

Comparison of Pulmonary Vascular Resistance Measurements by Echocardiography and Cardiac Catheterization

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ABSTRACT

Objective: To compare Pulmonary Vascular Resistance (PVR) measurements by echocardiography and cardiac catheterization.

Study Design: Analytical cross-sectional study

Place and Duration of Study: Department of Paediatric Cardiology, Armed Forces Institute of Cardiology & National Institute of Heart Diseases, Rawalpindi Pakistan from Jun to Nov 2024.

Methodology: Consecutive sampling was used to recruit thirty-one patients. Included patients were between the ages of 2 and 60, who were having cardiac catheterization for suspected pulmonary vascular disease or PH. A detailed echocardiographic assessment was performed within 24 hours, including tricuspid regurgitation velocity (TRV) and the velocity-time integral of the right ventricular outflow tract (VTIRVOT). These characteristics were then compared to PVR measurements obtained using catheterization.

Results: Out of 31 cases, 16(51.6%) were males and 15(48.38%) were females with median age of 6.90(22.00-3.00) years. A moderate positive correlation was observed between PVR measured by echocardiography and cardiac catheterization in Wood units (WU) ($r=0.41$; $p=0.02$). This r-value reflects moderate consistency between non-invasive and invasive measurements of PVR. Bland- Strong agreement between the PVR measured by catheterization and echocardiography was demonstrated by Altman analysis. The 78.6% sensitivity and 82.4% specificity were observed for a PVR of >2 WU with a TRV/VTIRVOT threshold of 0.179 (AUC: 0.752, 95% CI: 0.55 to 0.94; $p=0.001$).

Conclusion: Echocardiography can provide a simple, reliable, noninvasive and potentially cost-effective alternative to determine PVR. The strong correlation and agreement between echocardiography-derived and catheterization-based PVR measurements show its potential as a valuable diagnostic tool, reducing the need for invasive testing.

Keywords: Cardiac catheterization, Echocardiography, Pulmonary hypertension, Pulmonary vascular resistance

How to Cite This Article: Saeed M, Akhter K, Sadiq A, Ikram M, Ahmed K, Rafique S. Comparison of Pulmonary Vascular Resistance Measurements by Echocardiography and Cardiac Catheterization. *Pak Armed Forces Med J* 2025; 75(Suppl-7): S1099-S1104.

DOI: <https://doi.org/10.51253/pafmj.v75iSUPPL-7.13464>

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INTRODUCTION

The progressive and potentially fatal condition known as pulmonary hypertension (PH) is characterized by raised pulmonary arterial pressure, which increases mortality and causes right heart failure. It has a variety of causes and makes significant contributions to morbidity and mortality worldwide.¹ According to WHO estimates, PH may impact over 100 million people globally, with over 80% of cases happening in low- and middle-income nations, while younger people are disproportionately afflicted.² Moreover, PH affects about 10% of individuals over 65.³

Pulmonary Vascular Resistance (PVR) is an important parameter for assessing disease severity, guiding treatment, and monitoring outcomes.⁴⁻⁵ It is

measured using two main methods: echocardiography (echo) and cardiac catheterization(cath). Echo is a non-invasive tool that provides valuable information on cardiac structure and function, including estimate of PVR through Doppler-derived calculations such as peak Tricuspid Regurgitant Jet Velocity (TRV,ms) to the right Ventricular Outflow Tract Time-Velocity Integral (VTIRVOT, cm) ratio. In contrast, cardiac cath is an invasive procedure that directly measures of PVR through pulmonary pressures and flow assessment.⁶⁻⁷ Sohail *et al.*, reported that although cardiac cath is the gold standard for PH diagnosis, its routine use is limited by invasiveness, risks, and cost, making echo a safer, more accessible substitute.⁸⁻⁹ However, echo requires high specificity for accurate PH diagnosis and follow-up.¹⁰

Despite widespread use, the accuracy of echocardiographic PVR estimation compared to the gold standard remains unclear, particularly in local populations with limited validation studies.

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Received: 05 May 2025; revision received: 23 May 2025; accepted: 27 Jul 2025

Conflicting data and methodological inconsistencies highlight a significant research gap. To cater this gap this study aims to compare the PVR estimation by cath and echo. Assessing whether echocardiography can be a reliable substitute for PVR assessment is crucial for improving patient care in PH management, guiding treatment decisions, and improving diagnostic protocols due to the invasiveness, restricted accessibility, and increased costs of catheterization, especially in low-resource settings.

METHODOLOGY

This Analytical cross-sectional study was conducted from June 2024 to November 2024 in Paediatric Cardiology department of Armed Forces Institute of Cardiology & National Institute of Heart Diseases, Rawalpindi, after the approval of Institutional Ethical Review Board (letter# 9/2/R&D/2024/313; Date:30th May 2024). Study was conducted by selecting patients using non-probability consecutive sampling.

The sample size of n=31 patients was determined using WHO sample size calculation software, considering 2% prevalence of pulmonary hypertension¹¹, 95% confidence interval and 5% margin of error.

Inclusion Criteria: Patients aged 2 to 60 year, irrespective of gender, undergoing cardiac catheterization for PH or suspected pulmonary vascular disease, with echocardiography performed within 24 hours of cardiac catheterization were included in this study

Exclusion Criteria: Patients with severe valvular heart disease or cardiomyopathy were excluded from the study

Patients who met the inclusion criteria were selected after obtaining informed consent from them or their guardians. Those presenting to the OPD or emergency with symptoms were assessed via cardiac catheterization, followed by echocardiography within 24 hours for PH diagnosis. Cardiac cath was performed under general or local anesthesia through femoral access, measuring various hemodynamic parameters including Pulmonary artery pressures: systolic, diastolic and mean (PASP, PADP, and PAMP), pulmonary capillary wedge pressure (PCWP), mean left atrial pressure (MLAP). Blood gas analysis and an oximetry run were performed. The conventional spectroscopic technique was used to calculate the saturation and PaO₂. The FICK's

equation was used to determine the pulmonary flow (Qp) in liters per minute. Oxygen consumption (VO₂) was assessed. Lafarge *et al.*'s standard normogram was used to measure an assumed oxygen consumption value (in mL/min per square meter) for patients aged ≥3 years. This value was based on heart rate, gender, and age. A formula developed by Lundell *et al.*, (based on gender, height, weight, and heart rate) was applied to patients younger than three years.⁸

Table-I Demographic Characteristics and Echocardiographic Parameters of Study participants(n=30)

Variables		Median(IQR)
Age(year)		6.50(22.00-3.00)
Weight(kg)		16.00(65.00-12.00)
		Frequency(%)
Gender	Male	16 (51.6%)
	Female	15 (48.4%)
Diagnosis	ASD	15 (48.38%)
	VSD	12 (38.70%)
	AVSD	3 (9.67%)
	RA & RV Volume	1(3.22%)
Heart Rate(bpm)		100.97±17.56
Hb(g/ dl)		Mean±SD
		11.35±2.30
Echocardiographic Parameters		Mean±SD
TRV(ms)		3.23 ± 0.63
VTIRVOT(cm)		Median(IQR)
		22.70 (27.10-18.30)
Pulmonary Vascular Resistance(WU)		1.93± 0.61

ASD= Atrial Septal Defect; VSD= Ventricular Septal Defect; PDA=Patent Ductus Arteriosus; RA=Right Atrium; RV= Right Ventricle TRV= tricuspid regurgitation velocity; VTI RVOT=Velocity Time Integral of the Right Ventricular Outflow Tract.

Table-II Assesment of Catheterisation Parameters of Study Participants(n=30)

Cardiac Catheterization Parameters	Mean±SD
mPAP(mmHg)	32.94±14.93
PASP(mmHg)	51.45±21.11
mLAP(mmHg)	12.30±3.45
	Median(IQR)
PADP (mPAP)	14.00(29.00-10.00)
PVO2(ml/ min)	144.00(167.10-127.70)
PAO2(ml/ min)	136.40(157.80-125.30)
VO2(ml/ min)	150.00(60.00-125.00)
Pulmonary Vascular Resistance(WU) [Mean±SD]	1.96± 0.82

PASP=pulmonary artery systolic pressure; mPAP=mean pulmonary artery pressure; mLAP= mean left atrial pressure; Hb=Hemoglobin; PADP=Pulmonary Arterial diastolic Pressure; PVO2 =mixed venous oxygen partial pressure; PAO2 =Alveolar oxygen partial pressure ;VO2 =oxygen consumption

Using a 4, 8, or 10MHz frequency probe, echocardiographic investigations were conducted

using an Acuson SC2000 Prime Siemens equipment. The procedure involved a 24-hour cardiac catheterization and was carried out by a single operator. The patients were placed in the supine posture or left lateral decubitus while either conscious or unconscious sedation was applied. The right ventricular outflow tract time-velocity integral (TVIRVOT)(cm) were obtained by placing a 1-2mm pulsed wave Doppler sample volume in the proximal right ventricular outflow tract within the pulmonary valve when imaged from the parasternal short-axis view. Mean was taken after measuring the VTI of RVOT thrice. Peak tricuspid regurgitant velocity (TRV)(m/s) was determined by using continuous wave Doppler imaging in the parasternal, subcostal or apical four-chamber view. The highest velocity obtained from multiple views was used.⁵

Data analysis was conducted using Statistical Package of Social Sciences (SPSS) version 23.00. The normality of data was explored using the Shapiro-Wilk test, which showed that age, RVOT, Hb, PADP, PVO₂, PAO₂, and VO₂ were not normally distributed therefore, median (IQR) was reported. For normally distributed variables, including weight, HR, TRV, mPAP (mmHg), PASP (mmHg), mLAP (mmHg), and PVR, the Mean±SD was reported. Categorical variables such as gender and disease status were presented as frequency (%). Bland-Altman analysis was performed to find an agreement between echocardiography-derived and catheterization-estimated PVR. The Pearson correlation coefficient was applied to determine the relationship between PVR values obtained through echo and cath. The ROC curve was used to determine the sensitivity and specificity of the TRV/VTIRVOT ratio in predicting PVR > 2 WU, with $p < 0.05$ considered statistically significant.

RESULTS

Thirty-one patients were recruited in this study, with a median age of 6.50(22.00-3.00) years. Among them, 16(51.6%) were males, and 15(48.4%) were females. The most prevalent congenital heart disease was ASD, noted in 15(48.38%) patients, followed by VSD in 12(38.70%) cases. Echocardiographic assessment of our patients indicated a mean tricuspid regurgitation velocity (TRV) of 3.23 ± 0.63 m/s and a mean PVR of 1.93 ± 0.61 (WU) (Figure-1)

The mean pulmonary vascular resistance measured during catheterization was 1.96 ± 0.82 WU. The mean PASP, mPAP, and mLAP were found to be

32.94 ± 14.93 mmHg, 51.45 ± 21.11 mmHg, and 12.30 ± 3.45 mmHg, respectively.

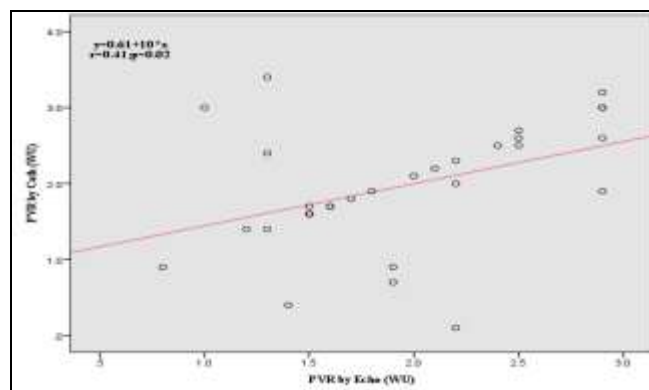


Figure-1: Correlation between PVR CATH and PVRecho

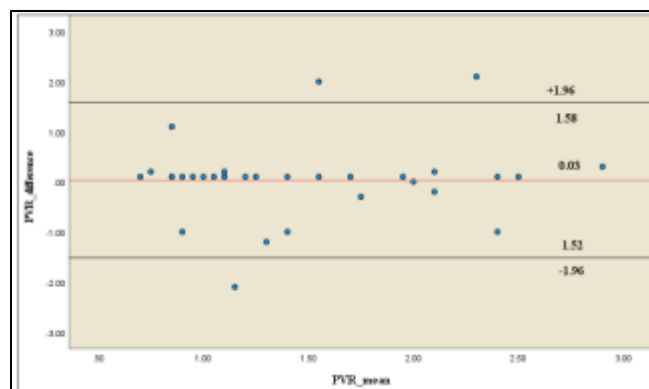


Figure-2: Bland-Altman analysis showing the limits of agreement between PVRECHO and PVR CATH.

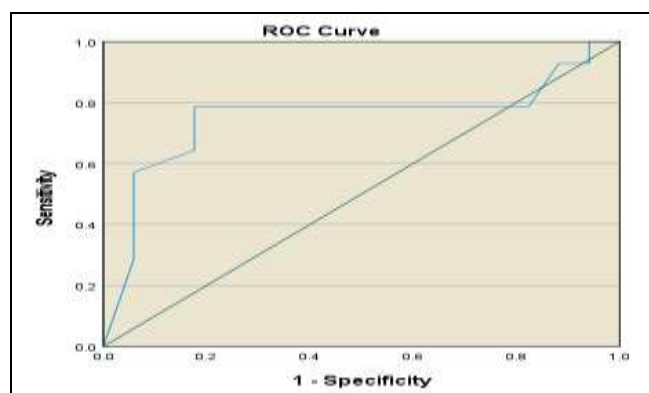


Figure-3: Receiver Operating Characteristic (ROC) curve for predicting PVR > 2 WU

A moderate positive correlation was observed between PVR measured by cath and estimated via echocardiography. The regression equation, $PVRECHO$ (WU) = $0.61 + 10 \times TRV/VTIRVOT$, indicates a moderate correlation between the two methods. The correlation coefficient ($r = 0.41$; $p = 0.02$) shows a slightly positive, but statistically significant

correlation. This r-value reflects moderate consistency between non-invasive and invasive measurements of PVR. The red line in the Bland-Altman plot represents the average difference between the methods, PVRecho and PVRcath. Figure-2 showed strong agreement between these methods, with a mean difference of 0.03. The limits of agreement range from -1.52 to 1.58, with most data falling within this range, depicting an acceptable variability. The majority of values are clustered around the mean, with only a few outliers, supporting the reliability of PVRecho as a non-invasive alternative to PVRcath.

Receiver operating characteristic (ROC) curve was plotted to evaluate the diagnostic performance of PVRecho in comparison to PVRcath using a cutoff value of 2 WU. For a PVR > 2 WU, a TRV/VTIRVOT threshold of 0.179 yielded a sensitivity of 78.6% and a specificity of 82.4%. The area under the curve (AUC) for TRV/VTIRVOT against PVRcath was 0.752, demonstrating a fair level of agreement between the two methods, with a (95% CI: 0.55- 0.94).

DISCUSSION

This study demonstrated that echocardiography-derived PVR values exhibited a significant association and strong agreement with invasive catheter-derived measurements. These findings support the use of echocardiography as a reliable, noninvasive tool for estimating PVR, potentially reducing the need for routine invasive procedures.

It is critical to identify people with increased PVR since novel surgical procedures and pharmaceutical treatments can greatly improve outcomes; progressive monitoring of PVR is essential to evaluate the effectiveness of these treatments.¹² A previous study conducted by Venkateshvaran et al., reported a strong correlation between echocardiographic and catheterization-derived methods ($r=0.635$; $p=0.003$).¹³ Similarly, our study also showed a slight linear correlation between PVRcath and PVRecho ($r=0.41$; $p=0.02$) in Pulmonary hypertension patients. Moreover, this association was independent of age or RVOT diameter.

Electrocardiography, chest radiography, and clinical examination may all indicate the existence of pulmonary hypertension, although their diagnostic sensitivity is low. Pulmonary hypertension is more accurately detected by echocardiography that uses pulsed Doppler in combination with tricuspid or pulmonary regurgitation velocity. These techniques are important for diagnosing PAH associated with

CHD, but they don't provide much information regarding the state of the pulmonary vascular bed or the course of pulmonary vascular disease.¹²

Reasonable agreement between echocardiographic and catheterization-based PVR values was shown by the Bland-Altman analysis in the current investigation. Since the majority of the data points were within the range of agreement, echocardiography could be a good noninvasive substitute. The ROC curve analysis evaluated the diagnostic accuracy of echocardiography for predicting PVR > 2 WU a clinically significant threshold. A TRV/VTIRVOT ratio of 0.179 yielded a sensitivity of 78.6% and a specificity of 82.4%. These results are aligned with previous findings, where a TRV/VTIRVOT cut-off of 0.14 demonstrated 93% sensitivity and 57% specificity in identifying patients with PVR > 2 WU.¹⁴

Previous studies have shown a linear relationship between PVR and TRV/VTIRVOT corrected for indexed RVOT diameter in people with high PVR and a variety of clinical circumstances. Furthermore, noninvasive PVR prediction can be a useful technique for analyzing and monitoring pulmonary vascular changes over time and it is applicable across a wide range of values. Despite the fact that these techniques are critical for diagnosing PAH associated with CHD, they do not show much about the status of the pulmonary vascular bed or the course of pulmonary vascular disease.¹⁵⁻¹⁶ In our study, a correlation coefficient of 0.41 indicates a moderate association, suggesting that while echocardiographic measurements are useful estimates, they may not fully substitute catheterization-based assessments.

In patients without systemic-to-pulmonary artery shunts and with normal or slightly elevated PVR, Abbas *et al.*⁶ found a strong association between catheterization-derived PVR and the TRV/VTIRVOT ratio. This indicator was used for the first time in this study to differentiate among patients with normal and increased PVR. Their findings support the role of echocardiography, particularly the TRV/VTIRVOT ratio, as a useful noninvasive estimate of PVR in children with CHD, potentially reducing the need for invasive catheterization for initial assessment and follow-up. Their findings are supported by current research.

Arafuri *et al.*,¹⁷ exclaimed, Doppler-derived TRV/VTIRVOT ratio was studied for its value in measuring PVR in patients with CHD and various left-

to-right shunts accompanied with severe PAH, as well as its potential as an operability indicator. Moreover, regardless of age, body surface area (BSA), or RVOT diameter, Pande *et al.*,¹⁸ mentioned a substantial linear connection between catheterization-derived PVR and Doppler-derived TRV/VTIRVOT ($R^2=0.562$, $p=0.008$). Based on their findings, they proposed a regression equation for estimating PVR noninvasively: $PVR (WU) = 0.61 + 10 \times TRV/VTIRVOT$. In alignment with their results and based on our data, we also used this simplified equation for noninvasive estimation of PVR.

In a sample of 150 patients, Abbas *et al.*⁶ validated the TRV/VTIRVOT technique by comparing invasive PVR measures with Doppler-derived estimates and this linear regression analysis demonstrated a strong correlation ($r=0.76$, $p<0.0001$, $Z=0.92$). This study concluded that the TRV/VTIRVOT ratio is a reliable noninvasive marker for identifying patients with elevated PVR, with values greater than 0.275 predictive of $PVR > 6$ WU. In contrast, current study observed a lower threshold of 0.179 for predicting $PVR > 2$ WU, with a sensitivity of 78.6% and specificity of 82.4%. This variation in cut-off values may be attributed to differences in clinical context and catheterization protocols. Notably, our cohort exclusively included patients with CHD and unobstructed pulmonary flow, whereas the aforementioned study analyzed a heterogeneous population pooled from multiple sources.

In our study, Pulmonary blood flow (QP) during catheterization was measured via Fick method. This contrasts with Volodarsky *et al.*,¹⁹ who employed the thermodilution method to measure cardiac output for PVR calculation. However, when there are intracardiac shunts present, thermodilution may not be accurate; in these situations, the Fick approach is better. While direct measurement of oxygen consumption offers greater accuracy, we used an indirect estimation depends upon the Lafarge and Meittinen tables. Currently, there is no universal consensus regarding the superiority of direct versus indirect oxygen consumption measurement methods.

This work is significant because it establishes echocardiography, specifically the TRV/VTIRVOT ratio, as a safe, noninvasive method of measuring PVR in CHD patients. This study supports the use of Doppler-based assessments in clinical settings to reduce reliance on invasive procedures, particularly for initial screening, monitoring treatment response,

and follow-up, by demonstrating a moderate correlation and high diagnostic accuracy when compared to invasive catheterization. This has significant effects on enhancing patient safety, cost-effectiveness, and accessibility while treating pulmonary hypertension in patients with congestive heart failure.

LIMITATION OF STUDY

TR is not always present in CHD patients with PAH. Therefore, the index and equation cannot be applied to patients who do not have TR. The study's sample size was somewhat small, which might have limited how far the results can be applied. Small sample numbers can impact the accuracy of the results and raise the possibility of bias. The accuracy of the data may be impacted by operator dependence in echo-based PVR measurement. Ensuring precise alignment and thorough tracing are crucial. By employing the mean of numerous measurements and single-operator service for echocardiography, we attempted to reduce these impacts. In this investigation, cardiac catheterization and echocardiography were not done at the same time. However, due to inadequate positioning of the echocardiogram, simultaneous readings may become erroneous.

ACKNOWLEDGEMENT

We would like to express our appreciation to the Comdt. Exec Dir. AFIC/NIHD & R & D department for their assistance and input in finishing the study paper.

CONCLUSION

This study concluded that while not a perfect substitute for catheterization, PVRecho offers a simple, reliable, non-invasive and potentially cost-effective alternative. This study contributes to the growing body of evidence supporting the use of echocardiography as a valuable tool for assessing PVR in patients with PH.

Conflict of Interest: None.

Funding Source: None.

Authors' Contribution

Following authors have made substantial contributions to the manuscript as under:

MS & KA: Data acquisition, data analysis, critical review, approval of the final version to be published.

AS & MI: Study design, data interpretation, drafting the manuscript, critical review, approval of the final version to be published.

KA & SR: Conception, data acquisition, drafting the manuscript, approval of the final version to be published.

Authors agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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