My Approach to Estimate Pulmonary Pressures: Practical Aspects

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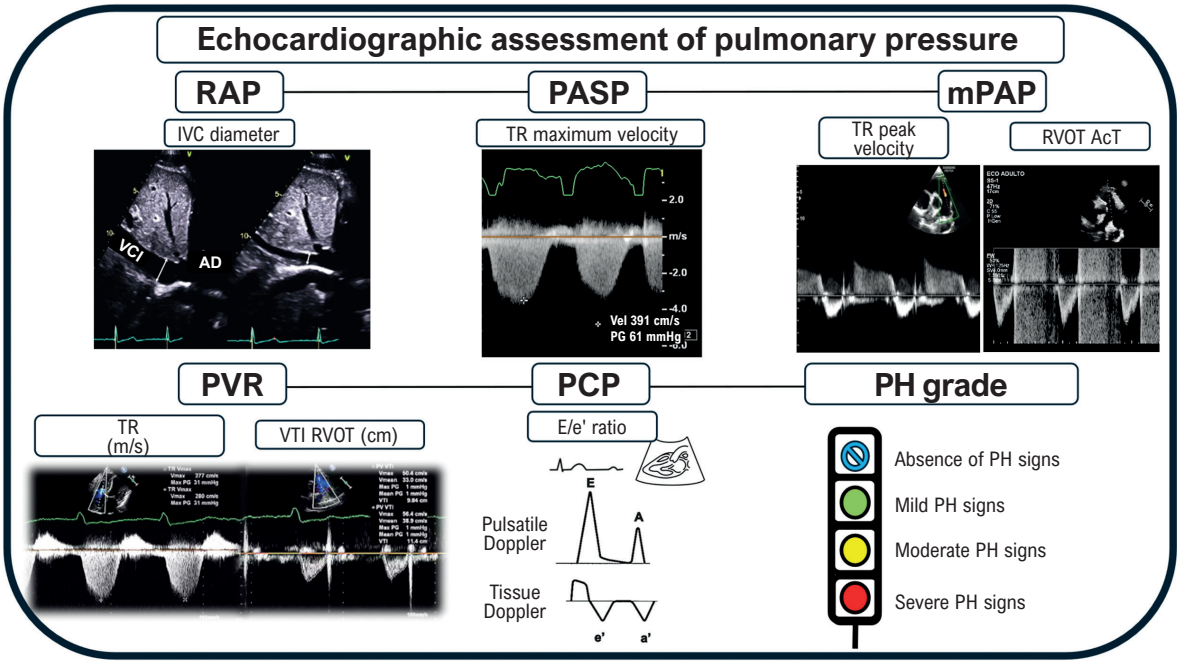
Central Illustration: My Approach to Estimate Pulmonary Pressures: Practical Aspects



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IVC: inferior vena cava; VTI RVOT: velocity-time integral right ventricular outflow tract; TR: Tricuspid regurgitation; PASP: Pulmonary artery systolic pressure.

Abstract

Pulmonary hypertension (PH) is a global disease and is increasingly present in echocardiography laboratories, given the progressive rise in its prevalence with population aging. Previously diagnosed only through invasive methods, echocardiographic assessment of pulmonary pressures has become fundamental in screening and monitoring patients with PH. This topic was revisited by the American Society of Echocardiography in a new guideline published in March 2025, reinforcing the importance of echocardiography in clinical stratification, considering recent updates in the definition of PH. Among the main assessment parameters, systolic pulmonary artery pressure, mean pulmonary artery pressure (mPAP), pulmonary vascular resistance (PVR), and pulmonary capillary pressure (PCP) are described, along with a series of precautions for the correct estimation and interpretation of these values. The classification of PH levels has also been revised in a new framework. As an accessible and reproducible method, the assessment of pulmonary pressures through echocardiography stands out as an essential tool in the evaluation and follow-up of PH.

Keywords

Pulmonary Hypertension; Echocardiography; Doppler Ultrasonography; Pulmonary Wedge Pressure.

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Introduction

Pulmonary hypertension (PH) has an estimated prevalence of about 1% of the global population, with a gradual increase

with aging due to its direct relationship with cardiac and pulmonary diseases. It has prognostic implications due to its severe hemodynamic consequences and serves as a fundamental parameter for clinical decision-making and therapeutic guidance.[1]

The assessment of pulmonary pressures has been a constant pursuit for a better hemodynamic understanding of diseases affecting both acquired and congenital heart conditions. Initially, this type of evaluation was performed exclusively through invasive hemodynamic methods, predominantly using pulmonary artery catheters such as the Swan-Ganz and right heart catheterization.[2]

The incorporation of the color Doppler method and the ability to correlate intracavitary flow velocities with hemodynamic gradients, with reasonable agreement with invasive parameters, has enabled the rapid evolution of echocardiography in this field. As a non-invasive, reproducible, and replicable method, performed at the bedside and with relatively low cost, echocardiography presents a significant advantage as an initial assessment tool.[3]

Definition of PH

The currently accepted definition of PH was proposed during the 6th World Symposium on PH in 2018, where 124 experts gathered and reviewed it.[4]

Previous definition of PH

- Mean pulmonary artery pressure (mPAP): ≥ 25 mmHg
- Pulmonary vascular resistance (PVR): ≥ 3 Wood Units (WU) – Precapillary hypertension
- Pulmonary capillary pressure (PCP): ≤ 15 mmHg – Precapillary hypertension

Proposed (current) definition:

- mPAP: > 20 mmHg
- PVR: > 2 Wood Units (WU) – Precapillary hypertension
- PCP: ≤ 15 mmHg – Precapillary hypertension

This change was based on data from healthy individuals, in which resting mPAP was identified as approximately 14.0±3.3 mmHg. Considering a variation of two standard deviations as still within normal limits, a mPAP value >20 mmHg would be the threshold for abnormal pulmonary artery pressure (above the 97.5th percentile).[4]

Based on these values, the echocardiographic parameters described below were studied in relation to invasive assessment, not with the aim of replacing this examination, but rather as an initial screening proposal, since performing right heart catheterization at a population level is impractical and not cost-effective.

The concept of communicating vessels

The echocardiographic assessment of pulmonary pressures is based on the concept of communicating vessels. This concept establishes that a fluid with the same specific mass and at the same height would have the same pressure at any point in the system. Based on this, echocardiography can identify the pulmonary artery systolic pressure (PASP) by calculating the right ventricular systolic pressure (RVSP).

To calculate RVSP, the systolic gradient between the right ventricle (RV) and the right atrium (RA) must first be evaluated using the maximum velocity of tricuspid regurgitation (TR), applying the modified Bernoulli equation, which derives the gradient based on velocity. By adding the estimated right atrial pressure (RAP) to this pressure estimate, RVSP is obtained, which, in a system without flow obstruction, will be similar to PASP.

Mean RAP

For this estimation, we first examine how to assess the mean RAP. Throughout its evolution, transthoracic echocardiography has identified various metrics to estimate RAP with reasonable accuracy. Despite several limitations in its analysis, the most commonly used parameter for this inference is the measurement of the inferior vena cava (IVC) diameter during an expiratory pause and its respiratory variability, as shown in Figure 1.

Depending on the initial measurement of the IVC and its variation with respiratory phasicity, RAP can be estimated as follows:

- RAP < 3 mmHg
- RAP: 3 mmHg (0 - 5 mmHg)
- RAP: 8 mmHg (5 - 10 mmHg)
- RAP: 15 mmHg (10 - 20 mmHg)
- RAP: 20 mmHg

The IVC can be measured in most patients using the subcostal window, making it easily reproducible during hospitalization or outpatient follow-up. As we will see below, this index is not always representative of RAP in certain scenarios, which should be interpreted with caution.

Precautions in the acquisition and interpretation of the IVC:

- Acquire the IVC image longitudinally with a central axis cut throughout the respiratory cycle (the measurement of the transverse axis can help avoid errors).
- Be cautious when evaluating patients with chronic hypervolemia (heart failure and advanced chronic kidney disease)
- Be cautious when evaluating athletes (IVC distended due to excellent preload).

In cases of diagnostic uncertainty or indeterminate cases, secondary indices for estimating RAP can be considered, such as a tricuspid E/e' ratio > 6, predominance of diastolic flow in the hepatic veins*, and identification of a restrictive diastolic filling pattern in the right chambers.[5]

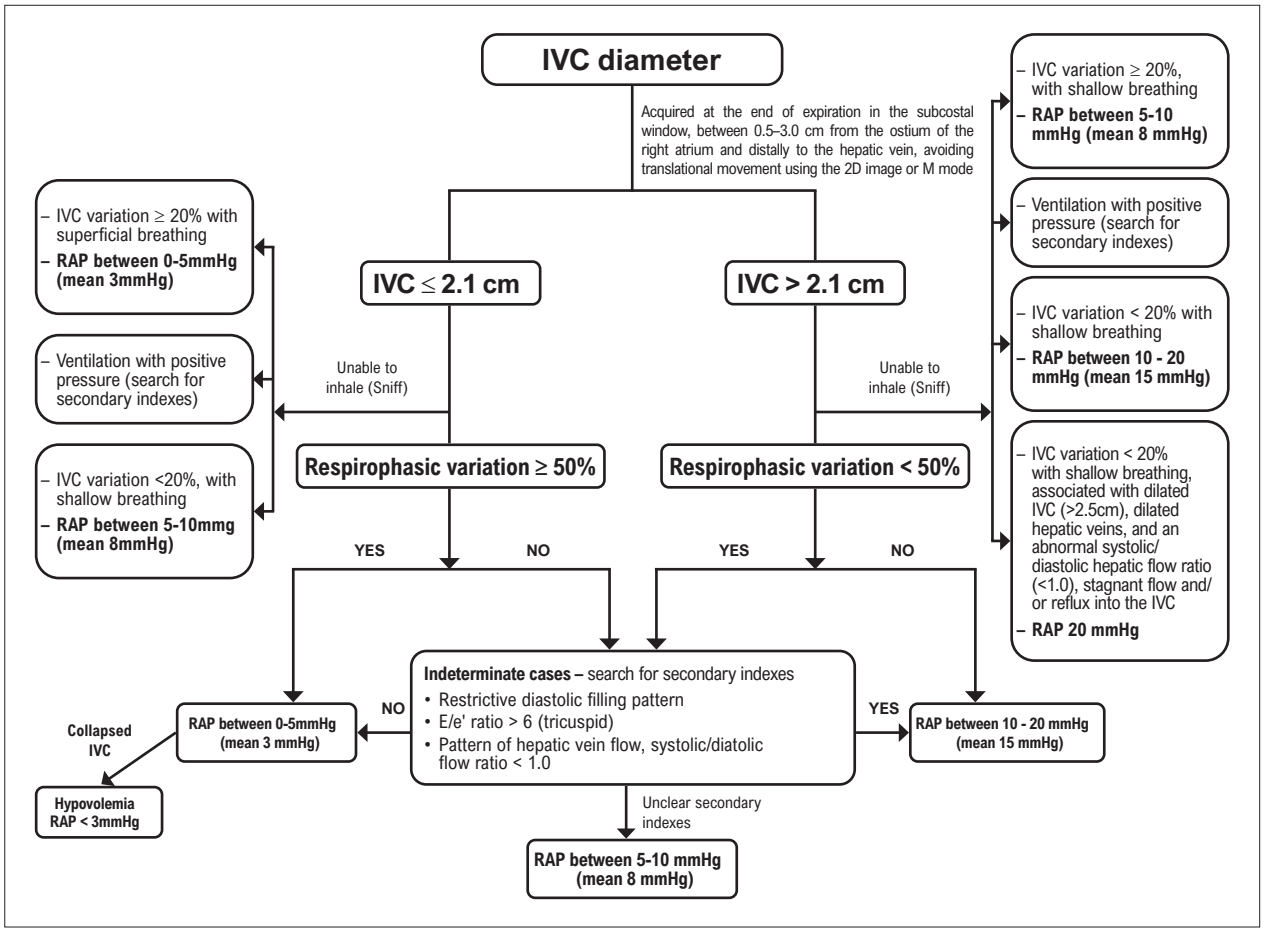

Figure 1 – *Flowchart for the assessment of mean RAP proposed by the American Society of Echocardiography in its latest guideline (ASE 2025);[5] IVC: inferior vena cava; RAP: right atrial pressure; RV: right ventricle.*

$$*SFF = \frac{VTI\ of\ the\ S\ wave}{VTI\ of\ the\ S\ wave + VTI\ of\ the\ D\ wave} \times 100$$

(Values < 55 % are sensitive and specific for increases in RAP)

The systolic filling fraction (SFF) equation VTI: velocity-time integral; S: systolic; D: diastolic

PASP

Still regarding the concept of communicating vessels without flow obstruction, it can be inferred that RVSP will be equivalent to PASP, which is the hemodynamic parameter that is actually being investigated.

Figure 2 illustrates a case of a significant increase in pulmonary pressures and how the final PASP value is calculated.

Precautions in the acquisition and interpretation of PASP:

– When measuring the maximum velocity of TR (RV–RA gradient), proper Doppler angle alignment (< 20°) should be maintained to avoid possible underestimation due to measurement error.

– Excessive gain should be avoided when acquiring the spectral Doppler image to prevent overestimation of the actual transvalvular gradient, as shown in Figure 3.

– To ensure accuracy and account for respiratory variation, RVSP should ideally be calculated from the average of three consecutive beats in echocardiographic windows with the highest velocities and best spectral Doppler envelopes.

Other parameters for estimating PASP have been described using the acceleration time (AcT) of systolic flow in the right ventricular outflow tract (RVOT). As a region of low pressure and high compliance, the pulmonary bed generates a flow pattern with low velocities and a long AcT during the systolic period.

A progressive increase in pulmonary pressures, particularly in its precapillary component, will result in a progressively shorter AcT, associated with an earlier-than-usual systolic peak (Figure 4).

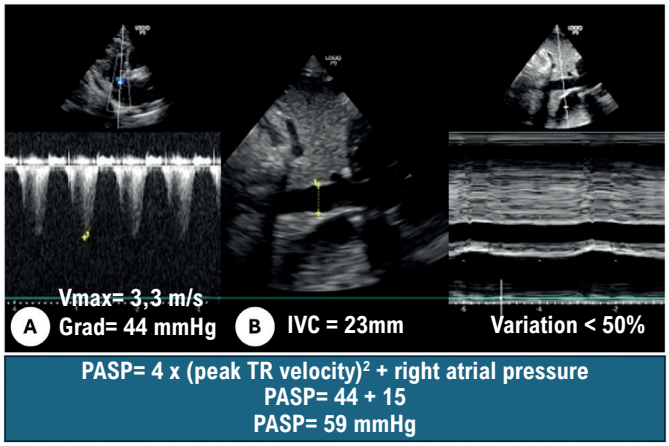

Figure 2 – *Estimation of PASP, measured at 59 mmHg in this patient. A) TR assessed with continuous-wave Doppler. B) Estimation of RAP by evaluating the diameter of the IVC and collapsibility with the respiratory cycle in M-mode. IVC: inferior vena cava; PASP: Pulmonary arterial systolic pressure; RAP: right atrial pressure.*

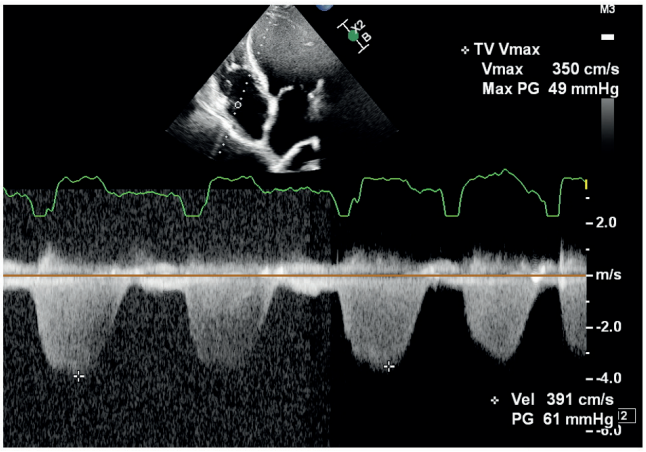

Figure 3 – *TR on continuous-wave Doppler with excessive gain (left, with a maximum velocity of 3.9 m/s) and with appropriate gain (right, with a maximum velocity of 3.5 m/s).*

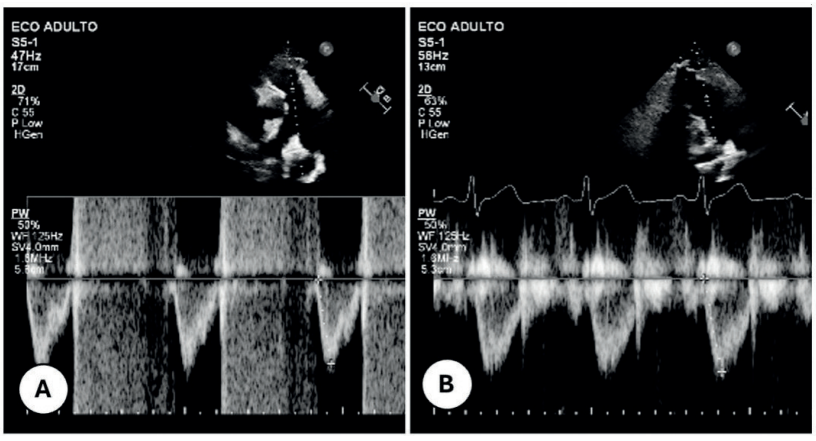

Figure 4 – *Assessment of AcT considering the sample volume at the level of the pulmonary valve; A) Shortened AcT (81ms) associated with mid-systolic notch in the tracing, confirming the finding of PH; B) Normal AcT (113ms).*

Based on this important hemodynamic concept, correlation studies between these two variables were conducted, allowing for the identification and validation of the formulas presented in Table 1.

Precautions in the acquisition and interpretation of AcT

- Window: Parasternal short-axis at the level of the great vessels, directed toward the pulmonary valve.
- Sample volume position: Acquired using pulsed Doppler in the RVOT, with the sample volume positioned just below the pulmonary valve at the end of expiration.
- Measurement: From the beginning of pulmonary flow to the peak velocity.
- The greatest validation of pulmonary ATc has been conducted in cases of precapillary PH.
- ATc in the RVOT may be less reliable in patients with low or high heart rates (<60 and >100 beats/min).

mPAP

Considered the reference measure for defining PH and severity stratification, the determination of mPAP is very important for clinical cardiologists. Over the past decades, several studies have validated this non-invasive estimate, culminating in the recommendation of the American guideline for the echocardiographic evaluation of the right heart in adults in 2010. This document provided a detailed description of the formulas for calculating both mPAP and pulmonary artery diastolic pressure (PADP), which were replicated in the 2025 guideline.

Based on current knowledge, mPAP values greater than 20 mmHg are considered pathological and should be reported in the echocardiographic report.

The step-by-step process for calculating mPAP after acquiring PASP and PADP is illustrated in Figure 5 and Table 2.

Precautions in acquisition and interpretation

- The calculation of mPAP involves other variables, so its estimation should be performed using the best possible technique.

Table 1 – Main Clinically Validated Formulas for Estimating Pulmonary Artery Systolic Pressure (PASP)

Parameter	Formula	Condition
PASP	4 x maximum velocity of TR + RAP [6,7]	
PASP	134 – (0,9 X AcT) [5]	
PASP		RVOT AcT > 105 ms (without PH*)[6] * Considered normal, based on epidemiological studies involving healthy populations.
PASP		RVOT AcT < 80 ms (probable HP)[5]

PASP: pulmonary artery systolic pressure; TR: tricuspid regurgitation; RAP: right atrial pressure; AcT: acceleration time; PH: pulmonary hypertension; RVOT: right ventricular outflow tract.[5]

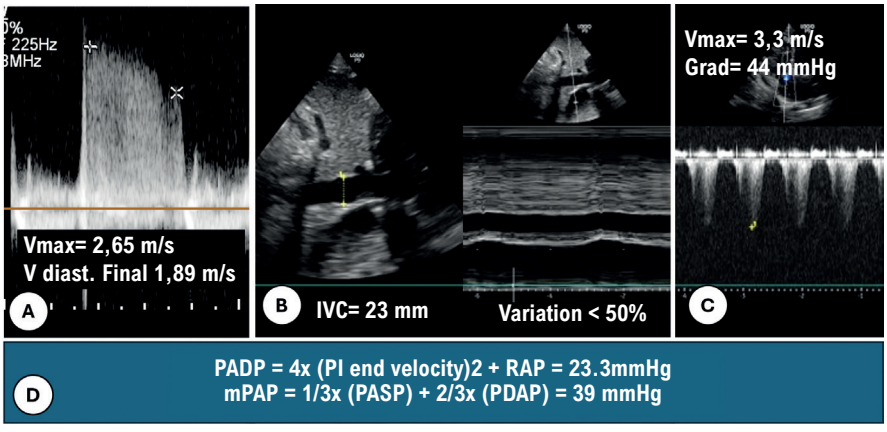

Figure 5 – *Estimation of mPAP and pulmonary artery diastolic pressure (PADP) using the initial and final velocities of pulmonary insufficiency (PI): A) PI on continuous-wave Doppler; mPAP is obtained by summing RAP with the peak gradient of PI calculated using the Bernoulli equation. PADP is obtained by calculating the gradient considering the end-diastolic velocity of PI added to RAP; B) Estimation of RAP by evaluating the IVC and its respiratory collapsibility; C) TR; D) Application of formulas for calculating mPAP and PADP; PASP: pulmonary artery systolic pressure; IVC: inferior vena cava.*

– mPAP uses Pulmonary Insufficiency (PI) as an analysis parameter for its calculation. The initial velocity of the PI jet, added to RAP, allows for the determination of mPAP.
– PADP uses the final velocity of the PI jet, added to RAP, to determine the PADP value.

PVR

Concept describing the measurement of resistance that the pulmonary vascular system offers to blood flow from the RV. This parameter provides an important discriminatory factor, allowing stratification between the precapillary and postcapillary components of PH.

The identification of this measurement in a non-invasive manner originates from the classic work of Abbas et al.[12] The study was published in the Journal of the American College of Cardiology (JACC) in 2003, and their formula was later validated in a new publication in 2013 in the Journal of the American Society of Echocardiography (JASE).[12]

For the calculation of PVR, two basic evaluation parameters are required (Figure 6).

The formulas used for PVR calculation are described in Table 3.

PCP

The combined assessment of pulmonary pressures, PVR, and PCP allows for the classification of PH into one of the five categories described below, according to the latest PH classification (ESC 2022):

1. Pulmonary Arterial Hypertension (PAH)
2. PH associated with left ventricular disease
3. PH associated with lung disease
4. PH associated with obstructive lung disease
5. PH with unclear or multifactorial mechanisms (etiology)

Table 2 – Main clinically validated formulas used for mPAP estimation

Parameter	Formula	Condition
mPAP	$\frac{1}{3}(PSAP) + \frac{2}{3} PADP$	
mPAP	79 – 0.45x(*AcT*)[8]	AcT ≥ 120ms (HR 60 – 100bpm)
mPAP	90 – 0.62x(*AcT*)[9]	AcT < 120ms (HR 60 – 100bpm)
mPAP	0.61x(*PASP*) + 2[10]	
mPAP	VTI da IT + RAP11	

mPAP: mean pulmonary artery pressure; AcT: acceleration time of the right ventricular outflow tract; HR: heart rate; TR: tricuspid regurgitation; VTI: velocity-time integral; RAP: right atrial pressure.[5]

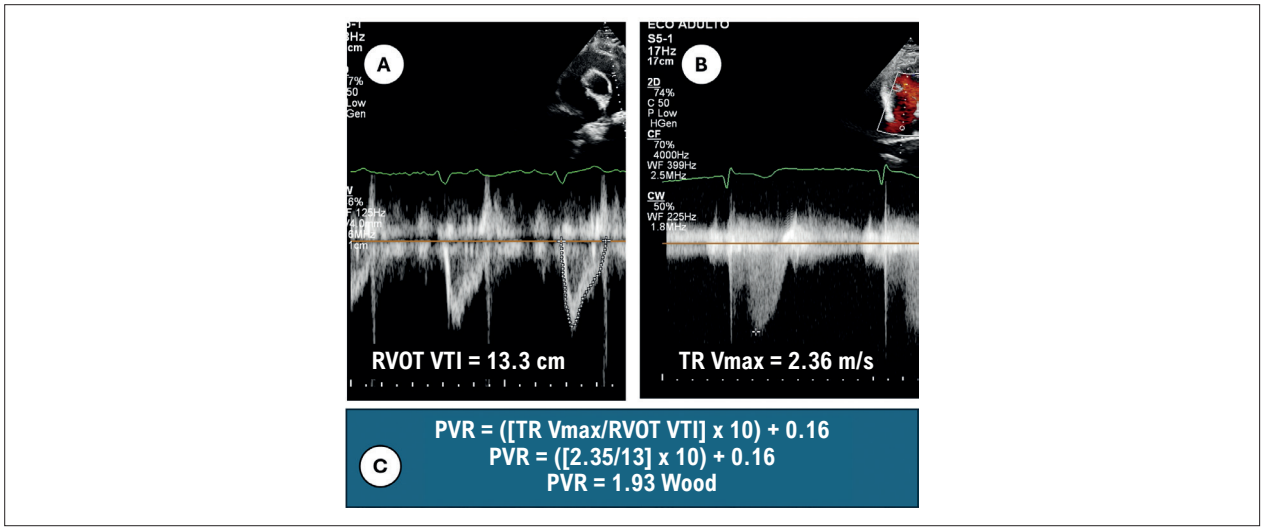

Figure 6 – *Calculation of PVR using velocity-time integral (VTI) of the RVOT' (A); maximum velocity of TR (B); application of the formula (C).*

Thus, the evaluation of PCP will determine whether capillary pressures – and consequently, left atrial pressure – are elevated or not, enabling an initial stratification between groups with and without left heart involvement (postcapillary and precapillary hypertension).

The main formulas for this calculation are described below (Table 4), with Nagueh's formula being the most widely used. In their original study, the findings of Nagueh et al.[13] were strongly correlated with left ventricular filling pressures and, therefore, with PCP.

Precautions in acquisition and interpretation:

– The evaluation of E/e' variables is routinely performed in the assessment of diastolic function and therefore does not add extra time to the examination.

Table 3 – Main clinically validated formulas used for PVR estimation

Parameter	Formula	Condition
PVR	$\frac{TR\ maximum\ velocity\ (m/s)}{RVOT\ VTI\ (cm)}$ X 10 + 0.16[12]	RVP > 2.0 Woods is considered abnormal
PVR	$\frac{RVSP - PCP}{CO}$	RVSP, RVDP and CO values may be obtained from echocardiographic data

PVR: Pulmonary Vascular Resistance; TR: Tricuspid Regurgitation; VTI: Velocity-Time Integral; RVOT: Right Ventricular Outflow Tract; RVSP: Right Ventricular Systolic Pressure; PCP: Pulmonary Capillary Pressure; CO: Cardiac Output.[5]

Table 4 – Main clinically validated formulas used for PCP estimation

Parameter	Formula	Condition
PCP	1,24 X E/e' ratio + 1,9 [13]	– Normality values: < 12 – 15 mmHg E/e' > 15 was correlated with PCP > 15 mmHg Original study: Sensitivity: 89% - Specificity: 91% - Accuracy: 0.92
PCP	PCP = 5,7 X E/VP + 4,6 [14]	Study with 45 patients (LVEF: 40% ± 15%)

PCP: pulmonary capillary pressure; LVEF: left ventricle ejection fraction; CO: cardiac output; PVR: Pulmonary vascular resistance.

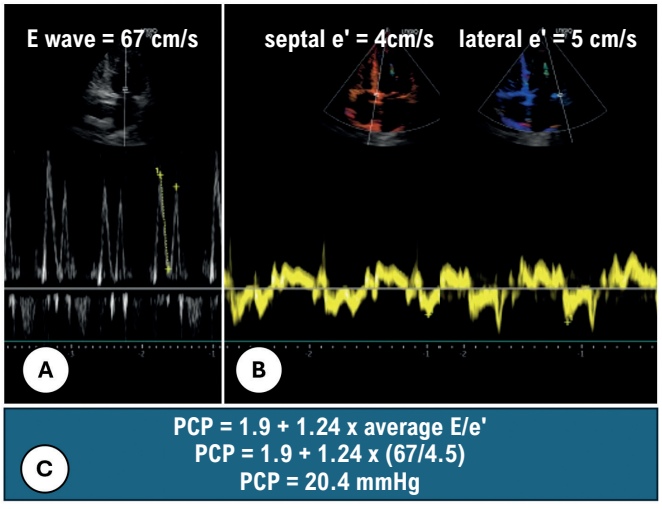

Figure 7 – *Calculation of PCP using Nagueh's formula; A) E and A waves of mitral inflow on pulsed Doppler; B) e' wave of the mitral annulus on tissue Doppler; C) Nagueh's formula with the estimation of a significant increase in PCP.*

Table 5 – Classification of pulmonary pressure levels according to the 2025 American Society of Echocardiography guidelines

Variable	Normality	Abnormality grade		
		Discrete	Moderate	Important
TR velocity	< 2.8m/s	2.8 -3.1m/s	3.2 - 3.5m/s	≥3.6m/s
PASP	≤34mmHg	35 – 49 mmHg	50 -69 mmHg	≥70mmHg
RAP	0 - 4 (Mean 3 mmHg)	5 a 9 (Médio 8 mmHg)	10 a 14 mmHg	≥15mmHg
AcT	>105ms	80 – 105ms	61 -79ms	≤ 60ms

PVR: pulmonary vascular resistance; TR: tricuspid regurgitation; PASP: pulmonary artery systolic pressure; RAP: right atrial pressure; AcT: acceleration time[3]

- Since they use the Doppler method, these variables are influenced by the insonation angle.
- In patients with significant mitral valve disease, the A wave may be increased due to the valvulopathy, limiting the analysis of PCP.
- PCP assessment is impaired in the presence of arrhythmias.

Summary of main warnings and pitfalls

1. Measure the densest primary edge of the continuous-wave Doppler signal ("chin") and not the faint spectra ("beard"), optimizing gain and brightness across multiple imaging planes.
2. Ultrasound contrast agents, saline blood contrast, and agitated saline solution can be used to enhance the Doppler signal, improving diagnostic accuracy.
3. Position the ultrasound beam parallel to the direction of blood flow, ideally at an angle $<20°$ to minimize any errors in velocity measurement.
4. If there is significant TR, the shape of the continuous-wave Doppler signal can help guide the accuracy of right ventricular systolic pressure (RVSP) estimates. A triangular continuous-wave Doppler contour will underestimate RVSP, while accuracy improves with a parabolic Doppler contour.
5. Do not consider velocity after a pause or premature contraction (extrasystole).
6. Consider the average velocity over five to seven beats in patients with arrhythmia.
7. Measure the IVC while visualizing its walls throughout the entire respiratory cycle, avoiding possible false collapsibility due to sampling error.
8. Physiological aging and an increase in body surface area can elevate pulmonary artery systolic pressure (PASP) without necessarily indicating pathology.

Clinical interpretation

Although there are several echocardiographic methods to calculate PMAP, the standard reporting of RVSP is generally preferred for several reasons. First, RVSP is measured directly from the peak velocity of the TR jet, making it a more direct and reliable estimate. In contrast, methods such as AcT of RVOT and the estimation of mPAP and PADP rely on indirect calculations and assumptions, which can introduce measurement errors.

The correct quantification and stratification of pulmonary pressures will allow their classification as mild, moderate, and severe. This classification was revisited in the guideline for echocardiographic assessment of the right heart in adults and special considerations in PH by the American Society of Echocardiography (ASE), grading the abnormality of the values presented in Table 5.

Due to the significant limitation in correctly identifying pulmonary vascular resistance (PVR), PH can be assessed by considering only the maximum velocity of TR to avoid inference errors, a parameter that had already been corroborated by the European Society of Cardiology (ESC) in its 2022 PH guidelines.

The definitive assessment of mPAP for diagnosis, classification, and initiation of pharmacological therapy for PH requires direct hemodynamic measurement via invasive right heart catheterization.

Conclusion

The evaluation of pulmonary pressures has been a cornerstone in identifying adult patients and patients with congenital diseases who have a worse clinical prognosis. The identification of pulmonary pressure levels is still based on invasive quantification through hemodynamic assessment, which remains the gold standard method. Doppler echocardiography emerges as an excellent alternative for the initial stratification of these patients, adding other parameters such as ventricular function and valve assessment.

Author Contributions

Conception and design of the research, writing of the manuscript and critical revision of the manuscript for intellectual content: Silva HAGP, Gomes HM.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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Study Association

This study is not associated with any thesis or dissertation work.

Ethics Approval and Consent to Participate

This article does not contain any studies with human participants or animals performed by any of the authors.

Use of Artificial Intelligence

The authors did not use any artificial intelligence tools in the development of this work.

Availability of Research Data

The underlying content of the research text is contained within the manuscript.

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