

Peripheral Venous Pressure Measurements in Patients With Acute Decompensated Heart Failure (PVP-HF)

BACKGROUND: Accurate assessment of volume status is essential in diagnosis and guidance of decongestive therapy in patients with acute heart failure. We sought to compare peripheral venous pressure (PVP) with central venous pressure (CVP), as well as other invasive hemodynamic measurements, in patients hospitalized with an acute heart failure syndrome.

METHODS AND RESULTS: PVP-HF (Peripheral Venous Pressure Measurements in Patients With Acute Decompensated Heart Failure) was a single-center prospective study, which enrolled patients admitted with acute heart failure, regardless of ejection fraction or disease pathogenesis. PVP and intracardiac pressures were obtained by transducing a peripheral intravenous and pulmonary artery catheter, respectively, after zeroing at the phlebostatic axis. Data were compared using Pearson's correlation coefficient and Bland–Altman plots. A total of 30 patients (median age 64 years, 73% male, 30% ischemic pathogenesis) were enrolled. Mean ejection fraction was 31%, and 60% had moderate or greater right ventricular dysfunction. Median PVP was 9.5 (6–17) mmHg, CVP was 8.5 (6–18) mmHg, and pulmonary capillary wedge pressure was 18 (14–21) mmHg. PVP and CVP were found to be highly correlated ($r=0.947$), while PVP and pulmonary capillary wedge pressure were found to be moderately correlated ($r=0.565$). The mean difference between PVP and CVP was 0.4 mmHg and between PVP and pulmonary capillary wedge pressure was 7.5 mmHg.

CONCLUSIONS: In patients with acute heart failure syndromes, a simple assessment of PVP demonstrates a high correlation with CVP. These findings suggest that PVP may be useful in the standard bedside clinical assessment of volume status in these patients to help guide decongestive therapy.

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■ pulmonary artery ■ venous pressure

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WHAT IS NEW?

- In this study, we sought to compare peripheral venous pressure (PVP) with central venous pressure, as well as other invasive hemodynamic measurements.
- PVP and intracardiac pressures were obtained by transducing a peripheral intravenous and pulmonary artery catheter, respectively, after zeroing at the phlebostatic axis.
- PVP and central venous pressure were found to be highly correlated ($r=0.947$), while PVP and pulmonary capillary wedge pressure were found to be moderately correlated ($r=0.565$).
- Thus, a simple assessment of PVP demonstrates a high correlation with central venous pressure in patients presenting to the hospital for acute heart failure.

WHAT ARE THE CLINICAL IMPLICATIONS?

- Accurate assessment of volume status is essential in the diagnosis and guidance of decongestive therapy in patients with acute heart failure.
- Our findings suggest that PVP may be useful in the standard bedside clinical assessment of volume status in patients presenting with acute heart failure to help guide management.
- PVP can be particularly valuable in patients presenting to the emergency department with shortness of breath and in cases when jugular venous examination is limited because of body habitus, patient positioning, or practitioner inexperience.

Acute decompensated heart failure remains one of the most predominant reasons for hospital admission and is associated with a substantial economic burden on the healthcare system.¹ The majority of admissions for decompensated heart failure result from volume overload, leading to signs and symptoms of pulmonary and systemic congestion. Thus, the management strategy is centered around achieving effective decongestion primarily through the use of loop diuretics.

Current standard of care advocates that diuresis be guided by a comprehensive evaluation of volume status using clinical symptoms, bedside examination, laboratory findings, and invasive hemodynamics (if necessary). The most important part of the bedside examination is the evaluation of the jugular venous pressure, which serves as an estimate of right atrial pressure. Unfortunately, measurement of jugular venous pressure is frequently overlooked or misinterpreted,² and $\approx 50\%$ of patients are discharged with persistent congestion, thereby, contributing to high rates of readmission.³ This observation suggests that additional mechanisms to assess volume status at the bedside are warranted.

One possible alternative is to directly assess peripheral venous pressure (PVP) by transducing a peripheral intra-

venous line (IV) as a way to estimate central venous pressure (CVP). Although a potentially promising addition to the current bedside assessment, the correlation between PVP and CVP has not been studied specifically in a heart failure population. To further examine this relationship, we sought to compare PVP and CVP in patients hospitalized with an acute heart failure syndrome.

METHODS

PVP-HF (Peripheral Venous Pressure Measurements in Patients With Acute Decompensated Heart Failure) was a single-center prospective study conducted between September 2014 and February 2015 at our institution. Patients >18 years old who were admitted with an acute heart failure syndrome with invasive central hemodynamic monitoring were enrolled. Patients either underwent right heart catheterization in the cardiac catheterization laboratory or were being managed in the heart failure intensive care unit with an indwelling pulmonary artery catheter. All patients were required to have an existing peripheral IV prior to enrollment. Patients were included regardless of ejection fraction or pathogenesis of heart failure. No peripheral or central venous lines were permitted to be placed solely for the purposes of this study. All patients or legal surrogate decision makers provided written informed consent prior to enrollment. Patients who did not consent and those with upper extremity venous pathology were excluded. The study protocol was approved by the institutional review board at the Cleveland Clinic Foundation.

Prior to study enrollment, peripheral venous access was obtained using an 18 to 22 gauge IV in the upper extremity. Internal jugular central venous access was obtained, and a 7-French pulmonary artery (PA) catheter was advanced into the PA using standard technique under fluoroscopic guidance. The tip of the PA catheter was positioned in the PA with the CVP port in the right atrium or superior vena cava. Prior to obtaining invasive measurements, baseline clinical and demographic data were recorded. Subsequently, CVP, PA, and pulmonary capillary wedge pressure (PCWP) were measured at end expiration using standard pressure transducers after being zeroed at the phlebostatic axis. Cardiac output and index were measured using the assumed Fick method.

PVP was then immediately measured by connecting the pressure line of the transducer directly to the peripheral IV while the pressure transducer remained zeroed at the phlebostatic axis. The patient's arm was placed parallel to the patient such that the position of the peripheral IV was at the level of the mid chest. Continuity of the peripheral IV line with the central venous system was confirmed by demonstrating augmentation of the venous pressure waveform after manual circumferential occlusion of the extremity proximal to the catheter. If the pressure waveform failed to augment appropriately, data were not collected, and the patient was documented for study purposes as a technique failure.

Statistical Analysis

Continuous variables are expressed as median (interquartile range) and categorical variables as n (%). The primary outcome of the study was the degree of correlation between CVP and PVP measurements. Pearson's correlation coefficients

were calculated to assess this correlation. Bland–Altman plots were developed to assess agreement between the different venous pressure modalities. PVP and PCWP were similarly compared. Linear regression was used to test the association between peripheral IV gauge and venous pressures.

All statistical tests were 2-sided, and *P* values <0.05 were considered significant. Statistical analysis was performed using Stata (version 13, StataCorp LP, College Station, TX).

RESULTS

A total of 30 patients were enrolled in the study, 7 with preserved ejection fraction (>50%) and 23 with reduced ejection fraction (<50%). Four patients were excluded because of peripheral catheter malfunction, where the peripheral waveform failed to augment on manual compression proximal to the site of insertion. Baseline clinical characteristics are seen in the Table. The

Table. Baseline Characteristics

Variable	Value (n=30)
Age, y	64 (54–68)
Male	22 (73%)
Ischemic pathogenesis	9 (30%)
BMI, kg/m ²	26 (23–30)
LV ejection fraction, %	25 (20–33)
RV ≥ moderate dysfunction	18 (60%)
Mitral regurgitation ≥3+	7 (23%)
Tricuspid regurgitation ≥3+	6 (20%)
HR, beats per minute	86 (77–95)
Systolic BP, mmHg	102 (95–111)
Diastolic BP, mmHg	60 (52–65)
Mean BP, mmHg	87.3 (83.3–95.3)
PVP, mmHg	9.5 (6–17)
CVP, mmHg	8.5 (6–18)
Systolic PAP, mmHg	46 (36–55)
Diastolic PAP, mmHg	20 (16–24)
Mean PAP, mmHg	31 (26–37)
PCWP, mmHg	18 (14–21)
Fick CO, L/min	5.2 (4.2–6.3)
Fick CI, L/min/m ²	2.7 (2.2–3.2)
SVR	992 (780–1254)
TPG, mmHg	12 (9–15)
PVR	2.3 (1.6–2.7)
SvO ₂ , %	64 (60–69)
NT-proBNP, pg/mL*	5811 (1721–13 000)

BMI indicates body mass index; BP, blood pressure; CI, cardiac index; CO, cardiac output; CVP, central venous pressure; HR, heart rate; LV, left ventricle; NT-proBNP, N-terminal pro-B-type natriuretic peptide; PAP, pulmonary arterial pressure; PCWP, pulmonary capillary wedge pressure; PVP, peripheral venous pressure; PVR, pulmonary arterial vascular resistance; RV, right ventricle; SvO₂, mixed venous oxygen saturation; SVR, systolic vascular resistance; and TPG, transpulmonary gradient.

*n=27.

median age was 64 years, 73% were male, and 30% had an ischemic pathogenesis of heart failure. Median ejection fraction was 25%, and 60% had moderate or greater right ventricular dysfunction on echocardiography. On average, invasive monitoring demonstrated elevated intracardiac pressures, mild pulmonary hypertension, and preserved cardiac output; the median PVP was 9.5 mmHg, CVP was 8.5 mmHg, and PCWP was 18 mmHg. Figure 1 demonstrates an example of PVP and CVP pressure tracings in a patient with heart failure and elevated intracardiac pressures.

Three patients had an 18-gauge peripheral IV, 17 had a 20-gauge IV, and 10 had a 22-gauge iv. There was no association between peripheral IV gauge and PVP (β , -2.72; 95% confidence interval, -6.45 to 1.02; *P*=0.15) or peripheral IV gauge and the PVP CVP difference (β , 0.06; 95% confidence interval, -1.14 to 1.03; *P*=0.92). There were 11 patients (37%) with hand or wrist IV placement; similarly, this was not associated with PVP (β , 0.47; 95% confidence interval, -4.48 to 5.43; *P*=0.85) or hand/wrist location and the PVP CVP difference (β , 0.35; 95% confidence interval, -1.03 to 1.73; *P*=0.61).

Scatter plots of invasive hemodynamics are seen in Figure 2. PVP and CVP values were highly correlated in these patients with acute heart failure (*r*=0.95). Bland–Altman plots (Figure 3) demonstrated good agreement between PVP and CVP values, with a mean difference of 0.4 mmHg. As anticipated, there was less correlation between PVP and PCWP values (*r*=0.57). Mean difference between PVP and PCWP was 7.5 mmHg. PVP performed well in identifying patients without elevated filling pressures; only 1 of 15 patients with PVP <10

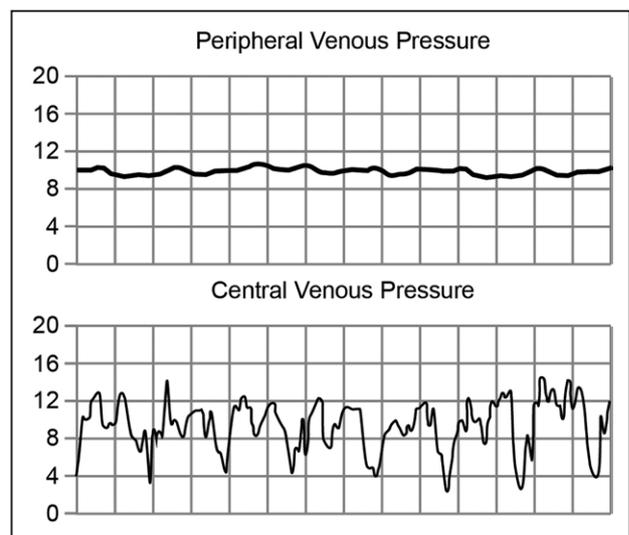


Figure 1. Clinical example.

Example of peripheral venous pressure (PVP) and central venous pressure (CVP) in a patient who was admitted for heart failure. The mean of both pressure measurements was 10 mmHg.

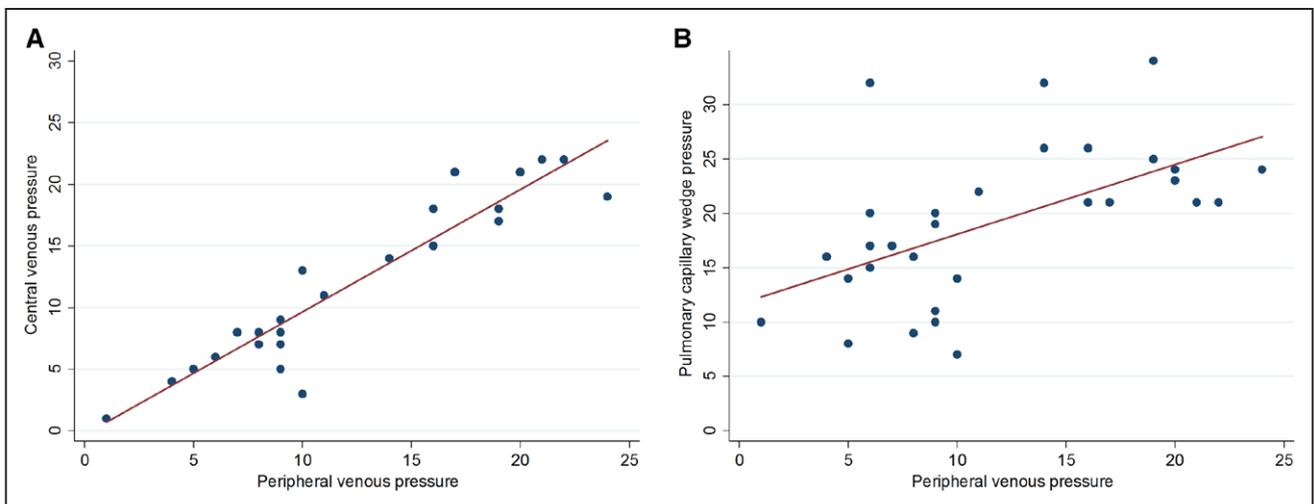


Figure 2. Scatter plot.

Scatter plot demonstrating the correlation between peripheral venous pressure (PVP) and central venous pressure (CVP; **A**) and PVP and pulmonary capillary wedge pressure (PCWP; **B**). Pearson's $r=0.95$ and 0.57 , respectively.

mmHg had PCWP >20 mmHg. PVP >10 had a 100% positive predictive value in identifying patients with PCWP ≥ 20 ($n=13$). PA diastolic pressure was found to be highly correlated with PCWP ($r=0.85$).

DISCUSSION

In the present study, we demonstrate a high correlation between CVP and PVP measurements in patients admitted with acute decompensated heart failure. Prior studies have demonstrated reasonable correlation between CVP and PVP in other patient populations including noncardiac transplant,^{4,5} gastrointestinal surgical,⁶⁻⁸ neurosurgical,^{9,10} and pediatric patients.¹¹ Studies in children with cardiac disease are mixed, with poor correlation seen in congenital heart disease¹² but good

correlation seen while on cardiopulmonary bypass¹³ and in those with Fontan circulation.¹⁴ To our knowledge, this is the first study to validate this process in a cohort of adults admitted with an acute heart failure syndrome. In addition, most prior studies used larger gauge peripheral IVs (16- to 18-gauge), while our study predominantly used smaller 20- to 22-gauge IVs, which are more commonly placed in clinical practice. The IVs used in our study were placed for other purposes, a feature that is unique to prior studies.

Notably, patients in our study were included with normal ejection fraction, as well as predominantly right ventricular dysfunction. These groups have previously been identified as pathologically diverse and at high risk for subsequent mortality and readmission after an index hospitalization.^{15,16} Careful in-hospital management of

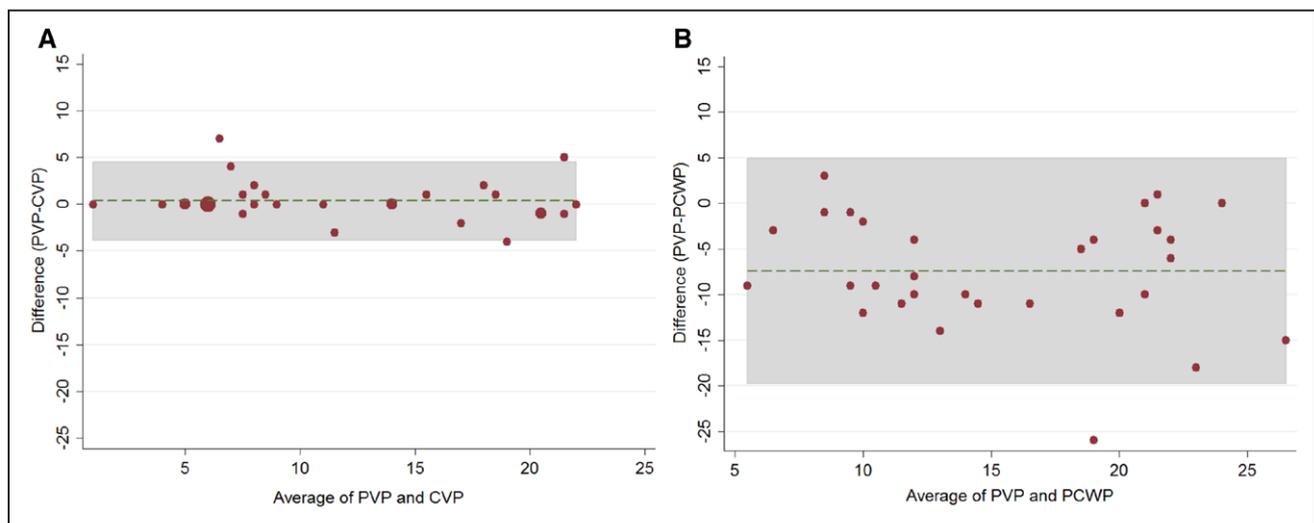


Figure 3. Bland–Altman plot.

Bland–Altman plot depicting mean difference for peripheral venous pressure (PVP) versus central venous pressure (CVP) and PVP versus pulmonary capillary wedge pressure (PCWP; 0.4 and 7.1 mmHg, respectively) and limits of agreement.

these distinct cohorts, including aggressive diuresis and implementation of guideline-directed medical therapy, is likely to have a direct impact on short and long-term clinical outcomes. Inclusion of these patients in our study adds to the generalizability of PVP assessment among a diverse population of patients admitted with an acute heart failure syndrome.

Four patients (12% of the overall population) were excluded because of peripheral IV malfunction. The cause of these technical failures is uncertain; however, it is likely that these peripheral IVs directly abutted a venous valve or there was thrombus within the lumen of the line. This can be remedied by reinsertion of a new peripheral IV and subsequent remeasurement. Prior to assessment of PVP, clinicians should be reminded to assess the augmentation of the waveform with proximal compression to ensure proper catheter position and correlation with CVPs.

It is important to note that PVP and CVP are not direct correlates of PCWP (or left ventricular end-diastolic pressure), as corroborated by this study. There was only moderate correlation between PVP and PCWP ($r=0.57$). This was previously addressed in a seminal study of 1000 patients with chronic heart failure who underwent right heart catheterization as a part of transplant evaluation, where modest correlation was seen between right atrial pressure and PCWP ($r^2=0.62$).¹⁷ A right atrial pressure >10 mm Hg predicted a PCWP >22 mm Hg, with a positive predictive value of 88%. However, only full PA catheter hemodynamic assessment allows for the recognition of patients with a disproportionate right atrial/PCWP ratio.

Clinical Implications

This study has significant clinical implications. PVP can be used to aid in the differential diagnosis of patients presenting to the emergency department with shortness of breath. PVP may be especially useful when jugular venous examination is limited because of body habitus or patient positioning. Practitioners less adept at assessing intracardiac pressures on physical examination may also be aided by PVP measurement. However, practitioners must be careful to not merely rely on this data point but to use it in conjunction with other means to judge volume status to increase the fidelity of the clinical assessment.

Restoration of clinical euvolemia is an essential aspect of managing patients with acute heart failure syndromes, both in an effort to recover functional status and to improve short- and long-term outcomes. However, owing to the potential difficulty in accurately assessing volume status at the bedside, as well as subclinical hemodynamic congestion, several patients are discharged with elevated filling pressures.³ It is this hypothesis that has driven the invention of implantable hemodynamic monitors in heart failure. A more accurate assessment of filling pressures at

the time of discharge may be able to decrease readmission rates, which approach 25% to 30% within the first few months after hospitalization.^{15,18} Although an invasive hemodynamic assessment is indicated to guide management when there is uncertainty regarding volume status, this approach is not feasible in all patients owing to its inherent risks. Therefore, the PVP may serve as a valuable inpatient clinical surrogate of CVP, thus, eliminating the need for central venous catheterization in some patients.

Limitations

Although our data support a significant correlation between PVP and CVP in patients hospitalized with acute heart failure syndromes, this must be interpreted with some limitations in mind. Namely, this was a single-center study and the generalizability to a larger multicenter population of patients with heart failure would need to be validated. Only one paired measurement of PVP and CVP was obtained at one point in time, as opposed to studying this correlation at several time points during the hospitalization. Accurate pressure measurements depend on correct patient and transducer positioning. In addition, one must be cautious of interpreting PVP or CVP as reliable surrogates for left-sided filling pressures because several variables may lead to discordant findings. Finally, our study was not designed to correlate PVP assessment with physical examination findings, treatment response, or clinical end points.

Conclusions

In patients with acute heart failure syndromes, a simple assessment of PVP demonstrates a high correlation with CVP and a moderate correlation with PCWP. These findings have clinical implications for the inpatient management of patients with acute heart failure and may be added to the standard bedside clinical assessment of volume status to help guide management.

DISCLOSURES

None.

AFFILIATIONS

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FOOTNOTE

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