Journal of Case Reports and Reviews in Medicine
https://doi.org/10.64978/jcrrm.2026.09140128

ISSN: 3069-0749

Research Article

Open Access Full Text Article

Echocardiographic Aspects of Pulmonary Heart Disease at the "Les Promoteurs De La Bonne Santé" Medical Center, Yaoundé: a Descriptive and Correlative Analysis of 73 Cases

Kuiate David[1,2,3,6], Kougoum Takam Natacha[2], Nanfack Fanny Nadege[2], Fomekong Dieuseul Lothner[2,4], Foundikou Vessah Dayib[2], Apouakone Mefire Aicha[2], Kouam Brice Bertrand[7], Nana Yasmine Laurie[8], Tankeu Ronni[5], Yiagnigni Euloge Mfopou[5**]**

[1]International School of Health Sciences and Medical Sciences, Kesmonds International University.
[2]Department of Radiology and Medical Imaging, "Les Promoteurs de la Bonne Santé" Medical Center.
[3]Faculty of Medicine and Biomedical Sciences, University of Yaoundé 1.
[4]"Santé sans frontières" School of Sanaga-Maritime-Edéa.
[5]Department of Cardiology, "Les Promoteurs de la Bonne Santé" Medical Center.
[6]Department of Biomedical Sciences, University of Ngaoundéré.
[7]Department of Radiology and Medical Imaging, Bafoussam Regional Hospital.
[8]"Le Samaritain Plus" Medico-Surgical Center.

[*]**Correspondence:**
KUIATE David,
Medical Imaging, Faculty of Medicine and Biomedical Sciences, Yaoundé, Cameroon. ORCID: 0009-0000-4337-1686

Received: August 05, 2026; **Published:** September 14, 2026.

How to cite this article:
David K, Natacha KT, Nadege NF, Lothner FD, Dayib FV, Aicha AM, Bertrand KB, Laurie NY, Ronni T, Mfopou YE. Echocardiographic Aspects of Pulmonary Heart Disease at the "Les Promoteurs De La Bonne Santé" Medical Center, Yaoundé: a Descriptive and Correlative Analysis of 73 Cases. *J Case Rep Rev Med*. 2026;2(3):1-5. https://doi.org/10.64978/jcrrm.2026.09140128

Abstract

Introduction: Chronic pulmonary heart disease, secondary to pulmonary arterial hypertension (PAH), represents a major diagnostic challenge in everyday cardiology practice. Severe PAH leads to progressive remodeling of the right heart, whose adaptive mechanisms and thresholds of decompensation remain to be clarified in large echocardiographic cohorts.

Objective: To describe the echocardiographic features of pulmonary heart disease in a cohort of patients with severe PAH, to establish correlations between pulmonary pressures and chamber dilation, and to identify critical thresholds associated with right ventricular (RV) failure.

Methodology: A retrospective, observational, single-center study including 73 consecutive patients. Echocardiographic data (diameters, areas, systolic function, valve disease) were correlated with demographic data. Statistical analyses included Student's t-test, the Chi-square test, and Pearson correlations.

Results: The mean age of the study population was 61.5 ± 17.8 years, with a female predominance (53.4%) and severe PAH (systolic pulmonary artery pressure [sPAP] = 75.2 ± 14.4 mmHg), marked dilation of the RV (43.4 ± 8.8 mm) and of the right atrium (RA) (27.6 ± 8.9 cm²). An anatomo-functional dissociation was observed: 90.4% had preserved RV systolic function, despite RA dilation in 75.3% and grade III tricuspid regurgitation (TR) in 80.8%. Correlations revealed a "cascade" effect (sPAP → RV → RA). Factors associated with right ventricular dysfunction were identified for an RV diameter > 50 mm combined with sPAP > 85 mmHg ($p<0.01$). Pericardial effusion was strongly correlated with left ventricular dilation ($p<0.01$).

Conclusion: Pulmonary heart disease is found mainly in older patients with a female predominance; the factors associated with right ventricular failure are an RV end-diastolic diameter > 50 mm and sPAP > 85 mmHg. Severe TR is a marker of RA dilation, and the presence of pericardial effusion signals global heart failure.

Keywords: Pulmonary arterial hypertension; Echocardiography; Tricuspid regurgitation.

Introduction

Pulmonary arterial hypertension (PAH) is a disease characterized by an elevation of pulmonary artery pressure (PAP) that progressively leads to remodeling of the right heart and, ultimately, to fatal right heart failure. The right ventricle (RV), an organ originally adapted to a low-pressure, low-resistance system, responds to the increase in afterload through adaptive mechanisms that include hypertrophy and dilation.[1]

While the pathophysiology of PAH is well documented, the precise echocardiographic semiology of decompensation remains complex. Indeed, an "anatomo-functional dissociation" is frequently observed, in which the RV appears markedly dilated yet retains a normal ejection fraction for a prolonged period.[2] Moreover, tricuspid regurgitation (TR), often considered secondary to annular dilation, worsens volume overload, although its prognostic role relative to isolated chamber dilation remains debated.[3]

Identifying the factors associated with dilation leading to RV failure is crucial in clinical practice for optimizing treatment timing (initiation of PAH-specific therapies, transplantation indication). The aim of this study is to characterize the pattern of right heart remodeling in a cohort with PAH, to analyze structure–function correlations, and to define the echocardiographic parameters that herald the shift toward decompensated right heart failure.

Methodology

This is a retrospective, observational, analytical study conducted on an echocardiographic database compiled consecutively at the Cardiac Imaging Department of the Les Promoteurs de la Bonne Santé Medical Center (SIM-CM-PBS). The study was approved by the local ethics committee, and data were collected anonymously, in accordance with the Declaration of Helsinki. The study population consisted of 73 adult patients who underwent a complete transthoracic echocardiogram (TTE). Examinations were performed in accordance with current recommendations of the European Association of Cardiovascular Imaging (EACVI) and the American Society of Echocardiography (ASE).[4] The quantitative variables used were:

- RV Diameter (RV-DIA): Basal diameter of the right ventricle in the apical 4-chamber view (normal < 40 mm).
- RA Area (RA-AREA): Planimetered right atrial area in end-systole (normal < 18–20 cm²).
- Systolic Pulmonary Artery Pressure (sPAP): Systolic pulmonary artery pressure derived from the tricuspid regurgitation velocity (normal < 35 mmHg).
- Inferior Vena Cava Diameter (IVC): Diameter of the inferior vena cava in the subcostal view during expiration (normal < 21 mm).

The qualitative variables studied were: sex, indication for the examination, presence of left ventricular dilation (LV-DIL), RV systolic function (assessed by TAPSE, categorized as normal/impaired), RA dilation (RA-DIL), severity of tricuspid regurgitation (TR, graded I to III according to guidelines), IVC dilation (IVC-DIL), and presence of pericardial effusion (PE).

Statistical analysis was performed using SPSS v25.0. Quantitative variables were expressed as mean ± standard deviation, with median and range (min–max). Qualitative variables were expressed as counts (n) and percentages (%). Normality of distributions was assessed using the Shapiro-Wilk test. Correlations between continuous quantitative variables were assessed using Pearson's correlation coefficient (r), with correlation strength defined as weak ($r<0.3$), moderate ($0.3\leq r<0.5$), strong ($0.5\leq r<0.7$), and very strong ($r\geq 0.7$). Comparison of means for qualitative variables used Student's t-test. The association between categorical variables was assessed using the Chi-square test, with Cramér's V calculated to evaluate the strength of association. Statistical significance was set at $p<0.05$.

Results

Demographic and quantitative descriptive statistics

The sample consisted of 73 patients. Anthropometric characteristics and quantitative echocardiographic measurements are detailed in Table 1.

Table 1: Demographic and quantitative descriptive statistics

Variable	N	Mean ± SD	Median	Min – Max
Age (years)	73	61.5 ± 17.8	65	14 – 88
RV diameter (mm)	73	43.4 ± 8.8	42	27 – 65
RA area (cm²)	73	27.6 ± 8.9	25	12 – 56
sPAP (mmHg)	73	75.2 ± 14.4	75	35 – 95
IVC diameter (mm)	73	23.1 ± 4.2	23	12 – 33

The mean age of the study population was 61.5 years (relatively advanced), with a wide dispersion (14 to 88 years), indicating an age-heterogeneous sample.

- On the cardiac level, the mean values for RV diameter (43.4 mm) and RA area (27.6 cm²) are well above standard echocardiographic norms (RV diameter < 40 mm; RA area < 18–20 cm²). This indicates that the sample is composed of a population with substantial right-sided chamber dilation.
- The mean sPAP was 75.2 mmHg, indicative of severe pulmonary arterial hypertension (PAH) in the majority of cases (normal < 35 mmHg). Inferior vena cava diameter (23.1 mm) was also increased (normal < 21 mm), reflecting volume overload or right-sided dependence.

Distribution of qualitative variables

The distribution of categorical variables is presented in Table 2.

Table 2: Distribution of Qualitative Variables (Counts and Percentages)

Variable	Category	n	%
Sex	Female (F)	39	53.4%
	Male (M)	34	46.6%
Indication	Work-up	30	41.1%
	PE (Pulmonary Embolism)	7	9.6%
	Hypertension	7	9.6%
	Dyspnea	6	8.2%
	HF / RHF / GHF	6	8.2%
	Other (CPD, arrhythmias…)	17	23.3%
LV dilation	Yes	41	56.2%
	No	32	43.8%
RV systolic function	Normal	66	90.4%

	Impaired	7	9.6%
Ventricular hypertrophy	Normal	71	97.3%
	Abnormal	2	2.7%
RA dilation	Yes	55	75.3%
	No	18	24.7%
Tricuspid regurgitation	Grade III	59	80.8%
	Grade II	10	13.7%
	Grade I	4	5.5%
IVC dilation	Yes	41	56.2%
	No	32	43.8%
Pericardial effusion	Yes	15	20.5%
	No	58	79.5%

The table above shows a slight female predominance. The main indication was "work-up" (41%), suggesting that these patients are often followed for chronic cardiovascular conditions. Pulmonary embolism (PE) and hypertension were the most frequent risk factors. Seventy-five percent of cases had a dilated right atrium and 56% a dilated LV, with preserved RV systolic function in 90.4% of cases. Eighty-one percent of patients had grade III tricuspid regurgitation (severe regurgitation), strictly consistent with right atrial dilation (tricuspid annular distension) and very elevated sPAP values.

Matrix of statistical correlations (quantitative variables)

Pearson linear correlations between continuous measurements are reported in Table 3.

Table 3: Matrix of Statistical Correlations (Pearson Coefficient)

Variables compared	Coefficient (r)	Strength	Clinical interpretation
RV diameter vs sPAP	0.42	Moderate	The higher the sPAP, the more the RV dilates.
RA area vs sPAP	0.51	Strong	PAH results in marked RA dilation.
RA area vs RV diameter	0.68	Very strong	RV and RA dilation are closely linked.
IVC diameter vs sPAP	0.15	Weak	Pulmonary pressure does not directly and linearly affect IVC size.
RA area vs IVC diameter	0.45	Moderate	RA dilation is moderately accompanied by IVC dilation.
Age vs sPAP	-0.05	None	Patient age has no relationship with PAH severity.

(Note: r>0.5 strong; 0.3−0.5 moderate; <0.3 weak)
The table above presents the linear correlations between the different variables (sPAP, RV diameter, RA area, IVC diameter).

Cross-correlations between variables (qualitative vs. quantitative / qualitative)

Inferential analyses using comparison of means and Chi-square tests are summarized in Table 4.

Table 4: Cross-Correlations of Variables (Student's t-test and Chi-square / Cramér's V)

Variables crossed	Statistical result	p-value	Interpretation
RV systolic function (normal vs impaired) vs sPAP	Mean normal = 74 mmHg Mean impaired = 85 mmHg	$p<0.05$	Patients with impaired RV systolic function have significantly higher pulmonary pressures.
RV systolic function vs RV diameter	Mean normal = 42 mm Mean impaired = 50 mm	$p<0.01$	Impaired RV function is strongly correlated with a larger diameter.
TR vs RA dilation	Chi^2 / Cramér's V = 0.32	$p<0.05$	Significant association: grade III TR is found in patients with RA dilation.
LV dilation vs pericardial effusion	PE in 34% of LV-dilated vs 6% of non-LV-dilated	$p<0.01$	Pericardial effusion is significantly correlated with LV dilation.
Sex vs RV dilation	Men: 47% vs Women: 62%	$p>0.05$ (NS)	Sex does not statistically influence RV dilation.

These results allow definition of a threshold for RV decompensation. RV dilation is well tolerated as long as systolic function remains normal. Once the diameter exceeds 50 mm and sPAP exceeds 85 mmHg, a shift toward right ventricular failure is observed. The severity of tricuspid regurgitation is not an isolated phenomenon but rather the direct reflection of right atrial dilation. The presence of pericardial effusion is very strongly linked to left ventricular dilation, signaling global heart failure with reactive pericardial congestion.

Table 5. Correlations and associations between echocardiographic parameters (★ $p < 0.05$; ★★ $p < 0.01$; ★★★ $p < 0.001$; NS: not significant)

Variable pair	Test	r / rs / χ^2	p-value	Significance
sPAP × TR grade	Spearman	rs = 0.716	< 0.0001	★★★ Very strong
sPAP × RA area	Pearson	r = 0.488	< 0.0001	★★★ Strong
sPAP × IVC dilation	Biserial	rpb = 0.448	0.0001	★★★ Moderate–strong
sPAP × IVC diameter	Pearson	r = 0.351	0.0023	★★ Moderate
sPAP × RV diameter	Pearson	r = 0.291	0.0124	★★ Weak–moderate
RV diameter × RA area	Pearson	r = 0.492	< 0.0001	★★★ Strong
RV diameter × RA dilation	t-test	t = 4.150	0.0001	★★★ Strong
RA dilation × IVC dilation	Chi^2	$\chi^2 = 23.2$	< 0.0001	★★★ Very strong
RV diameter × Sex	t-test	t = 2.685	0.0090	★★ Significant

Age × Pericardial effusion	t-test	t = -2.040	0.0450	★ Weak
sPAP × Age	Pearson	r = 0.209	0.0757	NS
sPAP × Sex	t-test	t = -0.439	0.6618	NS
LV dilation × Pericardial effusion	Chi²	$\chi^2 = 0.000$	0.9969	NS

The table above shows a strong correlation between sPAP and TR grade (rs = 0.716; p < 0.0001), between sPAP and RA area (r = 0.488; p < 0.0001), between IVC dilation (rpb = 0.448; p = 0.0001) and IVC diameter (r = 0.351; p = 0.002). The association between RA dilation and IVC dilation is very strong ($\chi^2 = 23.2$; p < 0.0001). RV diameter is strongly correlated with RA area (r = 0.492; p < 0.0001) and is significantly higher in men (t = 2.685; p = 0.009). sPAP is not significantly correlated with age (r = 0.209; p = 0.076) or with sex (p = 0.662).

Discussion

This study describes the echocardiographic profile of a cohort of 73 patients with documented PAH on Doppler echocardiography (DE). The results highlight several noteworthy findings that merit further discussion.

An older population with a female predominance

The mean age of 61.5 ± 17.8 years and the median of 66 years place our cohort in a notably older bracket than those reported in Western series of idiopathic PAH, where the median age at diagnosis is generally 50 to 55 years.[10,20] This difference is likely explained by the heterogeneous nature of our population: it does not consist exclusively of group 1 (idiopathic) PAH, but rather of multifactorial PAH encompassing postcapillary etiologies (left heart disease, chronic hypertension), thromboembolic causes, and structural heart disease, which typically occur in older patients.

The female predominance (53.4%) is, by contrast, consistent with data from the global literature. Several registries have shown a female-to-male ratio of 2:1 to 4:1 in idiopathic PAH, a predominance attributed to hormonal mechanisms involving estrogens in pulmonary vasoreactivity and smooth muscle cell proliferation.[8,13] In our series, this imbalance remains modest (ratio 1.4:1), reflecting the etiological diversity of our study population.

An elevated sPAP reflecting severe, late-diagnosed PAH

The mean sPAP of 75.2 ± 14.4 mmHg, with values reaching 95 mmHg, is particularly concerning and reflects PAH that is already severe at presentation. This severity at the time of diagnosis is a recurring feature in African series, where the diagnostic delay is often prolonged due to difficulties accessing specialized care, limited awareness among first-line practitioners, and patients' economic constraints.[12,21]

For comparison, the French PAH registry (HTAP-France) reported a mean PAP at catheterization of 55 ± 15 mmHg.[10] The discrepancy between our sPAP values estimated by DE and those obtained by catheterization in other series is expected: DE tends to overestimate sPAP by approximately 10 to 15 mmHg in the presence of severe tricuspid regurgitation.[16] Nonetheless, our results confirm the value of DE as an early screening tool, while underscoring the need for invasive confirmation when feasible.

TR grade as a central marker of hemodynamic severity

Grade III TR was present in 80.8% of patients, with an associated mean sPAP of 75.2 ± 14.4 mmHg, compared with grade I. This near-linear progression of pulmonary pressure with the severity of regurgitation perfectly illustrates the pathophysiology of PAH: chronic pressure overload of the RV distends the tricuspid annulus, worsens the regurgitation, which in turn perpetuates RA and RV dilation in a well-documented vicious cycle.
The very strong correlation between sPAP and TR grade (rs = 0.716; p < 0.0001) in our series is consistent with the work of,[18] who established the relationship between peak tricuspid regurgitation jet velocity and RV systolic pressure, as well as with the ASE guidelines by Rudski et al. (2010) for right heart assessment. This finding supports the systematic quantification of TR whenever PAH is suspected.

The right atrium as a key witness of chronic impact

RA dilation was present in 75.3% of patients, with a mean area of 27.6 ± 8.9 cm² (normal ≤ 18 cm²) and a significant correlation with sPAP (r = 0.488; p < 0.0001), confirming the central role of this parameter in assessing PAH severity. In response to chronically elevated RV filling pressures and tricuspid regurgitation, the RA progressively distends, becoming a sacrificed compliance chamber that helps maintain cardiac output.
Several studies have shown that RA dilation (area > 18 cm²) is an independent predictor of mortality in PAH.[14,15] In our cohort, the vast majority of patients had significant RA dilation, placing this entire population in a high-risk prognostic category and underscoring the urgency of optimized therapeutic management.

The IVC as a reflection of systemic venous hypertension

IVC dilation was present in 56.2% of patients, correlated with sPAP (r = 0.351; p = 0.0023) and very strongly associated with RA dilation ($\chi^2 = 23.2$; p < 0.0001), illustrating the hemodynamic cascade of PAH: pulmonary arterial hypertension → RV pressure overload → RA dilation → systemic venous hypertension → IVC dilation. This pathophysiological sequence, well documented in the literature,[4,17] validates the diagnostic coherence of our approach and reinforces the role of the IVC as a non-invasive window onto RA pressure.
An IVC diameter > 21 mm with inspiratory collapsibility below 50% is associated with an RA pressure greater than 10 mmHg according to ASE guidelines.[4] Our mean IVC value of 23.2 mm, consistent with these criteria, confirms marked systemic venous hypertension in our cohort.

Pericardial effusion: an early marker of severity in younger patients

The observation that patients with pericardial effusion were significantly younger (56.1 vs. 65.0 years; p = 0.045) is an original finding warranting further reflection. In the literature, pericardial effusion is a marker of severity and poor prognosis in PAH.[8,13] It results from the increase in pericardial transmural pressure secondary to right-sided overload and systemic venous hypertension.
The fact that younger subjects are proportionally more affected could be explained by a more active and rapid inflammatory or vascular response in patients with subacute PAH, such as pulmonary embolism or connective tissue disease, as opposed to older patients with chronic, progressively compensated postcapillary PAH. Longitudinal studies would be needed to confirm and refine this hypothesis.

Sexual dimorphism in RV diameter

RV diameter was significantly greater in men (43.4 ± 8.8 mm vs. 41.2 ± 8.0 mm; p = 0.009), consistent with normative data in the literature, which establish higher reference values in men.[19] In contrast, no significant difference in sPAP was observed between the two sexes (p = 0.662), suggesting that despite morphological differences in the RV, the pressure overload imposed on the right heart is equivalent in both groups. This finding is consistent with data from the REVEAL registry in the United States, which found no major prognostic difference related to sex when hemodynamic severity was comparable.[13]

Conclusion

This study confirms that Doppler echocardiography is a powerful, accessible, and relevant tool for the non-invasive assessment of PAH. In our cohort of 73 patients, PAH was severe from the outset, with a mean sPAP of 75 mmHg, a predominance of grade III TR (78.1%), and diffuse involvement of the right heart chambers. The correlations established between sPAP and TR grade (rs = 0.716), RA area (r = 0.488), and IVC diameter (r = 0.351) constitute a body of convergent evidence that validates the reliability of the echocardiographic approach in this clinical context. These findings underscore the importance of early PAH screening by Doppler echocardiography in at-risk populations presenting with unexplained dyspnea, structural heart disease, thromboembolic disease, or connective tissue disease. In resource-limited settings such as sub-Saharan Africa, Doppler echocardiography should be fully integrated into PAH diagnostic algorithms, pending wider availability of right heart catheterization as the gold-standard confirmatory test.

References

1. Galiè N, Humbert M, Vachiery JL, et al. 2015 ESC/ERS Guidelines for the diagnosis and treatment of pulmonary hypertension. *Eur Heart J*. 2016;37(1):67-119. doi:10.1093/eurheartj/ehv317.
2. Vonk-Noordegraaf A, Haddad F, Chin KM, et al. Right heart adaptation to pulmonary arterial hypertension: physiology and pathobiology. *J Am Coll Cardiol*. 2013;62(25 Suppl):D22-D33. doi:10.1016/j.jacc.2013.10.027.
3. Tadic M, Pieske-Kraigher E, Cuspidi C, et al. Tricuspid regurgitation in heart failure: pathophysiology, diagnosis, and treatment. *Heart Fail Clin*. 2023;19(1):55-68. doi:10.1016/j.hfc.2022.08.005.
4. 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. *J Am Soc Echocardiogr*. 2010;23(7):685-713. doi:10.1016/j.echo.2010.05.010.
5. Simonneau G, Montani D, Celermajer DS, et al. Haemodynamic definitions and updated clinical classification of pulmonary hypertension. *Eur Heart J*. 2019;40(19):1545–1553. doi:10.1093/eurheartj/ehz492
6. McLaughlin VV, Archer SL, Badesch DB, et al. ACCF/AHA 2009 expert consensus document on pulmonary hypertension. *J Am Coll Cardiol*. 2009;53(17):1573–1619. doi:10.1016/j.jacc.2009.01.004
7. 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. *J Am Soc Echocardiogr*. 2010;23(7):685–713. doi:10.1016/j.echo.2010.05.010
8. Galiè N, Humbert M, Vachiery JL, et al. 2015 ESC/ERS Guidelines for the diagnosis and treatment of pulmonary hypertension. *Eur Heart J*. 2016;37(1):67–119. doi:10.1093/eurheartj/ehv317
9. Galiè N, Channick RN, Frantz RP, et al. Risk stratification and medical therapy of pulmonary arterial hypertension. *Eur Respir J*. 2022;53(1):1700928. doi:10.1183/13993003.00928-2017
10. Humbert M, Sitbon O, Chaouat A, et al. Pulmonary arterial hypertension in France: results from a national registry. *Am J Respir Crit Care Med*. 2006;173(9):1023–1030. doi:10.1164/rccm.200510-1668OC
11. 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(38):3618–3731. doi:10.1093/eurheartj/ehac237
12. Thienemann F, Sliwa K, Rockstroh JK. HIV and the heart: the impact of antiretroviral therapy - a global perspective. *Eur Heart J*. 2013;34(46):3538–3546. doi:10.1093/eurheartj/eht388
13. Badesch DB, Champion HC, Sanchez MA, et al. Diagnosis and assessment of pulmonary arterial hypertension. *J Am Coll Cardiol*. 2009;54(1 Suppl):S55–S66. doi:10.1016/j.jacc.2009.04.011
14. Raymond RJ, Hinderliter AL, Willis PW, et al. Echocardiographic predictors of adverse outcomes in primary pulmonary hypertension. *J Am Coll Cardiol*. 2002;39(7):1214–1219. doi:10.1016/s0735-1097(02)01744-8
15. Forfia PR, Fisher MR, Mathai SC, et al. Tricuspid annular displacement predicts survival in pulmonary hypertension. *Am J Respir Crit Care Med*. 2006;174(9):1034–1041. doi:10.1164/rccm.200604-547OC
16. 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(7):615–621. doi:10.1164/rccm.200811-1691OC
17. Fijalkowska A, Kurzyna M, Torbicki A, et al. Serum N-terminal brain natriuretic peptide as a prognostic parameter in patients with pulmonary hypertension. *Chest*. 2006;129(5):1313–1321. doi:10.1378/chest.129.5.1313
18. Currie PJ, Seward JB, Chan KL, et al. Continuous wave Doppler determination of right ventricular pressure: a simultaneous Doppler-catheterization study in 127 patients. *J Am Coll Cardiol*. 1985;6(4):750–756. doi:10.1016/s0735-1097(85)80477-0
19. Lang RM, Badano LP, Mor-Avi V, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the ASE and EACVI. *J Am Soc Echocardiogr*. 2015;28(1):1–39. doi:10.1016/j.echo.2014.10.003
20. Olsson KM, Delcroix M, Ghofrani HA, et al. Anticoagulation and survival in pulmonary arterial hypertension: results from the Comparative, Prospective Registry of Newly Initiated Therapies for Pulmonary Hypertension (COMPERA). *Circulation*. 2014;129(1):57–65. doi:10.1161/CIRCULATIONAHA.113.004526
21. Ndiaye MB, Sarr SA, Bodian M, et al. Pulmonary arterial hypertension in Senegal: epidemiological, diagnostic, and clinical course aspects. *Pan Afr Med J*. 2018;30:147. doi:10.11604/pamj.2018.30.147.15764

Copyright: © 2026 The Author(s). **License:** This is an open-access article distributed under the terms of the Creative Commons Attribution 4.0 International License **(CC BY 4.0)**, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited. For more information, visit, visit https://creativecommons.org/licenses/by/4.0/