Original Article

Correlating Invasive and Echocardiographic Hemodynamic Measures in Patients with Heart Failure with Reduced Ejection Fraction

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Abstract

Background: **The management of heart failure with reduced ejection fraction (HFrEF) has advanced with the use of invasive hemodynamic markers such as cardiac power output (CPO) and pulmonary artery pulsatility index (PAPI). Simultaneously, interest has grown in noninvasive prognostic indicators, especially the left ventricular outflow tract velocity–time integral (LVOT-VTI).**

Objective: **This study aimed to assess the correlation between invasive and echocardiographic parameters, particularly CPO, PAPI, and LVOT-VTI, in patients with HFrEF.**

Methods: **This cross-sectional, single-center study included inpatients and outpatients with chronic stable or acutely decompensated HFrEF. Individuals with cardiogenic shock (CS) were excluded. Right heart catheterization (RHC) and echocardiography were performed on the same day. Statistical significance was defined as a two-sided p value < 0.05.**

Results: **Significant correlations were observed between invasive and echocardiographic CPO ($r = 0.737$, $p < 0.001$) and PAPI ($r = 0.604$, $p < 0.05$). LVOT-VTI showed significant correlation with CPO measured via pulmonary artery catheter (PAC) ($r = 0.469$, $p < 0.01$). Echocardiographic PAPI was measurable in only 16 patients. A sensitivity analysis of those receiving inotropes showed an even stronger correlation between invasive and echocardiographic CPO ($r = 0.812$, $p = 0.005$).**

Conclusion: **Echocardiographic and invasive hemodynamic parameters showed significant correlations in HFrEF, particularly CPO, supporting echocardiography as a potential alternative for hemodynamic assessment. However, the low feasibility of noninvasive PAPI measurement highlights important limitations in this population.**

Keywords: **Systolic Heart Failure; Hemodynamic Monitoring; Heart Rate; Echocardiography.**

Introduction

Heart failure (HF) is a growing global health issue, affecting an estimated 60 million people worldwide. Advancements in cardiac care and improved patient survival have contributed to the rising prevalence of this syndrome.[1] The use of pulmonary artery catheter (PAC) for hemodynamic assessment remains a cornerstone in HF management. These invasive measurements are essential for diagnosing and optimizing treatment strategies in both chronic and decompensated HF, providing insights into disease progression and therapeutic response.[2,3] PAC use has increased in recent years, guided by a systematic approach that prioritizes parameters with the greatest prognostic impact, such as cardiac power output (CPO) and pulmonary artery pulsatility index (PAPI). These hemodynamic parameters have become essential tools in determining the need for mechanical circulatory support in patients with cardiogenic shock (CS).[4] Although initially studied in CS, CPO and PAPI also have significant prognostic value in chronic and acutely decompensated heart failure with reduced ejection fraction (HFrEF).[5-7]

Research on noninvasive measures with prognostic value in patients with heart disease has also expanded, aiming to shorten evaluation time and improve safety. Among echocardiographic parameters, the left ventricular outflow tract velocity–time integral (LVOT-VTI) has demonstrated good prognostic value in observational studies.[8,9] The LVOT-VTI is a Doppler-derived echocardiographic measurement that quantifies the distance blood travels through the LVOT during systole, serving as a key parameter in assessing stroke volume, cardiac output (CO) and overall hemodynamic status.[10,11]

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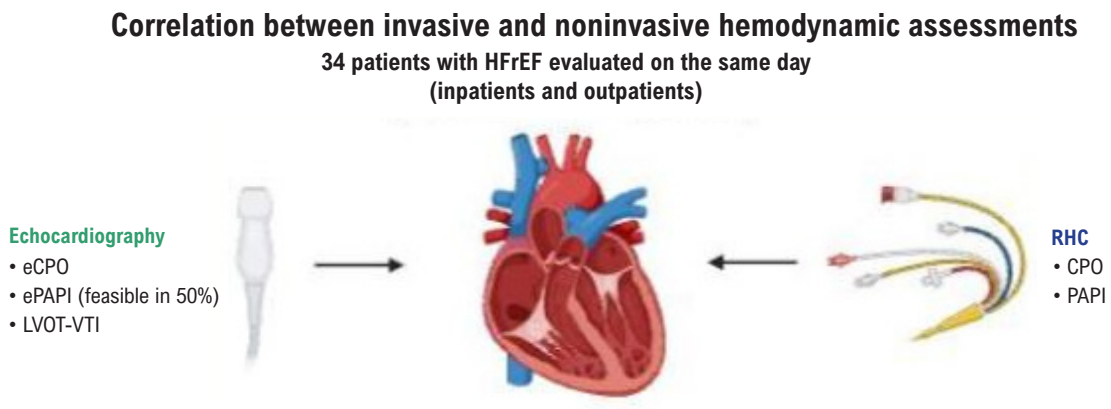
Central Illustration: Correlating Invasive and Echocardiographic Hemodynamic Measures in Patients with Heart Failure with Reduced Ejection Fraction



Echocardiography	RHC	r
eCPO	CPO	0.73
*eCPO	CPO	0.82
ePAPI	PAPI	0.60
LVOT-VTI	CPO	0.46

**Patients receiving inotropic support.*

Strong correlation for CPO

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CPO: cardiac power output; e-PAPI: echocardiography-derived pulmonary artery pulsatility index; PAPI: pulmonary artery pulsatility index; LVOT-VTI: left ventricle outflow tract velocity time integral.

The correlation between invasive and noninvasive measurements in acute decompensated HF-related CS has been recently investigated, revealing associations between CPO and PAPI measured via PAC and echocardiography.[12] Similarly, the relationship between PAC-derived and echocardiographic parameters has been explored in patients with chronic HF;[13,14] however, these studies have not focused on parameters such as CPO, PAPI, and LVOT-VTI.

Therefore, the objective of this study was to evaluate the correlation between invasive and noninvasive measurements in inpatients and outpatients with chronic stable or decompensated HFrEF, with particular emphasis on CPO, PAPI, and LVOT-VTI. The main findings of this study are summarized in the Central Illustration.

Methods

This is a single-center cross-sectional study conducted at a university hospital in the South Region of Brazil. Participants indicated for routine evaluation by the HF team were recruited by actively searching outpatient and inpatient lists. Recruitment occurred between May 2023 and June 2024, using phone calls and direct contact with hospitalized patients. All patients were invited to undergo echocardiography before or after right heart catheterization (RHC). The research protocol was approved by the institutional ethics committee, and written informed consent was obtained from all participants.

The study included patients aged 18 years or older with a diagnosis of HFrEF (left ventricular ejection fraction < 40%). Patients were excluded if there were technical difficulties in obtaining echocardiographic measurements or if RHC measurements could not be performed. Additionally, patients with CS, active infection, those on mechanical ventilation, or undergoing hemodialysis were excluded. Patients who received diuretics or vasodilators between the two evaluation methods were also excluded.

The study protocol involved performing a comprehensive echocardiographic assessment by two experienced echocardiographers (SVB and WRM) within the shortest possible time before or after the invasive hemodynamic evaluation using a PAC. All evaluations occurred within a six-hour interval between the two examination methods.

RHC

The invasive hemodynamic evaluation was performed using RHC with PAC inserted via micropuncture technique of the right basilic vein or the right internal jugular vein. The zero-reference

level was established at the mid-axillary line, and measurements were recorded at end-expiration. The operator performing RHC was blinded to the echocardiographic measurements.

CO was assessed using the thermodilution technique. Central venous pressure (CVP), pulmonary pressures, and pulmonary artery occlusion pressure (PAOP) were determined as the mean values over eight cardiac cycles. CPO was calculated using mean arterial pressure (MAP) according to the formula: CPO = CO × MAP / 451. The PAPI was calculated as: PAPI = PASP — PADP / CVP, where PASP and PADP indicate pulmonary artery systolic pressure and pulmonary artery diastolic pressure, respectively. The remaining derived hemodynamic parameters were calculated using the formulas described in Table 1.

Echocardiography

Transthoracic echocardiography was performed by two experienced examiners (Berger SV e Menegazzo WR), blinded to the results of the RHC measurements. All examinations were performed using the Philips Epiq CVx system. To assess interobserver variability, a preliminary study was conducted in a subset of 10 patients, who were not included in the 34 patients of the study sample. The evaluation was performed by the two echocardiographers. Echocardiography was performed on the same day of the RHC, with the shortest possible interval between the two tests, and patients were required to rest for at least 10 minutes before the exam. Measurements obtained via echocardiography followed the guidelines established by the American Society of Echocardiography.[10]

CO was calculated using the formula: CO = LVOT-VTI × LVOT area × heart rate. CPO was derived using the formula: CPO = CO × MAP / 451, and PAPI was calculated using the formula: PAPI = (PASP − PADP) / eRAP [estimated right atrial pressure], where eRAP indicates estimated right atrial pressure. CVP was estimated based on the maximum diameter of the inferior vena cava (greater or less than 2.1 cm) and its variability during inspiration (greater or less than 50%). Ejection fraction was assessed using Simpson's method. PASP, PADP, mean pulmonary artery pressure (MPAP), and other derived formulas are described in Table 2.

Statistical analysis

Table 1 – PAC-derived parameters

Parameter	Formula
CPO	CO × MAP / 451
PAPI	(PASP − PADP) / CVP
TPG	MPAP − PAOP
PVR	TPG / CO

CO: cardiac output; CPO: cardiac power output; CVP: central venous pressure; MAP: mean arterial pressure; MPAP: mean pulmonary artery pressure; PADP: pulmonary artery diastolic pressure; PAOP: pulmonary artery occlusion pressure; PAPI: pulmonary artery pulsatility index; PASP: pulmonary artery systolic pressure; PVR: pulmonary vascular resistance; TPG: transpulmonary gradient.

The normality of continuous variables was assessed using histograms and the Shapiro–Wilk test. All continuous variables showed a normal distribution and were therefore expressed as mean ± standard deviation. Categorical variables were expressed as absolute and relative frequencies (n, %). Comparisons between groups were performed using the unpaired Student's t-test. For correlation analysis between invasive hemodynamic and echocardiographic measures, Pearson's correlation coefficient was used, as all continuous variables were normally distributed. Interobserver agreement was evaluated using intraclass correlation coefficient analysis, based on a two-way random-effects model, assessing consistency and average measurements. A statistical significance level of 5% ($p < 0.05$) was adopted. All statistical analyses were conducted using SPSS Statistics for Windows, Version 26.0 (IBM Corp., Armonk, NY, United States).

A sample of 27 patients was required based on the correlation between LVOT-VTI and stroke volume measured by PAC of $r = 0.52$ (95%, $p < 0.01$) in a prior study,[15] assuming an alpha value of 0.05 and a power of 80% to identify a similar correlation in our sample. The correlation between LVOT-VTI and CPO assessed via PAC was not utilized in the sample size calculation because such data were not available in the published literature at the time of the study design.

Results

Between May 2023 and June 2024, a total of 34 patients were included in the study. The mean age was 50.8 ± 6.2 years, 61.7% were male, and 82.4% of patients had nonischemic HF. The mean left ventricular ejection fraction was 21.1% ± 6.6%. The majority of the sample consisted of inpatients (67.4%), whereas 26.5% were receiving inotropic therapy at the time of the examinations. Table 3 provides an overview of the patient baseline characteristics and ongoing therapies.

All patients receiving inotropic support were classified as having INTERMACS III HF, as their mean serum lactate was 1.4 ± 0.2 mmol/L, and none of them were receiving vasopressor support (clinical characteristics in Supplementary Table S1).

Regarding clinical characteristics reviewed retrospectively in medical records, 24 of 34 patients underwent cardiopulmonary exercise testing, and the mean peak oxygen consumption was 15.9 ± 5.3 mL/kg/min; 9 patients (26.4%) underwent orthotopic heart transplantation, and 5 (14%) died.

PAC parameters

PAC measurements demonstrated elevated pulmonary pressures and impaired left and right ventricular hemodynamic parameters. Detailed invasive hemodynamic measurements are presented in Table 4.

Echocardiographic parameters

Echocardiography demonstrated marked left ventricular dilation and mild right ventricular enlargement, accompanied by severe biventricular systolic dysfunction. Pulmonary artery pressures were elevated, consistent with the advanced HF profile of the study population. Detailed echocardiographic structural, functional, and hemodynamic parameters are

Table 2 – Echocardiographic parameters

Parameter	Formula
CO	LVOT-VTI × LVOT area × heart rate
CPO	CO × MAP / 451
PAPI	(PASP − PADP) / eRAP
PASP	TR peak gradient + CVP
PADP	4 × (end-diastolic pulmonary regurgitation velocity)² + CVP
MPAP	4 × (protodiastolic pulmonary regurgitation velocity)² + CVP

CO: cardiac output; CPO: cardiac power output; CVP: central venous pressure; eRAP: estimated right atrial pressure; LVOT: left ventricular outflow tract; LVOT-VTI: left ventricular outflow tract velocity–time integral; MAP: mean arterial pressure; MPAP: mean pulmonary artery pressure; PADP: pulmonary artery diastolic pressure; PAPI: pulmonary artery pulsatility index; PASP: pulmonary artery systolic pressure; TR: tricuspid regurgitation.

Table 3 – Baseline characteristics and ongoing therapies

Parameter	Mean (SD) or Median (IQR)	n (%)
Age (years)	50.8 ± 6.2	34 (100)
Male sex (%)	61.7	34 (100)
Nonischemic HF etiology (%)		28 (82.4)
Heart rate (bpm)	78.2 ± 15.2	34 (100)
Systolic blood pressure (mmHg)	101.6 ± 17.9	34 (100)
Diastolic blood pressure (mmHg)	66.2 ± 13.2	34 (100)
MAP (mmHg)	73.4 ± 11.0	34 (100)
Weight (kg)	77.0 ± 18.9	34 (100)
Height (cm)	167.6 ± 10.6	34 (100)
Use of CRT-D (%)		3 (8.8)
ARNI (%)		10 (41.2)
ACE inhibitors/ARBs (%)		14 (38.2)
Mineralocorticoid receptor antagonists (%)		31 (91.2)
Beta-blockers (%)		31 (91.2)
Sodium-glucose cotransporter-2 inhibitors (%)		22 (64.7)
Digoxin (%)		17 (50.0)
Hydralazine plus nitrates (%)		6 (17.6)

ACE: angiotensin-converting enzyme; ARB: angiotensin II receptor blocker; ARNI: angiotensin receptor–neprilysin inhibitor; CRT-D: cardiac resynchronization therapy with defibrillator; HF: heart failure; IQR: interquartile range; SD: standard deviation; MAP: mean arterial pressure.

presented in Table 5.

Echocardiographic PAPI was successfully obtained in 16 of the 34 enrolled patients, whereas it was possible to estimate MPAP and PADP in 18 and 16 patients, respectively, owing to the absence of pulmonary regurgitation required for their calculation.

Interobserver agreement was excellent for CPO, LVOT-VTI, left ventricular ejection fraction, and E/e' ratio, whereas PAPI and fractional area change showed poor agreement (Supplementary Table S2).

Correlation between invasive and noninvasive hemodynamic measures

This study demonstrated a correlation between the invasive and echocardiographic assessments of CPO (Figure 1). The CPO measured via PAC showed a positive correlation with CO obtained by echocardiography ($r = 0.616$, $p < 0.001$). Similarly, a positive correlation was observed between CPO measured via PAC and LVOT-VTI (Figure 2). There was also a positive correlation between CPO assessed by PAC and left ventricular ejection fraction, with a correlation coefficient of 0.365 ($p < 0.05$).

Regarding the assessment of right ventricular function,

Table 4 – PAC parameters

Measure	Mean (± SD)	n (%)
CO (L/min)	4.2 ± 1.2	34 (100)
CI (L/min/m²)	2.2 ± 0.5	34 (100)
PAOP (mmHg)	18.6 ± 6.2	34 (100)
CPO (W)	0.7 ± 0.2	34 (100)
PAPI	3.2 ± 1.5	34 (100)
RVSWI (mmHg·mL/m²)	7.7 ± 2.9	34 (100)
CVP (mmHg)	7.7 ± 4.1	34 (100)
PASP (mmHg)	41.2 ± 10.3	34 (100)
MPAP (mmHg)	27.8 ± 7.7	34 (100)
PADP (mmHg)	20.8 ± 6.2	34 (100)
PVR (Wood units)	2.3 ± 1.5	34 (100)
Transpulmonary gradient (mmHg)	9.3 ± 4.7	34 (100)
CVP over PAOP	0.42 ± 0.2	34 (100)

CI: cardiac index; CO: cardiac output; CPO: cardiac power output; CVP: central venous pressure; MPAP: mean pulmonary artery pressure; PADP: pulmonary artery diastolic pressure; PAOP: pulmonary artery occlusion pressure; PAPI: pulmonary artery pulsatility index; PASP: pulmonary artery systolic pressure; PVR: pulmonary vascular resistance; RVSWI: right ventricular stroke work index; SD: standard deviation.

correlations were detected between PAPI assessed by PAC and by echocardiography (Figure 3). Furthermore, PAPI measured via PAC correlated with tricuspid annular plane systolic excursion (TAPSE), with $r = 0.553$ ($p < 0.01$), and right ventricular free wall strain, with a negative correlation coefficient of $r = -0.344$ ($p < 0.05$). Additionally, a positive correlation was observed between PAPI assessed by PAC and peak systolic velocity at the tricuspid annulus (S′ wave), with a correlation coefficient of 0.356 ($p < 0.05$).

With respect to LVOT-VTI, in addition to the previously mentioned correlation with PAC-derived CPO, correlation was observed between LVOT-VTI and CO measured by PAC ($r = 0.538$, $p < 0.01$), as well as cardiac index measured by PAC ($r = 0.542$, $p < 0.01$).

Sensitivity analysis

A sensitivity analysis was conducted to evaluate the population receiving inotropic support. The correlation between invasive and echocardiographic CPO was higher in this subgroup compared to the overall population: $r = 0.812$ ($p = 0.008$). Additionally, a sensitivity analysis was also conducted in patients with CPO < 0.6 W. In this subset, the correlation between invasive and echocardiographic CPO was $r = 0.704$ ($p = 0.005$). In both groups, it was not possible to assess the correlation between invasive and echocardiographic PAPI because noninvasive PAPI measurement was feasible in only 3 of the 9 patients included in this subgroup.

Discussion

The assessment of correlations between invasive and noninvasive hemodynamic measurements aims to identify echocardiographic parameters that reliably reflect prognostically significant hemodynamic variables, such as CPO and PAPI. This approach intends to provide a more practical and safer diagnostic strategy for patients with advanced HF by minimizing the evaluation time and risks associated with invasive procedures.

As reported by Burstein et al., in a retrospective cohort study, noninvasive CPO demonstrated a prognostic role in patients admitted to cardiac intensive care units. The study analyzed nearly 5000 patients, with 50% presenting with decompensated HF. CPO below 0.89 ± 0.37 W was associated with greater hospital mortality.[16] Echocardiographic PAPI also demonstrated prognostic value, being associated with 60-day mortality in acute decompensated HF-related CS (hazard ratio 2.96, 95% confidence interval 1.23–7.17, $p = 0.02$).[12] LVOT-VTI, as demonstrated by Kentzer et al. and Machado et al., also demonstrated significant prognostic value among cardiology patients, including those with HF and acute myocardial infarction.[9,17]

In our study, regarding left ventricle function parameters, invasively measured CPO and CPO estimated by echocardiography showed the strongest correlation. This association was further enhanced in the subgroup of patients receiving inotropic support. Furthermore, CPO measured by PAC also demonstrated correlation with CO assessed through echocardiography and with the LVOT-VTI and left ventricular ejection fraction.

Table 5 – Echocardiographic measurements

Parameter	Mean (SD)	n (%)
LA diameter (mm)	48 (± 10.7)	34 (100)
LA reservoir strain	9.9 (± 7.2)	32 (94.1)
LA conduit strain	−6.7 (± 4.6)	32 (94.1)
LA contractile strain	−3.3 (± 4.8)	32 (94.1)
LV strain (%)	−6.6 (± 3.2)	34 (100)
LVDD (mm)	71.8 (± 7.7)	34 (100)
LVSD (mm)	64.8 (± 8.5)	34 (100)
LVDV (mL)	236.9 (± 69.7)	34 (100)
LVSV (mL)	189.2 (± 62.3)	34 (100)
LVEF (%)	21.1 (± 6.6)	34 (100)
CO (L/min)	3.6 (± 1.2)	34 (100)
CPO (W)	0.61 (± 0.35)	34 (100)
LVOT-VTI (cm)	12.8 (± 4.3)	34 (100)
E/e′ ratio	18.0 (± 7.8)	32 (94.1)
PAPI	4.4 (± 3.7)	16 (47)
CVP (mmHg)	7.29 (± 4.8)	34 (100)
PASP (mmHg)	44.1 (± 14.8)	27 (79.4)
MPAP (mmHg)	28.8 (± 9.8)	18 (52.9)
PADP (mmHg)	20.2 (± 7.7)	16 (47)
TAPSE (mm)	16.1 (± 4.1)	34 (100)
S′ wave velocity (cm/s)	8.8 (± 2.1)	34 (100)
RV free wall strain (%)	−14.0 (± 7.3)	33 (97)
FAC (%)	27.8 (± 9.3)	33 (97)
RV basal diameter (mm)	43.8 (± 8.4)	34 (100)

CO: cardiac output; CPO: cardiac power output; CVP: central venous pressure; E/e′: ratio of early mitral inflow velocity (E) to early diastolic mitral annular velocity (e′); FAC: fractional area change; LA: left atrium; LV: left ventricle; LVDD: left ventricular diastolic diameter; LVSD: left ventricular systolic diameter; LVDV: left ventricular diastolic volume; LVSV: left ventricular systolic volume; LVEF: left ventricular ejection fraction; LVOT-VTI: left ventricular outflow tract velocity–time integral; MPAP: mean pulmonary artery pressure; PADP: pulmonary artery diastolic pressure; PAPI: pulmonary artery pulsatility index; PASP: pulmonary artery systolic pressure; RV: right ventricle; S′: systolic velocity of the tricuspid annulus; SD: standard deviation; TAPSE: tricuspid annular plane systolic excursion.

Regarding PAPI, the results revealed correlation between its invasive measurement via PAC and its echocardiographic estimation, as well as with TAPSE. The correlations between PAPI and S′ wave velocity and between PAPI and right ventricular free wall strain were weaker. Based on literature review, this appears to be the first study to evaluate the correlations between invasive CPO and PAPI with echocardiographic measurements in patients with HFrEF.

Stein et al. and Temporelli et al. reported a strong correlation between invasive and echocardiographic hemodynamic measurements in patients with chronic HF; however, CPO, PAPI and LVOT-VTI were not included among the variables analyzed in these studies.[13,14] Frea et al. published the largest study to date investigating the correlation between invasive and echocardiographic hemodynamic parameters in CS.[9] Their population consisted of patients with CS secondary to decompensated HF. In our cohort, 26.5% of patients were receiving inotropic support and classified as having INTERMACS III HF. Although our population had normal lactate levels and were not on vasopressor therapy at the time of evaluation, these two populations may share similar pathophysiological conditions. In the study by Frea et al.,[12] correlation between the two methods showed a Pearson coefficient $r = 0.82$, $p < 0.001$ for CPO. The

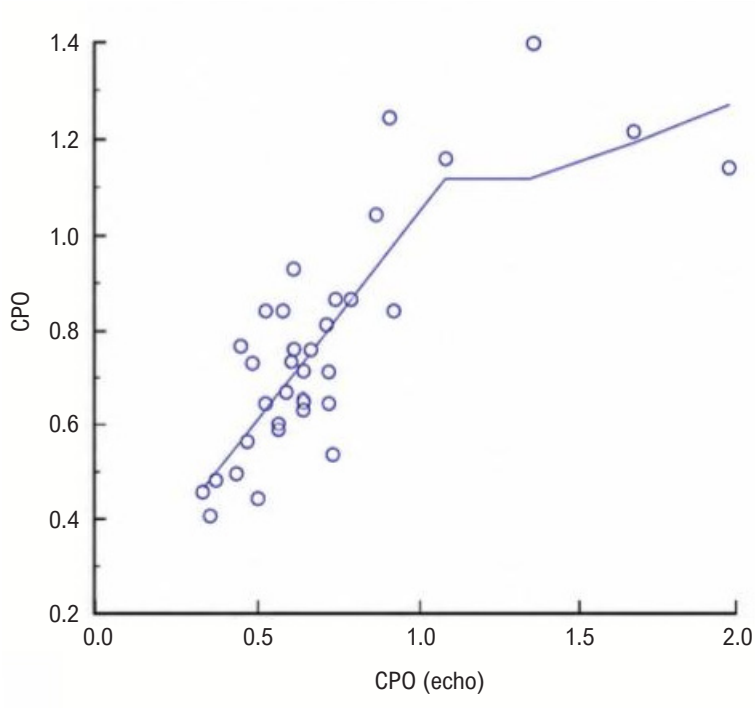

Figure 1 – *Correlation analysis for CPO assessed by PAC and echocardiography. r = 0.737 (p < 0.001).*

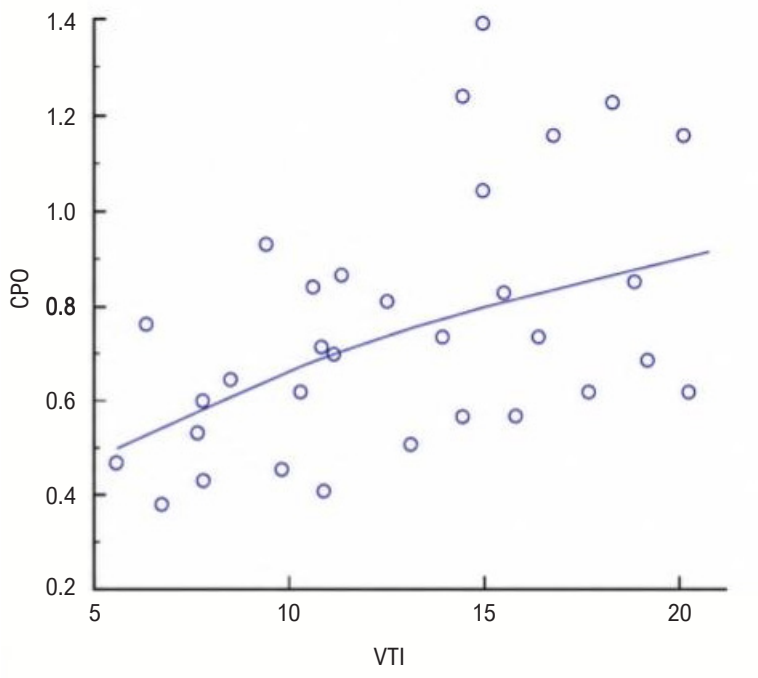

Figure 2 – *Correlation analysis for CPO and LVOT-VTI. r = 0.469 (p < 0.01)*

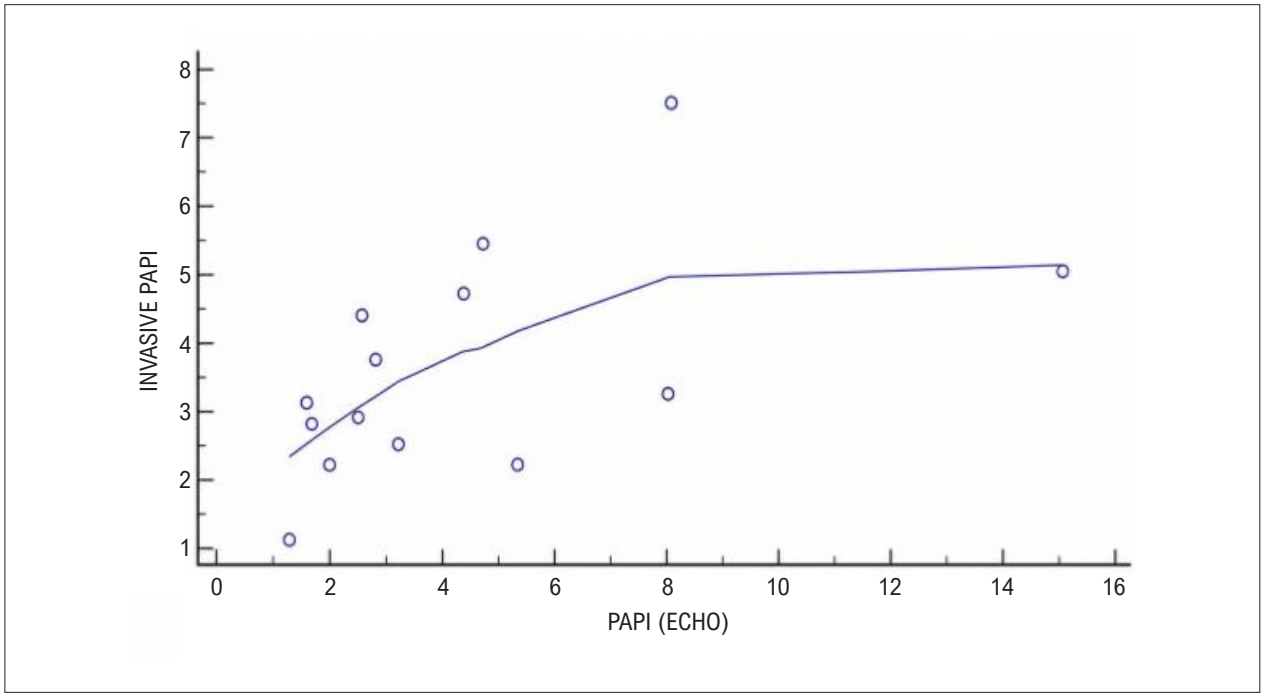

Figure 3 – *Correlation analysis for PAPI assessed by PAC and echocardiography. r = 0.604 (p < 0.05)*

results of our study were in line with these findings, especially among patient in INTERMACS III, given that CPO measured by PAC and echocardiography showed the strongest correlation between the two methods in our population (r = 0.73, p < 0.001), which was even greater in patients receiving inotropic support (r = 0.81, p = 0.008). Our study reaffirms the strength of correlation between invasive and echocardiographic CPO and its reproducibility, extending the findings observed in CS to patients with chronic and acutely decompensated HFrEF.

Frea et al.[12] also demonstrated a strong correlation between invasive and echocardiographic PAPI assessments. One notable difference between the two studies lies in the reproducibility of echocardiographic PAPI. In the cited study, PAPI estimation was feasible in 97 out of 101 patients, whereas in our study, it was achievable in only 16 of 34 patients. Pulmonary regurgitation is a rare finding on echocardiography in patients with pulmonary hypertension, consequently reducing the accuracy of methods that evaluate measures derived from it.[18] There is also significant variability in PAC-derived PAPI thresholds capable of predicting adverse outcomes across different studied populations. Consequently, the cutoff values for PAPI differ between CS and HF.[19,20] For this reason, low sensitivity of pulmonary regurgitation and the distinction in PAPI thresholds between different populations may have contributed to the differences in reproducibility observed between the two studies. Therefore, based on our findings, we conclude that echocardiographic PAPI may not be a reliable diagnostic parameter in patients with chronic and acutely decompensated HF due to its low reproducibility.

This study presents some limitations. First, the size of the analyzed population is a significant aspect to emphasize, especially after the publication by Frea et al.,[12] which occurred during the final recruitment phase of our study. Furthermore, the heterogeneity of our cohort represents another limitation, as our study includes individuals with diverse hemodynamic conditions and varying requirements for therapeutic support. To reduce population heterogeneity, we performed a sensitivity analysis among patients receiving inotropic support, attempting to exclude patients with less severe disease. The inability to obtain echocardiographic PAPI in part of the sample represents another limitation. Lastly, the extended interval between the two assessment methods may also be regarded as a potential limitation.

Conclusion

This study demonstrated significant correlations between invasive and noninvasive hemodynamic measures in patients with HFrEF, particularly for prognostic parameters such as CPO, PAPI, and LVOT-VTI. CPO showed the strongest agreement between methods, especially in patients receiving inotropic support. While PAPI correlated across techniques, its limited reproducibility by echocardiography reduces its clinical utility in this population.

Author Contributions

Conception and design of the research: Barbato JPR, Scolari FL, Berger SV, Menegazzo WR, Araujo GN, Valle

FH, and Wainstein RV; acquisition of data: Barbato JPR, Berger SV, Menegazzo WR, Crivelaro PAM, Amon AB; analysis and interpretation of the data: Barbato JPR, Scolari FL, Machado GP, Berger SV, Menegazzo WR, Amon AB, Araujo GN, Valle FH, Wainstein RV; statistical analysis: Barbato JPR, Scolari FL, Machado GP, Araujo GN; obtaining financing: Barbato JPR; writing of the manuscript: Barbato JPR, Scolari FL, Araujo GN, Valle FH, and Wainstein RV; critical revision of the manuscript for intellectual content: Barbato JPR, Scolari FL, Machado GP, Berger SV, Menegazzo WR, Crivelaro PAM, Amon AB, Araujo GN, Valle FH, and Wainstein RV.

Potential Conflict of Interest

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

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Study Association

This article is part of the master's thesis submitted by João Pedro da Rosa Barbato, from the Federal University of Rio Grande do Sul.

Ethics Approval and Consent to Participate

This study was approved by the Ethics Committee of Hospital de Clínicas de Porto Alegre under the protocol number 5920490. All the procedures in this study were in accordance with the 1975 Helsinki Declaration, updated in 2013. Informed consent was obtained from all participants included in the study.

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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*Supplemental Materials

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