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Predictive value of simple echocardiographic parameters for screening pulmonary hypertension under the revised definition: a study for general hospitals

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ABSTRACT

Background The current guideline recommends a peak tricuspid regurgitation velocity (TRV) ≥2.9 m/s on echocardiography for pulmonary hypertension (PH) screening; however, this threshold was based on the previous PH definition (mean pulmonary arterial pressure (mPAP) ≥25 mm Hg) and derived largely from PH referral centres.

Methods We retrospectively analysed 755 patients who underwent both transthoracic echocardiography and right heart catheterisation at two general hospitals. The discrimination of peak TRV and estimated right atrial pressure (eRAP), derived from inferior vena cava diameter and respiratory variation, for screening for PH was assessed by receiver operating characteristic curve analysis. Optimal cut-off values were determined with the Youden Index.

Results The c-statistic for peak TRV in detecting PH was 0.82 (95% CI 0.79 to 0.85). An optimal cut-off of 2.7 m/s provided higher sensitivity (72%) than the conventional 2.9 m/s threshold (60%) while maintaining high specificity (82%). In 681 patients with available TRV and eRAP data, adding eRAP improved discrimination compared with TRV alone (c-statistic 0.83 vs 0.80; net reclassification improvement=0.14, p=0.002). eRAP ≥5 mm Hg was associated with a higher risk of PH, and the combination of elevated TRV and eRAP yielded the strongest association.

Conclusion For screening under the revised PH definition, a peak TRV of 2.7 m/s is suggested as the optimal cut-off. Although TRV alone showed good discriminative performance, combining it with eRAP further improved diagnostic accuracy using simple echocardiographic measures.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ A peak tricuspid regurgitation velocity threshold of 2.9 m/s is recommended for screening for pulmonary hypertension, but this cut-off value was established under the previous definition and mainly in referral-centre populations.

WHAT THIS STUDY ADDS

⇒ In a general-hospital population evaluated under the revised diagnostic definition, a lower threshold of 2.7 m/s improved screening performance and adding estimated right atrial pressure derived from inferior vena cava measurements further enhanced risk stratification.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ Adopting these echocardiographic criteria may enable earlier identification and referral of patients with pulmonary hypertension in routine clinical practice.

INTRODUCTION

Pulmonary hypertension (PH) is a progressive and life-threatening disease characterised by elevated pulmonary arterial pressure.[1] In the absence of timely treatment, PH ultimately leads to right ventricular failure and severe complications, including sudden cardiac death and increased mortality.[2 3] Historically, PH was defined as a mean pulmonary arterial pressure (mPAP) ≥25 mm Hg, as measured by right heart catheterisation (RHC).[4] However, accumulating evidence indicates that even modest elevations in mPAP (>20 mm Hg) are associated with adverse outcomes and increased mortality risk.[1] This risk is particularly evident in patients with underlying lung disease or connective tissue disorders, in whom modest pressure elevations may signify a clinically meaningful stage.[5 6] Consequently, the 2022 guideline from the European Society of Cardiology (ESC) and the European Respiratory Society (ERS) define PH as mPAP >20 mm Hg, highlighting the need to refine non-invasive screening strategies to enable earlier detection and timely therapeutic intervention.[7]

The current guidelines recommend echocardiography as a central tool in PH screening. Among echo parameters, peak tricuspid regurgitation velocity (TRV) ≥2.9 m/s is used as a key criterion for determining which patients should be referred to a PH centre. Although this cut-off value of 2.9 m/s was established under the previous PH definition (ie, mPAP ≥25 mm Hg), the current guidelines continue to recommend the same peak TRV threshold even after the mPAP threshold for PH was lowered. However, several studies have re-evaluated the optimal TRV threshold under the revised definition, and no consistent conclusion has yet been reached. One study recommends lowering the cut-off value of peak TRV,[8] whereas others support maintaining the current threshold of 2.9 m/s.[9 10] Therefore, the optimal cut-off value remains uncertain. There are also a few caveats in the previous reports. First, most studies were limited to patients referred to PH centres—populations who completed screening and had a high PH probability. Evidence from general hospital settings, where patients present with a broader spectrum of disease, is lacking. Second, the interval between echocardiography and RHC was approximately 4 weeks, which may have led to an inaccurate evaluation of the association between peak TRV and mPAP. To address this gap, our study aims to evaluate echocardiographic performance in screening for PH under the revised diagnostic definition of mPAP >20 mm Hg in multiple non-PH centres.

STUDY DESIGN AND METHODS

Study design and participants

This was a retrospective, multicentre, observational study. In this study, general hospitals were defined as institutions without specialised PH centres; specifically, they lacked a multidisciplinary team, direct links and rapid referral pathways to other specialised services, such as genetic counselling, pulmonary endarterectomy, balloon pulmonary angioplasty, lung transplantation and adult congenital heart disease services, as described in the 2022 ESC/ERS guidelines.[7]

We retrospectively studied 1714 patients who underwent both transthoracic echocardiography and RHC at Okayama University Hospital (January 2018–December 2023) and Kure Kyosai Hospital (January 2020–December 2023). Of them, we excluded 668 patients with an interval between echocardiography and RHC ≥6 days, 17 patients aged younger than 18 years, 239 patients with missing values in peak TRV or mPAP data and 35 patients with valvular or congenital heart disease including pulmonary valve stenosis, tetralogy of Fallot, single-ventricle physiology, right ventricular outflow tract obstruction, transposition of the great arteries and congenitally corrected transposition, which causes impairment in the correlation between TRV and pulmonary arterial pressure.[11] A total of 755 patients were included in the final analysis (figure 1). Since our aim was to assess the relationship between peak TRV and mPAP, we analysed the initial assessment by echocardiography and RHC during the study period when multiple examinations were performed.

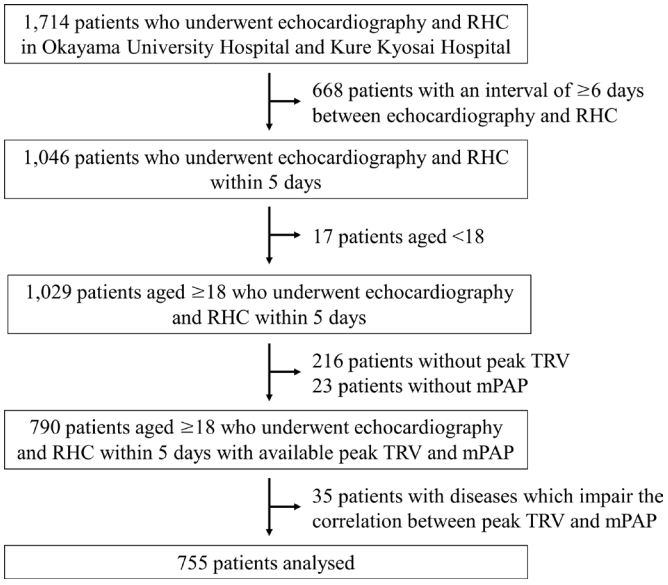

Figure 1 Flow chart of the study population. Flow diagram illustrating patient selection at Okayama University Hospital and Kure Kyosai Hospital. Of 1714 patients initially identified, 755 patients were included in the final analysis. mPAP, mean pulmonary arterial pressure; RHC, right heart catheterisation; TRV, tricuspid regurgitation velocity.

The study was approved by the institutional review boards of both institutions (approved number, Okayama University Hospital: #2410-034; Kure Kyosai Hospital: #kk202510). The informed consent was waived because of the low-risk nature of this retrospective study and the inability to obtain consent directly from all subjects. Instead, the study protocol was publicly disclosed on the website (http://www.hsc.okayama-u.ac.jp/ethics/koukai/med/), allowing patients to opt out. The study was conducted according to the principles expressed in the Declaration of Helsinki.

Patient and public involvement

Patients and/or members of the public were not involved in the design, conduct, reporting or dissemination plans of this research. This retrospective observational study used routinely collected clinical data, and there was no direct recruitment of participants.

Echocardiographic assessment

Two-dimensional and Doppler echocardiographic examinations were performed using one of the following machines: ALOKA (Aplio Verifia, Aplio i700/i800/i900), Artida, Epic, Vivid E9, S6 or S9 in Okayama University Hospital and Vivid 7, E90, E95, S60 or S70 in Kure Kyosai Hospital. All measurements adhered to guidelines from the American Society of Echocardiography and the European Association of Cardiovascular Imaging.[12–14] Right atrial pressure was estimated from inferior vena cava (IVC) diameter and respiratory variation during inspiration. Based on the guideline from the American

Society of Echocardiography, estimated right atrial pressure (eRAP) was categorised into three groups using the cut-off value of IVC diameter (≤21 mm or >21 mm) and respiratory variation (≥50% or <50%): 0–5 mm Hg (IVC ≤21 mm and collapse ≥50%); 5–10 mm Hg (IVC ≤21 mm and collapse <50% or IVC >21 mm and collapse ≥50%); 10–20 mm Hg (IVC >21 mm and collapse <50%).[15] Interobserver and intraobserver differences for peak TRV were analysed in 50 randomly selected cases. Peak TRV was measured by two blinded observers and by a single observer at two different times. Interobserver agreement and intraobserver consistency were calculated using intraclass correlation coefficients and a 95% CI.

Haemodynamic assessment and PH definition

PH was defined as mPAP >20 mm Hg by RHC at rest. As mentioned in Study design and participants section, we analysed RHC data obtained within 5 days of echocardiographic examination. Haemodynamic parameters were recorded, including systolic pulmonary arterial pressure, mPAP, mean right atrial pressure, pulmonary arterial wedge pressure (PAWP), mean left atrial pressure (if applicable), cardiac output, cardiac index and heart rate.

Pulmonary vascular resistance (PVR) was calculated using the following formula:

$$PVR = \frac{(mPAP - PAWP^{*})}{Cardiac\ output}$$

*Otherwise left atrial pressure.

In patients with intracardiac shunts (eg, atrial septal defect), both PAWP and left atrial pressure were measured when feasible; the higher value was used for analysis.

Covariates

Clinical information, including diagnoses and comorbidities, was obtained from the Diagnosis Procedure Combination database at Okayama University Hospital and medical records at Kure Kyosai Hospital. We also collected data on underlying diseases based on codes of International Classification of Diseases, 10th Revision, including pulmonary valve stenosis (I37.0 and I37.2), congenital heart diseases (Q20-28), lung diseases (J43, J44, J80-84 and G47.3), connective tissue diseases (M30-36) and chronic thromboembolic PH (I27.2).

Statistical analysis

Continuous variables are presented as medians (IQR) and categorical variables as counts (percentages). Correlation between peak TRV and both systolic and mPAP was assessed with Pearson's correlation coefficient. Logistic regression models were run to estimate OR and 95% CI according to peak TRV value. To identify the threshold value of peak TRV for PH risk, we also modelled TRV as a continuous variable with restricted cubic spline terms with 1.8 m/s as the reference value[16] and four knots at fifth, 35th, 65th and 95th percentiles of TRV.

We evaluated diagnostic performance using receiver operating characteristic curve analysis and determined the optimal TRV cut-off using the Youden Index. To address potential confounding due to differences in baseline age and sex between groups, we performed 1:1 propensity score matching based on age and sex. Propensity scores were estimated by logistic regression and nearest-neighbour matching without replacement was applied. Covariate balance before and after matching was assessed using standardised mean differences. In the matched cohort, the diagnostic performance of peak TRV for PH was reassessed using receiver operating characteristic curve analysis and the optimal cut-off value was again determined using the Youden Index. To internally validate the optimal cut-off value of peak TRV, we performed repeated 10-fold cross-validation. In each iteration, the dataset was randomly divided into 10 folds; nine folds were used as the training set and the remaining fold as the test set. This procedure was repeated 100 times, yielding a total of 1000 train-test evaluations. To assess the impact of the revised PH definition on the optimal cut-off value of peak TRV, we also performed the same cross-validation analysis using the previous PH definition.

Next, we evaluated whether adding eRAP to peak TRV improved the screening performance for PH diagnosis defined by mPAP >20 mm Hg. First, we assessed the correlation between right atrial pressure measured by RHC and mPAP. Then, logistic regression models were constructed to evaluate the association between right atrial pressure measured by RHC and the risk of PH. Then, we compared model performance between peak TRV alone and peak TRV+eRAP using the c-statistic values, DeLong test and net reclassification improvement. In addition, to internally validate the combined model, we performed repeated 10-fold cross-validation in patients with complete data for peak TRV and eRAP. Within this complete-case cohort, we evaluated both the peak TRV-alone model and the model incorporating both peak TRV and eRAP and summarised their test-set discriminative performance. We further assessed the joint association of peak TRV and eRAP with the risk of PH using a cross-categorisation analysis based on threshold values that showed a significant association with PH risk in our original analysis.

Statistical significance was set at two-sided p<0.05. Analyses were conducted using R V.4.4.1 (The R Foundation for Statistical Computing, Vienna, Austria).

RESULTS

Patient characteristics

Baseline clinical characteristics are summarised in table 1. Among the study cohort, 366 patients (48%) were diagnosed with PH. All patients underwent RHC for one of the following indications: (1) evaluation of heart failure, (2) suspected PH or (3) underlying diseases associated with an increased risk of PH, such as congenital heart disease. Compared with those without PH, patients with PH exhibited significantly higher right-sided and left-sided filling pressures, including right atrial pressure and PAWP, as well as lower cardiac output. There was no

Table 1 Baseline clinical characteristics

	Available data, n	Overall	mPAP ≤20 mm Hg	mPAP >20 mm Hg	P value
n, (%)		755 (100)	389 (52)	366 (48)	
Age, years		68 (54–76)	67 (51–76)	69 (57–77)	0.044
Female, n (%)		313 (42)	175 (45)	138 (38)	0.046
Haemodynamic data					
Heart rate, beats per minute	697	70 (60–80)	65 (60–75)	74 (64–86)	<0.001
Systolic blood pressure, mm Hg	653	126 (111–146)	126 (110–146)	131 (108–150)	0.171
Diastolic blood pressure, mm Hg	653	68 (58–78)	64 (55–74)	70 (60–82)	<0.001
RAP, mm Hg	750	5 (3–7)	4 (2–5)	6 (4–9)	<0.001
sPAP, mm Hg	755	32 (24–43)	25 (21–29)	44 (37–58)	<0.001
mPAP, mm Hg	755	20 (15–28)	15 (13–18)	28 (24–36)	<0.001
PAWP or LAP, mm Hg	706	9 (6–13)	7 (5–10)	12 (8–18)	<0.001
(PAWP or LAP) >15 mm Hg, n (%)		126 (17)	3 (0.8)	123 (33.6)	<0.001
Cardiac output, L/min	681	3.8 (3.1–4.8)	4.0 (3.2–4.8)	3.6 (2.8–4.8)	0.006
Cardiac index, L/min/m^2	679	2.4 (1.9–2.9)	2.5 (2.1–2.9)	2.3 (1.9–2.8)	0.002
PVR, Wood Units	725	2.3 (1.4–4.0)	1.7 (1.1–2.5)	3.8 (2.2–6.8)	<0.001
Echocardiographic parameters					
LVEF, %	681	62 (50–69)	62 (51–67)	63 (46–69)	0.342
Peak TRV, m/s	755	2.6 (2.3–3.1)	2.4 (2.2–2.6)	3.0 (2.6–3.6)	<0.001
IVC diameter, mm	753	12 (9–16)	11 (8–14)	14 (10–19)	<0.001
IVC respiratory variation, %	681	58 (44–67)	63 (50–70)	53 (36–67)	<0.001
WHO functional class*, n					
I/II/III/IV	750	201/295/212/42	164/148/69/5	37/147/143/37	
Laboratory data					
Haemoglobin, g/L	755	131 (118–143)	133 (121–144)	129 (112–141)	0.002
BNP, pg/mL	700	109 (36–316)	79 (26–174)	184 (52–517)	<0.001
Creatinine, mg/dL	755	0.8 (0.7–1.1)	0.8 (0.7–1.0)	0.9 (0.7–1.1)	0.023
eGFR, mL/min/1.73 m^2	755	62 (46–78)	64 (48–82)	59 (43–74)	<0.001
CT-based anatomical parameters					
Main pulmonary artery diameter, mm	529	29 (25–34)	27 (24–30)	31 (28–36)	<0.001
MPA/AA ratio	529	0.9 (0.8–1.1)	0.9 (0.8–1.0)	1.0 (0.9–1.1)	<0.001
Medical therapy					
Diuretics, n (%)	755	323 (43)	134 (34)	189 (52)	<0.001
ACE-I or ARB or ARNI, n (%)	755	253 (34)	137 (35)	116 (32)	0.316
Beta-blockers, n (%)	755	304 (40)	166 (43)	138 (38)	0.188
Aldosterone antagonist, n (%)	755	238 (32)	106 (27)	132 (36)	0.011
SGLT2 inhibitor, n (%)	755	70 (9)	35 (9)	35 (10)	0.887
Oral PH therapy, n (%)	755	99 (13)	24 (6)	75 (21)	<0.001
Parenteral prostacyclin therapy, n (%)	755	14 (2)	2 (1)	12 (3)	0.006
Underlying diseases					
Left heart diseases, n (%)	755	361 (48)	190 (49)	171 (47)	0.406
Congenital heart diseases, n (%)	755	181 (24)	126 (32)	55 (15)	<0.001
Lung diseases, n (%)	755	126 (17)	36 (9)	90 (25)	<0.001
Connective tissue diseases, n (%)	755	64 (8)	24 (6)	40 (11)	0.02
CTEPH, n (%)	755	78 (10)	14 (4)	64 (17)	<0.001

Summary of clinical, haemodynamic and echocardiographic parameters in patients with and without PH. Data are expressed as median (IQR) for continuous variables and n (%) for categorical variables. Comparisons were made using Wilcoxon rank-sum test for continuous variables and χ^2 test for categorical variables.
*WHO functional class is typically used in patients with PH; however, in the present study, functional status was classified according to criteria analogous to WHO functional class in all patients, regardless of PH status.
AA, ascending aorta; ACE-I, ACE inhibitor; ARB, angiotensin II receptor blocker; ARNI, angiotensin receptor-neprilysin inhibitor; BNP, B type natriuretic peptide; CTEPH, chronic thromboembolic pulmonary hypertension; eGFR, estimated glomerular filtration rate; IVC, inferior vena cava; LAP, left atrial pressure; LVEF, left ventricular ejection fraction; MPA, main pulmonary artery; mPAP, mean pulmonary arterial pressure; PAWP, pulmonary arterial wedge pressure; PH, pulmonary hypertension; PVR, pulmonary vascular resistance; RAP, right atrial pressure; SGLT2, sodium-glucose cotransporter 2; sPAP, systolic pulmonary arterial pressure; TRV, tricuspid regurgitation velocity.

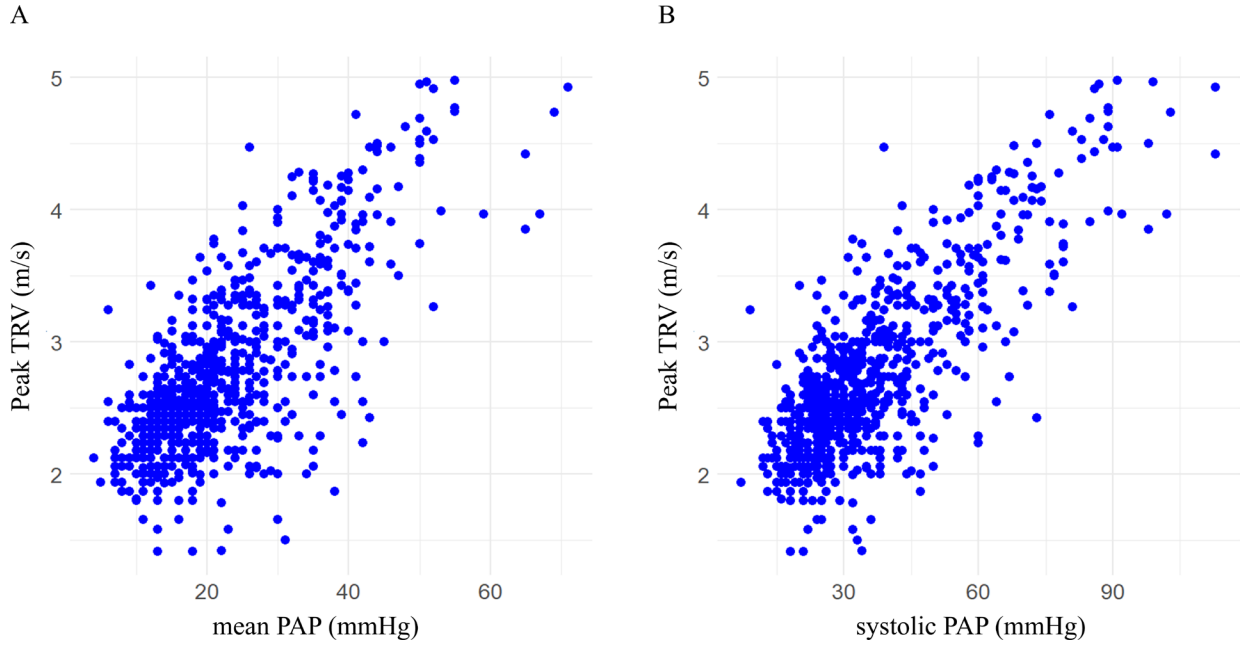

Figure 2 Correlation between pulmonary arterial pressures and peak TRV. Scatter plots demonstrate the association between peak TRV and (A) mean PAP and (B) systolic PAP, with significant correlations (r=0.73 and r=0.79; p<0.001 and p<0.001, respectively). PAP, pulmonary arterial pressure; TRV, tricuspid regurgitation velocity.

significant difference in left ventricular ejection fraction between the two groups. Patients with PH showed significantly larger IVC diameter and lower respiratory variation, consistent with elevated right atrial pressure. Regarding medical therapy, patients with PH were more likely to receive diuretics and aldosterone antagonists, whereas the use of most other standard medical therapies for left-sided heart failure, such as beta-blockers, was comparable between groups. Congenital heart disease was present in 24% of the cohort (181/755), mainly atrial septal defect (176/181 cases), but these patients showed relatively lower PH prevalence.

Screening performance of peak TRV

In the overall cohort, peak TRV correlated moderately with both mPAP (r=0.73, p<0.001) and systolic PAP (r=0.79, p<0.001) (figure 2A,B). Correlation strength varied by peak TRV strata: patients with TRV ≥3.4 m/s showed a moderate correlation with mPAP (r=0.66, p<0.001), those with TRV between 2.9 m/s and 3.4 m/s exhibited only a weak and non-significant correlation (r=0.12, p=0.12), and those with TRV<2.9 m/s demonstrated a weak but statistically significant correlation (r=0.21, p<0.001). The reproducibility of peak TRV measurements was excellent. The intraobserver intraclass correlation coefficient was 0.95 (95% CI 0.91 to 0.97), and the interobserver intraclass correlation coefficient was 0.96 (95% CI 0.92 to 0.97).

Restricted cubic spline curve revealed a nonlinear association between peak TRV and PH risk, with a steep increase of PH risk at peak TRV of 2.7 m/s (OR 2.12, 95% CI 1.09 to 4.13, figure 3).

The discriminative ability of peak TRV, assessed by the c-statistic (area under the curve (AUC)), was 0.82 (95% CI 0.79 to 0.85). At the guideline-recommended cut-off of peak TRV ≥2.9 m/s, the specificity for diagnosing PH was high (93%), while sensitivity remained modest (60%). The Youden Index identified an optimal peak TRV cut-off of 2.7 m/s, yielding improved sensitivity (72%) while maintaining high specificity (82%) (table 2 and figure 4A). We additionally performed 1:1 propensity

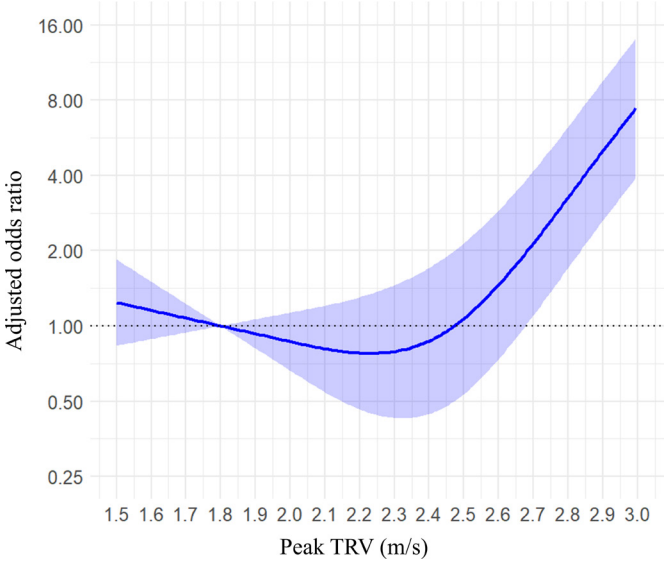

Figure 3 Restricted cubic spline curve for peak TRV and PH risk. Restricted cubic spline curve showed the adjusted OR (blue line) and 95% CI (blue shades) for PH according to peak TRV (peak TRV 1.8 m/s as the reference value (OR=1.0)), with a non-linear association between peak TRV and PH. A steep increase of PH risk at peak TRV of 2.7 m/s was observed (OR 2.12, 95% CI 1.09–4.13). PH, pulmonary hypertension; TRV, tricuspid regurgitation velocity.

Table 2 The predictive performance of different cut-off values of peak tricuspid regurgitation velocity (TRV) for pulmonary hypertension

Peak TRV, m/s (TRPG, mm Hg)	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)	Accuracy (%)
2.4 (23)	85	50	62	79	67
2.5 (25)	82	56	64	77	69
2.6 (27)	75	74	73	76	75
2.7 (29)	72	82	79	76	77
2.8 (31)	65	87	83	73	77
2.9 (34)	60	93	88	71	77
3.0 (36)	56	94	90	69	75

NPV, negative predictive value; PPV, positive predictive value; TRPG, tricuspid regurgitation pressure gradient.

score matching using age and sex. This yielded 366 matched pairs. After matching, the imbalance in baseline age and sex was substantially reduced (standardised mean differences: age 0.10, sex 0.08). In the matched cohort, peak TRV retained good discriminative ability for PH, with the c-statistic of 0.82 (95% CI 0.78 to 0.85), and the optimal cut-off remained 2.7 m/s.

The currently recommended threshold of peak TRV at 2.9 m/s demonstrated high specificity but limited sensitivity for detecting PH. In contrast, the optimal threshold of 2.7 m/s provided improved sensitivity while maintaining high specificity.

Cross-validation

In the repeated 10-fold cross-validation analysis, the mean optimal cut-off value for peak TRV was 2.7 m/s (SD, 0.05). The distribution of selected cut-off values was narrow, with a median of 2.7 m/s. When this internally derived cut-off was applied to the test sets, the mean sensitivity was 0.71 and the mean specificity was 0.83. The mean positive predictive value was 0.80, the mean negative predictive value was 0.75 and the mean accuracy was 0.77. The mean test-set c-statistic was 0.82, indicating stable discriminative performance across repeated validation samples. Under the previous PH definition, the mean optimal cut-off value for peak TRV was 2.9 m/s (SD, 0.03), and the median selected cut-off was likewise 2.9 m/s. The 2.5th–97.5th percentile range was 2.9–2.9 m/s. The mean test-set c-statistic was 0.77, accompanied by lower sensitivity (0.60) and higher specificity (0.87).

Improved screening using eRAP

We further evaluated whether integrating echocardiographic eRAP could enhance the screening performance for PH. In our cohort, right atrial pressure measured by RHC and mPAP showed a modest correlation (r=0.39, p<0.001). Logistic regression analysis demonstrated that right atrial pressure measured by RHC was significantly associated with PH, with an OR of 1.30 per 1 mm Hg increase (95% CI 1.24 to 1.38, p<0.001). In 681 patients

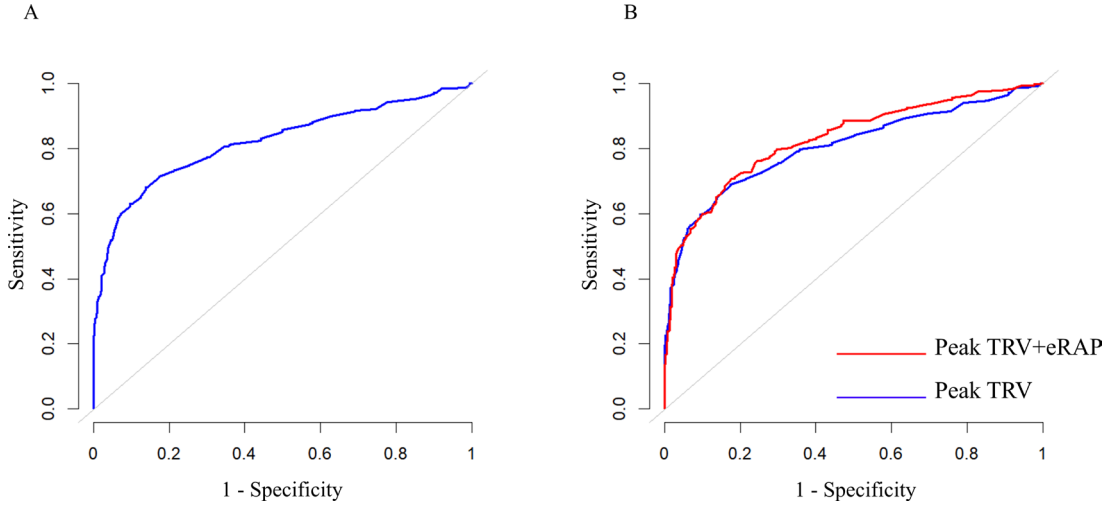

Figure 4 ROC curve analysis of different cut-off values of peak TRV and eRAP for screening PH. ROC curve of peak TRV and PH diagnosis shows the c-statistic (area under the curve) of 0.82 (95% CI 0.79 to 0.85) (A). Comparison of ROC curves between peak TRV alone and in combination with eRAP for PH diagnosis (B). The combined model yielded a higher c-statistic (0.83, 95% CI 0.80 to 0.86) than TRV alone (0.80, 95% CI 0.77 to 0.84), with a net reclassification improvement of 0.14 and a significant difference by DeLong test (p=0.002). eRAP, estimated right atrial pressure; PH, pulmonary hypertension; ROC, receiver-operating characteristic; TRV, tricuspid regurgitation velocity.

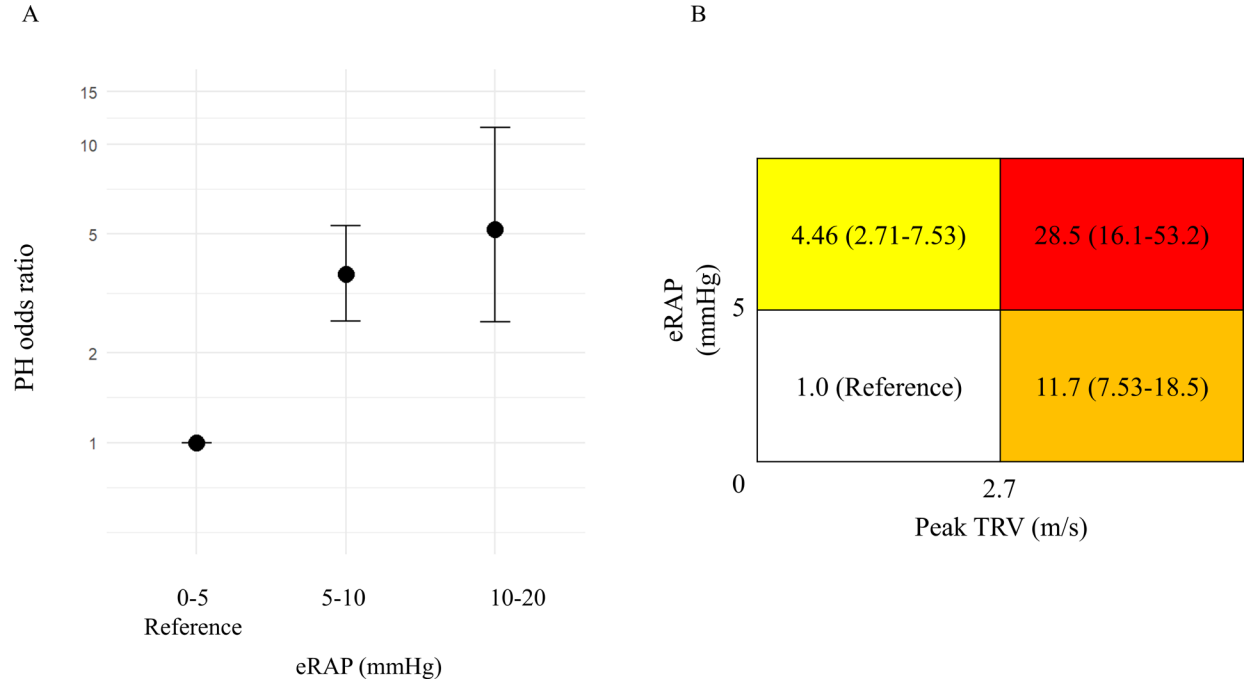

Figure 5 Association between eRAP and PH risk. (A) ORs for PH across eRAP categories of 5–10 mm Hg and 10–20 mm Hg compared with 0–5 mm Hg as the reference. The odds of PH were 3.67 (95% CI 2.55 to 5.32) at 5–10 mm Hg and 5.18 (95% CI 2.54 to 11.4) at 10–20 mm Hg, indicating a higher risk of PH, particularly at eRAP ≥5 mm Hg. (B) Cross-categorisation table shows the association of peak TRV and eRAP with PH risk. Each cell presents the OR and 95% CI. The cells are highlighted depending on the OR (≤1.00, white; 1.01–10.0, yellow; 10.1–20.0, orange and ≥20.1, red). The subgroup with eRAP ≥5 mm Hg and peak TRV ≥2.7 m/s showed the highest risk of PH (OR 28.5, 95% CI 16.1 to 53.2). eRAP, estimated right atrial pressure; PH, pulmonary hypertension; TRV, tricuspid regurgitation velocity.

with both peak TRV and eRAP data, adding eRAP to TRV improved predictive performance for PH compared with TRV alone (c-statistic 0.83, 95% CI 0.80 to 0.86 vs 0.80, 95% CI 0.77 to 0.84; p=0.002), with a net reclassification improvement of 0.14 (figure 4B). In repeated 10-fold cross-validation, the combined model retained good discriminative performance, with a mean test AUC of 0.83, indicating that the incremental value of eRAP was preserved after internal validation.

In the subgroup analysis stratified by underlying disease, adding eRAP to TRV significantly improved predictive performance in patients with left heart disease, whereas no significant improvement was observed in patients with other underlying conditions, including lung diseases, congenital heart diseases and connective tissue diseases (online supplemental figures 1 and 2). Although the addition of other parameters to TRV, such as B-type natriuretic peptide and main pulmonary artery diameter, also improved discriminative ability, their incremental benefit was comparable to or smaller than that provided by eRAP (online supplemental figure 3).

When compared with an eRAP of 0–5 mm Hg as the reference category, eRAP of 5–10 mm Hg and 10–20 mm Hg were significantly associated with PH risk, as the odds of PH were 3.67 (95% CI 2.55 to 5.32) at 5–10 mm Hg and 5.18 (95% CI 2.54 to 11.4) at 10–20 mm Hg (figure 5A). The cross-categorisation analysis revealed that elevations in either eRAP or peak TRV were associated with a higher risk of PH, while simultaneous elevation of both parameters was significantly associated with PH, with an OR of 28.5 (95% CI 16.1 to 53.2) compared with the reference group (eRAP <5 mm Hg and peak TRV <2.7 m/s) (figure 5B).

DISCUSSION

This study assessed the screening performance of echocardiographic parameters for PH under the revised diagnostic definition of mPAP >20 mm Hg in general hospitals. The main findings were: (1) PH risk increased significantly at a TRV threshold of 2.7 m/s, (2) this threshold provided a better balance of sensitivity and specificity than the conventional 2.9 m/s cut-off and (3) combining eRAP with TRV further improved discrimination and risk stratification.

Importantly, our study population included all patients who underwent RHC and echocardiography, regardless of PH suspicion, thereby reflecting real-world screening conditions. The 2022 ESC/ERS guideline recommends initial assessment using peak TRV, followed by evaluation of additional echocardiographic signs to estimate PH probability. Thus, we first analysed the correlation between peak TRV and mPAP across the entire population. Previous studies examining optimal TRV thresholds after the revised definition were conducted primarily in PH referral centres. Consequently, these studies enrolled populations with extremely high PH prevalence—approximately 80% in one cohort and 91% in

another—resulting in a substantially elevated pre-test probability.[9 10] Moreover, the haemodynamic severity in these cohorts was considerable, with reported mean mPAP values around 31 mm Hg and 36 mm Hg, respectively. Within these high-risk and haemodynamically advanced populations, a TRV threshold near 2.9 m/s generally achieved high sensitivity and acceptable specificity, supporting its utility for confirming PH in patients already suspected of having the disease. In contrast, our study evaluated a broader and more heterogeneous population, typified by a substantially lower PH prevalence (48%) and a considerably milder haemodynamic profile (median mPAP 20 mm Hg). When applied to this lower-risk, real-world cohort, the same TRV threshold demonstrated a marked decline in diagnostic sensitivity (60%), indicating that its performance is substantially attenuated in general screening settings. Taken together, these findings suggest that TRV cut-offs derived from referral centre-based, high-prevalence cohorts may not be fully generalisable to routine clinical populations with lower PH probability. Additionally, in the analysis using the previous PH definition, the optimal peak TRV cut-off was 2.9 m/s, which was consistent with the threshold recommended for screening of previously defined PH. In contrast, under the revised PH definition, a lower cut-off yielded better screening performance. Therefore, the difference between our findings and previous reports may be explained by both the distinct clinical setting of our study and the change in the haemodynamic definition of PH. Our results indicate that it may be appropriate to reassess TRV-based screening thresholds in settings where PH is less prevalent and haemodynamic impairment is less advanced.

Our results support lowering the TRV threshold to 2.7 m/s for screening in non-specialised centres. Although specificity declined modestly (from 93% to 82%), sensitivity improved meaningfully, thereby reducing the likelihood of missing patients with early disease. This is clinically important, as even mild elevations of systolic pulmonary artery pressure (>30 mm Hg)—which may be detected with a peak TRV of 2.7 m/s but missed at 2.9 m/s—is associated with a 5-year mortality rate of 25–40%.[17] Therefore, identifying patients with suspected pulmonary pressure elevation, even in the absence of typical symptoms, remains clinically important. Recent PH registry data also support a 2.7 m/s threshold for screening for PH, with a 95% sensitivity and 0.12 negative likelihood ratio,[8] consistent with our findings.

Although peak TRV has been shown to correlate with pulmonary artery pressure in population-based studies, its utility for estimating pulmonary artery pressure at the individual level may be limited due to moderate precision.[18 19] Moreover, a previous study reported that peak TRV does not accurately correlate with invasively measured systolic pulmonary artery pressure at low pressures.[9] Consistent with the previous report, our data showed a weak correlation between TRV and mPAP at lower peak TRV values, suggesting the need for additional parameters to detect early-stage PH. This diminished correlation might be one of the reasons why solely lowering the peak TRV threshold does not always enhance screening performance.[9 17] To address these limitations, several echocardiographic parameters—such as right ventricular outflow tract acceleration time and right atrial size—have been proposed to complement peak TRV.[8 9] However, these measures may be constrained by heart rate dependency and technical challenges in accurately acquiring right atrial images.[20 21] To complement TRV-based assessment, IVC-derived eRAP has emerged as a potential complementary parameter for PH screening.

IVC-derived eRAP does not strongly correlate with invasively measured right atrial pressure,[18 22] yet it may provide physiologically distinct and complementary information to TRV. TRV primarily reflects the pressure gradient across the tricuspid valve, whereas eRAP reflects systemic venous congestion; thus, the two parameters capture different components of right-sided haemodynamics. For example, in heart failure, a markedly dilated IVC may coexist with normal TRV values due to the reservoir function of the IVC buffering volume overload.[23 24] In such cases, eRAP captures congestion that TRV alone may fail to detect. Importantly, IVC measurements do not require complex imaging acquisition and exhibit high reproducibility, with low intraobserver and interobserver variability.[25] Taken together, IVC-derived eRAP may serve as a useful complementary parameter in TRV-based screening for early-stage PH in general clinical practice. Our findings support this complementary role: combining TRV with eRAP improved PH detection, and elevated eRAP increased PH likelihood even when TRV remained subthreshold.

This study has several limitations. First, we did not account for clinical context—such as disease severity or urgency—which may influence pre-test probability and echocardiographic interpretation. Second, although the reproducibility of peak TRV was excellent in our study, the tricuspid regurgitation signal may have been suboptimal in patients with low peak TRV values, as suggested by the weak correlation between peak TRV and mPAP in this range. Third, our findings may not be generalisable to Western populations, as the patients included in this study were Japanese. Fourth, peak TRV may be unreliable in certain cases, such as when tricuspid annular dilation with inadequate leaflet coaptation limits accurate velocity measurement. Fifth, although the interval between echocardiography and RHC was relatively short in our study (within 5 days), haemodynamic changes during this period may still have affected the correlation between mPAP and both peak TRV and eRAP. Finally, although the cut-off value for peak TRV identified in this study was internally validated using cross-validation, external validation was not performed. These limitations highlight the need for cautious interpretation in specific populations. External validation in independent cohorts will be important to confirm the generalisability of this cut-off. Further studies are warranted to address these limitations.

CONCLUSION

In this multicentre, retrospective, observational study including individuals who underwent echocardiography and RHC, lowering the peak TRV threshold of 2.9 m/s to 2.7 m/s showed favourable diagnostic performance under the revised PH definition, facilitating earlier detection of disease. Furthermore, incorporating eRAP with TRV enhanced diagnostic accuracy and enabled straightforward risk stratification. These results support the use of TRV ≥2.7 m/s, together with eRAP assessment, as a practical echocardiographic strategy for PH screening in routine clinical settings, potentially enabling earlier referral and intervention.

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REFERENCES

1 Maron BA, Hess E, Maddox TM, *et al*. Association of Borderline Pulmonary Hypertension With Mortality and Hospitalization in a Large Patient Cohort: Insights From the Veterans Affairs Clinical Assessment, Reporting, and Tracking Program. *Circulation* 2016;133:1240–8.
2 Beshay S, Sahay S, Humbert M. Evaluation and management of pulmonary arterial hypertension. *Respir Med* 2020;171:106099.
3 Demerouti EA, Manginas AN, Athanassopoulos GD, *et al*. Complications leading to sudden cardiac death in pulmonary arterial hypertension. *Respir Care* 2013;58:1246–54.
4 Galiè N, Humbert M, Vachiery J-L, *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): Endorsed by: Association for European Paediatric and Congenital Cardiology (AEPC), International Society for Heart and Lung Transplantation (ISHLT). *Eur Heart J* 2016;37:67–119.
5 Kimura M, Taniguchi H, Kondoh Y, *et al*. Pulmonary hypertension as a prognostic indicator at the initial evaluation in idiopathic pulmonary fibrosis. *Respiration* 2013;85:456–63.
6 Suzuki A, Taniguchi H, Watanabe N, *et al*. Significance of pulmonary arterial pressure as a prognostic indicator in lung-dominant connective tissue disease. *PLoS ONE* 2014;9:e108339.
7 Humbert M, Kovacs G, Hoeper MM, *et al*. 2022 ESC/ERS Guidelines for the diagnosis and treatment of pulmonary hypertension. *Eur Heart J* 2022;43:3618–731.
8 Montané BE, Fiore AM, Reznicek EC, *et al*. Optimal Tricuspid Regurgitation Velocity to Screen for Pulmonary Hypertension in Tertiary Referral Centers. *Chest* 2021;160:2209–19.
9 Gall H, Yogeswaran A, Fuge J, *et al*. Validity of echocardiographic tricuspid regurgitation gradient to screen for new definition of pulmonary hypertension. *EClinicalMedicine* 2021;34:100822.
10 D'Alto M, Di Maio M, Romeo E, *et al*. Echocardiographic probability of pulmonary hypertension: a validation study. *Eur Respir J* 2022;60:2102548.
11 Dimopoulos K, Condliffe R, Tulloh RMR, *et al*. Echocardiographic Screening for Pulmonary Hypertension in Congenital Heart Disease: JACC Review Topic of the Week. *J Am Coll Cardiol* 2018;72:2778–88.
12 Rudski LG, Lai WW, Afilalo J, *et al*. Guidelines for the echocardiographic assessment of the right heart in adults: a report from the American Society of Echocardiography endorsed by the European Association of Echocardiography, a registered branch of the European Society of Cardiology, and the Canadian Society of Echocardiography. *J Am Soc Echocardiogr* 2010;23:685–713; .
13 Lang RM, Badano LP, Mor-Avi V, *et al*. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. *Eur Heart J Cardiovasc Imaging* 2015;16:233–70.
14 Nagueh SF, Smiseth OA, Appleton CP, *et al*. Recommendations for the Evaluation of Left Ventricular Diastolic Function by Echocardiography: An Update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. *Eur Heart J Cardiovasc Imaging* 2016;17:1321–60.
15 Mukherjee M, Rudski LG, Addetia K, *et al*. Guidelines for the Echocardiographic Assessment of the Right Heart in Adults and Special Considerations in Pulmonary Hypertension: Recommendations from the American Society of Echocardiography. *J Am Soc Echocardiogr* 2025;38:141–86.
16 D'Andrea A, Naeije R, D'Alto M, *et al*. Range in pulmonary artery systolic pressure among highly trained athletes. *Chest* 2011;139:788–94.
17 Jankowich M, Maron BA, Choudhary G. Mildly elevated pulmonary artery systolic pressure on echocardiography: bridging the gap in current guidelines. *Lancet Respir Med* 2021;9:1185–91.
18 Fisher MR, Forfia PR, Chamera E, *et al*. Accuracy of Doppler echocardiography in the hemodynamic assessment of pulmonary hypertension. *Am J Respir Crit Care Med* 2009;179:615–21.
19 D'Alto M, Romeo E, Argiento P, *et al*. Accuracy and precision of echocardiography versus right heart catheterization for the assessment of pulmonary hypertension. *Int J Cardiol* 2013;168:4058–62.
20 Ferrara F, Gargani L, Contaldi C, *et al*. A multicentric quality-control study of exercise Doppler echocardiography of the right heart and the pulmonary circulation. The RIGHT Heart International NETwork (RIGHT-NET). *Cardiovasc Ultrasound* 2021;19:9.
21 Sun Z-Y, Li Q, Li J, *et al*. Echocardiographic evaluation of the right atrial size and function: Relevance for clinical practice. *Am Heart J Plus* 2023;27:100274.
22 Farber HW, Foreman AJ, Miller DP, *et al*. REVEAL Registry: correlation of right heart catheterization and echocardiography in patients with pulmonary arterial hypertension. *Congest Heart Fail* 2011;17:56–64.

23 Pellicori P, Carubelli V, Zhang J, *et al*. IVC diameter in patients with chronic heart failure: relationships and prognostic significance. *JACC Cardiovasc Imaging* 2013;6:16–28.
24 Iaconelli A, Cuthbert J, Kazmi S, *et al*. Inferior vena cava diameter is associated with prognosis in patients with chronic heart failure independent of tricuspid regurgitation velocity. *Clin Res Cardiol* 2023;112:1077–86.
25 Hedman K, Nylander E, Henriksson J, *et al*. Echocardiographic Characterization of the Inferior Vena Cava in Trained and Untrained Females. *Ultrasound Med Biol* 2016;42:2794–802.