; its potential prognostic impact was tested along with canonical right ventricle function parameters. Right ventricular wall tension correlated significantly with invasive estimation of right ventricle end-diastolic pressure (\(R: 0.343, p < 0.001\)) and with several other haemodynamic variables, such as mean pulmonary artery pressure, pulmonary artery compliance, transpulmonary gradient, pulmonary vascular resistance, right atrial pressure and right ventricle stroke work index (all \(p < 0.001\)). At a mean follow-up of five years and three months, only right ventricular wall tension was associated to all-cause mortality (\(p = 0.036\)), while tricuspid annular plane systolic excursion (\(p = 0.536\)), right ventricle fractional area change (\(p = 0.383\)), right ventricle fractional area change (\(p = 0.076\)), tricuspid regurgitation peak gradient (\(p = 0.107\)) and tricuspid annular plane systolic excursion/tricuspid regurgitation peak gradient (\(p = 0.181\)) could not. We identified a novel bedside echocardiographic predictor of altered right ventricle haemodynamics, which is precociously altered in patients without overt right ventricle failure and is associated to all-cause mortality at a long-term follow-up. Further studies are needed to confirm its role in pulmonary hypertension patients.

Keywords
right ventricle, wall tension, echocardiography, pulmonary hypertension

Introduction
Pulmonary hypertension (PH) is a severe disease with a poor prognosis, \(^1\) which may originate from a primary pulmonary vascular dysfunction or from right ventricle (RV) pressure overload secondary to left heart disease. \(^2\) In both cases, the natural history of the disease evolves into progressive RV failure that represents the main determinant of PH prognosis. \(^3,4\)

Several studies have focused on the coupling between RV function and pulmonary artery (PA) resistance, hence shaping a pathophysiological background that accurately describes how RV haemodynamics adapt to the pressure overload that accompanies PH. \(^5,6,7\) According to this model, RV–PA coupling is mainly defined by \(Ees/Ea\), where \(Ees\) represents end-systolic RV elastance and \(Ea\) is the RV stroke work index (W).
describes PA elastance; Ees is defined as the slope between the end-systolic pressure versus the end-systolic volume in pressure–volume loop under different loading status, while Ea is defined as the ratio between end-systolic pressure and stroke volume (SV).

PH is characterized by an initial increase of Ea, to which the RV responds with enhanced inotropism, thus leading to higher Ees; since both factors increase in a similar fashion, in this first phase Ees/Ea will remain normal or nearly normal. Nevertheless, this adaptive mechanism occurs at the cost of increased right ventricular end-diastolic pressure (RVEDP), which translates into raised RV wall tension (RVWT) and altered RV energetics.6,9 While PH progresses and PA elastance increases, Ea will eventually reach a critical point that cannot be further tolerated by the RV; as RVEDP rises beyond this point, Ees/Ea will then decrease and signs and symptoms of RV failure will be manifest.10

We investigated RVWT, derived from transthoracic echocardiography, in comparison with contemporary available methods estimating RV haemodynamics, and we assessed whether it can stratify prognosis in a precocious phase of PH, when canonical echocardiographic parameters are expected to be normal or nearly normal.

Methods

Study population

The study population of the present work was selected from the RIGHT1 study,11 a prospective, blinded study designed to compare the performance of several echocardiographic indices of pulmonary haemodynamics. RIGHT1 was conducted between July 2011 and November 2013, enrolling 200 patients with a generic indication for right heart catheterization (RHC) referred to the Haemodynamic Laboratory of the AOU Città della Salute e della Scienza of Turin. Exclusion criteria included ongoing infusion of inotropes, overt heart failure, known pulmonary stenosis or ventilatory support.

Haemodynamic assessment

RHC was performed through femoral or jugular access according to operator’s preference. Right atrial pressure, RV pressure, PA pressure and pulmonary capillary wedge pressure were acquired. The zero reference level was always set at the midthoracic level. Cardiac output was evaluated using Fick’s method and/or thermodilution. PA compliance was calculated according to the single element model as SV/PA pulsatory pressure (namely, systolic–diastolic PA pressure).12 All measurements were made at end-expiration, and values used in our analysis were a mean of three acquisitions, in case sinus rhythm was present, or five in case of atrial fibrillation. Physicians performing the RHC were blinded to the results of the transthoracic echocardiography.

Hemodynamic values were interpreted according to international consensus.13,14

Echocardiographic assessment

All patients underwent a complete transthoracic echocardiography within 30 min prior to RHC; RHC was started immediately after echocardiography. A Philips IE33 machine (Philips Medical Systems, Andover, MA) with a S5 probe was used for all 2D and Doppler acquisitions. All echocardiographic examinations were performed with a respirometer for the precise timing of respiratory cycles. Patients were instructed to breathe normally. All morphologic and functional chamber evaluations were made according to the international guidelines available in 201315,16; multiple indexes of RV diastolic and systolic function were acquired accordingly. Two operators (C.M. and E.A.) performed most of the exams. All measurements were obtained offline using a dedicated software (ComPACS; MediMatic S.R.L., Genoa, Italy) by an experienced operator, blinded to the RHC results.

Right ventricular wall tension

RVWT, based on Laplace–Young’s law, was calculated as follows

\[
RVWT = \frac{TRPG \times RVD3}{C^2}
\]

TRPG represents transtricuspid pressure gradient, defined as the maximum gradient across the tricuspid valve, and it was acquired according to international guidelines.15,16 Multiple measurements were made from different echocardiographic windows and the highest peak gradient recorded was used in the analysis. RVD3 represents end-diastolic RV length: this index was preferred to other RV diameters because the systolic motion of the RV occurs mainly along its length, and therefore the RV motion vector runs more parallel to RVD3 than to other RV diameters. However, we are aware that this may be an oversimplification of the complex RV geometry. RVD3 was measured in the RV dedicated four-chamber-view, as suggested by current guidelines.15,16 RV wall thickness was not included in the traditional Laplace equation due to its significant inter- and intra-observer variability, which could harmfully confound the results.17

Follow-up

The participants in this study were retrospectively investigated for all-cause death via a dedicated software used in our centre (TrakCare, InterSystems Corporation, Cambridge, MA, USA); when an event could not be traced this way, a telephonic follow-up was performed. Follow-up was censored at death occurrence or at last contact with the patient, be it either clinical or by telephone.
Statistical analysis

Data were presented as mean ± standard deviation or as median and interquartile range when appropriate. The parametric distribution of variables was analysed using the Shapiro–Wilk test. Differences between means were examined using the Student t test or analysis of variance for normally distributed variables. The results of the Wilcoxon rank-sum test or the Kruskal–Wallis test were analysed for non-normally distributed variables. The relationship between the echocardiographic estimates and RHC values was tested using Pearson correlation and linear regression. Fisher’s R-to-Z transformation was used to examine the statistical significance of the differences between correlations. Kaplan–Meier cumulative survival was calculated. In order to assess the cut-off value for RVWT, a receiver operating characteristic (ROC) analysis was performed and Youden’s index was used. Two-tail p-value < 0.05 was considered statistically significant. Statistical analysis was conducted using SPSS, version 21.

Results

Study population

Two-hundred patients were enrolled in this study; 10 patients were excluded due to technical issues during catheterization, inconclusive catheterization results or because echocardiography could not be performed. One-hundred and ninety patients were included in the baseline data analysis; 157 (83%) patients underwent RHC for diagnostic purposes, 27 (14%) for known PH follow-up and 6 (3%) for cardiac transplantation follow-up. Basic anthropometric, haemodynamic and echocardiographic characteristics are summarized in Table 1. Mean age was 61.9 ± 13.7 years; 103 (54%) were male; and 21 (11%) had prior myocardial infarction. One-hundred and twenty-six (66%) years; 103 (54%) were male; and 21 (11%) had prior myocardial infarction. One-hundred and twenty-six (66%) patients were excluded due to technical issues during catheterization; 157 (83%) patients underwent RHC for diagnostic purposes; 23 patients (62%) had a novel diagnosis of pre-capillary PH patients, 14 (38%) patients were on PH therapies; 23 patients (62%) had a novel diagnosis of pre-capillary PH.

Echocardiographic parameters of RV diastolic function are summarized in Table 2; except for E/e’, isovolumetric relaxation time and deceleration time of the E wave, which were consistent with altered RV diastolic function, mean values were within normal limits according to guidelines. RVWT was associated with invasive assessment of RVEDP, with a R index equal to 0.343 (R²: 0.117, p < 0.001), as depicted in Fig. 1. Contrarily, correlation between validated echocardiographic methods to estimate RVEDP was not statistically significant, except for tricuspid E wave velocity (p = 0.002), as shown in Table 3. After Fisher’s R-to-Z transformation, the correlation between RVWT and RVEDP was significantly higher than the correlation between canonical parameters and RVEDP (all p < 0.01), except for tricuspid E wave velocity (p = 0.198). RVWT significantly correlated with several indexes of RV uncoupling, such as pulmonary vascular resistance (PVR), PA pulsatory

| Table 1. Demographic, haemodynamic and echocardiographic characteristics of the study population (n = 190). |
| Baseline features | Value |
| Age (years) | 61.85 ± 13.72 |
| Male sex | 103 (53.9%) |
| NYHA I–II | 144 (75.80%) |
| COPD | 10 (5.26%) |
| PH | 126 (66%) |
| Precapillary PH | 37 (29%) |
| Prior myocardial infarction | 21 (11.06%) |
| RHC parameters | Value |
| Cardiac output by thermodilution (l/min) | 5.35 ± 1.87 |
| mPAP (mmHg; n = 190) | 31.21 ± 12.35 |
| PVR (WU; n = 184) | 2.68 ± 2.22 |
| PAWP (mmHg; n = 190) | 17.70 ± 7.67 |
| RVSWI (g/m/beat; n = 178) | 8.27 ± 4.75 |
| Echocardiographic left heart parameters | Value |
| EF (%; n = 190) | 50.29 ± 16.21 |
| LVEDD (mm; n = 185) | 50.46 ± 9.79 |
| LVEDV (ml; n = 161) | 111.36 ± 79.08 |
| Lateral mitral S’ wave (cm/s; n = 184) | 7.27 ± 2.66 |
| Mean mitral E/A (n = 135) | 1.37 ± 1.79 |
| Mean mitral E/E’ (n = 169) | 13.85 ± 10.94 |
| Mild or moderate mitral regurgitation | 180 (94.8%) |
| Echocardiographic right heart parameters | Value |
| RV FAC (%; n = 150) | 41.43 ± 10.45 |
| TAPSE (mm; n = 183) | 20.31 ± 5.59 |
| TRPG (mmHg; n = 184) | 35.17 ± 17.64 |
| TAPSE/TRPG (mm/mmHg; n = 183) | 0.72 ± 0.41 |
| RVSWI (g/m/beat; n = 190) | 8.27 ± 4.75 |
| Mild or moderate tricuspid regurgitation | 176 (92.6%) |

Note: Values in brackets indicate the unit of measurement and number of patients for each variable.

NYHA: New York Heart Association; COPD: chronic obstructive pulmonary disease; PH: pulmonary hypertension; mPAP: mean pulmonary artery pressure; PVR: pulmonary vascular resistance; PAWP: pulmonary artery wedge pressure; RVSWI: right ventricle stroke work index; EF: ejection fraction; LVEDV: left ventricle end-diastolic volume; LVEDD: left ventricle end-diastolic diameter; TAPSE: tricuspid annulus plane systolic excursion; TRPG: tricuspid regurgitation peak gradient.

Haemodynamic comparison

RVWT was associated with invasive assessment of RVEDP, with a R index equal to 0.343 (R²: 0.117, p < 0.001), as depicted in Fig. 1. Contrarily, correlation between validated
pressure, PA compliance, right ventricular stroke work index (RVSWI) and the ratio between cardiac index (CI) and mean right atrial pressure (mRAP) (all \( p < 0.001 \)), as shown in Fig. 2 and Table 4.

RVWT was significantly higher in patients diagnosed with PH (2747 mmHg x mm vs 1422 mmHg x mm, \( p < 0.001 \)), as were TRPG (41.37 mmHg vs 23.26 mmHg, \( p < 0.001 \)) and RVD3 (66.95 mm vs 62.02 mm, \( p < 0.001 \)). Moreover, RVWT showed a slightly higher correlation with mean pulmonary artery pressure than TRPG and RVD3 alone (R: 0.74, 0.72 and 0.26, respectively, all \( p < 0.001 \)).

Follow-up and survival
Among the 190 patients who were included in the study, 31 (16%) were lost at follow-up, and 159 patients were included in the analysis. Mean follow-up was five years and three months; death was observed in 47 (30%) patients. Among the 27 patients (14%) who were enrolled for PH follow-up, 5 (19%) died at follow-up.

The 159 patients available for analysis were divided into two groups by the cut-off value of 1945 mmHg x mm, defined by low (RVWT < 1945 mmHg x mm) or high RVWT (RVWT > 1945 mmHg x mm), as calculated by Table 2.

Table 2. Validated echocardiographic parameters for evaluation of right ventricular diastolic function (n = 190).

| Characteristics | \( R^2 \pm \text{SD} \) |
|-----------------|-------------------------|
| E (m/s)         | 0.43 ± 0.157            |
| A (m/s)         | 0.43 ± 0.130            |
| E/A             | 0.97 ± 0.42             |
| E DcT (s)       | 0.75 ± 0.34             |
| IVRT (ms)       | 84.86 ± 34.25           |
| E' (cm/s)       | 9.19 ± 3.45             |
| A' (cm/s)       | 12.24 ± 4.08            |
| E'/A'           | 0.8267 ± 0.6259         |
| E/E'            | 5.17 ± 2.52             |

Note: Values are expressed as mean ± standard deviation.
E DcT: E wave deceleration time; IVRT: isovolumetric relaxation time.

Table 3. Correlation between echocardiographic parameters of right ventricular diastolic function and right ventricular end-diastolic pressure.

| Characteristics | \( R^2 \pm \text{SD} \) |
|-----------------|-------------------------|
| E (m/s)         | 0.25–0.06 0.002         |
| A (m/s)         | 0.08–0.01 0.388         |
| E/A             | 0.08–0.01 0.399         |
| E DcT (s)       | 0.01< 0.01 0.921        |
| IVRT (ms)       | 0.06< 0.01 0.499        |
| E' (cm/s)       | 0.08< 0.01 0.307        |
| A' (cm/s)       | 0.13–0.02 0.131         |
| E'/A'           | 0.12–0.02 0.159         |
| E/E'            | 0.03–0.01 0.714         |

E DcT: E wave deceleration time; IVRT: isovolumetric relaxation time.

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**Fig. 1.** Relationship between right ventricular end-diastolic pressure and right ventricular wall tension (n = 159).
RVWT: right ventricular wall tension; RVEDP: right ventricular end-diastolic pressure.
Youden’s index in conjunction with ROC analysis. Kaplan Meier analysis for overall survival showed a global $p$-value of 0.036 (OR: 1.67; Fig. 2). In patients with high RVWT, stratifying survival analysis by PH aetiology (pre vs post-capillary) did not result significant ($p=0.801$). Conversely, stratification of the study population by low or preserved tricuspid annulus plane systolic excursion (TAPSE), RV fractional area change or RV S’ according
RV end-diastolic area: 0.347–0.120
RV FAC: 0.382–0.146
Right atrium area: 0.204–0.042
RV medium diameter: 0.403–0.162
RV basal diameter: 0.326–0.106
PVR: 0.531–0.282
PA compliance: 0.449–0.202
RVSWI: 0.326–0.106
CI/mRAP: 0.209–0.044
mRAP: 0.326–0.106
PA pulsatory pressure: 0.740–0.547
mPAP: 0.742–0.550
RV differential pressure: 0.794–0.630
PA pulsatory pressure: 0.740–0.547

Discussion

PH prognosis strictly depends on the interplay between pulmonary circulation and the RV. The coupling between RV afterload, i.e. pulmonary circulation, and RV global function is defined by the ratio between RV end-systolic elastance and PA elastance (Ees/Ea), as previously described. During the first phases of PH, the RV maintains a pseudophysiological coupling at the cost of altered metabolism and increased RVEDP, thus leading to raised RVWT while patients are still asymptomatic and coupling is preserved.6–9

To our knowledge, this is the first study aiming to find an index able to estimate RV wall stress; few methods have been proposed to estimate left ventricular wall tension,20 but none had been assessed for the RV so far.

In our study, an increase in RVWT reflected into a significant increase of all-cause mortality: RVWT showed a significant prognostic impact with a cut-off of 1945 mmHg × mm, while traditional parameters of systolic RV function could not significantly predict prognosis. This might be due to the characteristics of our population, consisting of patients without PH or at an early/intermediate phase of the disease, when the RV still performs normally or near normally, and therefore canonical parameters of impaired systolic function might not be significantly altered, while the RV silently starts to strain against growing PVR. We would also like to stress the fact that TRPG could not significantly stratify prognosis with a cut-off value of 30 mmHg; however, only 26 patients with TRPG < 30 mmHg underwent RHC and a net, albeit non-significant, separation can be seen between the two curves of TRPG (p = 0.107, Fig. 3). This, however, stresses the concept that RVWT might have a significant impact on PH prognosis, which might be more meaningful than TRPG alone, denoting an effective and silent RV derangement.

Several limits must be addressed. First, in the absence of a ‘gold standard’ method to estimate RVWT, we had to rely on indirect hemodynamic indexes of early RV diastolic (and systolic) dysfunction, such as RVEDP. Furthermore, using RVD3 as the only diameter in the formula for RVWT calculation is an oversimplification of the complex RV anatomy; nevertheless, as previously explained, since RV contraction occurs mainly on the longitudinal axis, RV length, which runs parallel to this axis, was chosen to find RVWT. Moreover, a significant association was consistently found between the other RV diameters and RVWT in several sub-analyses not presented in this paper. Another limitation of the present study is the inability to trace back the specific cause of death; however, it may be inferred that an excess of all-cause mortality in this population could be largely driven by cardiovascular death due to the relatively high prevalence of PH (66%).

Despite its limitations, this method may be the first step in the research of predictors of early RV stress in a population where the diagnosis must be as premature as possible. We strongly believe that a deeper analysis of RV stress could have a huge impact in this field of research, be it either with magnetic resonance imaging or by invasive haemodynamic assessment, which could plot a pressure/volume loop to derive Ees/Ea.21 Our method, however, is simple, bed-side disposable and cheap, and it could identify patients who may require further analyses and strict follow-up, due to the fact that the patient’s RV may be facing a subtle, but risky, increase of wall tension.
Conclusions

In conclusion, we identified a novel bedside echocardiographic predictor of altered RV haemodynamics, which is precociously altered in patients without overt RV failure and is associated to all-cause mortality at a long-term follow-up. Further studies are needed to confirm its role in PH patients, primarily with a prospective and not a retrospective analysis. We strongly hope that in the near future, new and more precise methods to identify silent RV failure will be conceived.

Fig. 3. Kaplan–Meier curves describing cumulative survival for all-cause mortality according to right ventricular wall tension (RVWT) (top left panel), tricuspid annular plane systolic excursion over tricuspid regurgitation peak gradient (TAPSE/TRPG) (top right panel), TRPG (middle left panel), TAPSE (middle right panel), tricuspid S’ wave (bottom left panel) and right ventricle fractional area change (RV FAC) (bottom right panel).
Authors’ contributions
Each author made substantial contributions to the conception, the design of the work, the interpretation of data, revised the present article and approved the submitted version. Each author has agreed both to be personally accountable for the author’s own contributions and to ensure that questions related to the accuracy or integrity of any part of the work, even ones in which the author was not personally involved, are appropriately investigated, resolved and the resolution documented in the literature.

Availability of data and material
The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Conflict of interest
The author(s) declare that there is no conflict of interest.

Ethics approval and consent to participate
The present study was approved by the local ethics committee and it was performed according to the principles of the Declaration of Helsinki. All patients provided written informed consent.

Funding
This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

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