Research Square

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Comparative Analysis of Transthoracic Echocardiographic and Invasive Hemodynamic Assessment of Pulmonary Artery Pressures in Acute Pulmonary Embolism Patients Undergoing Thrombectomy

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Abstract

Purpose:

Transthoracic echocardiography (TTE)-derived pulmonary artery systolic pressure (PASP) is often used to estimate right ventricular (RV) afterload in acute pulmonary embolism (PE), yet its agreement with invasive measures during catheter-directed thrombectomy (CDT) remains unclear.

Methods:

We retrospectively analyzed adults with acute PE who underwent CDT and had a TTE at least 48 hours before CDT. PASP was compared with invasively measured mean pulmonary artery pressure (mPAP) and peak pulmonary artery pressure (pPAP). Agreement was assessed using Bland–Altman analysis, intraclass correlation coefficient (ICC), Pearson correlation coefficient (r) and weighted κ. Sub-analyses were performed for studies ≤ 12 hours and ≤ 24 hours apart, as well as PAP pressure ranges < 30 mmHg, 30–44 mmHg, 45–59 mmHg and ≥60 mmHg.

Results:

TTE-derived PASP was consistently higher than mPAP (mean bias − 12.8 mmHg; ICC 0.006; r = 0.15) with negligible categorical agreement. In contrast, PASP demonstrated substantially better agreement with pPAP (mean bias + 2.6 mmHg; ICC 0.49; r = 0.52) and fair categorical agreement (κ = 0.31). Agreement between PASP and pPAP improved modestly when TTE was performed within 12–24 hours of CDT. Pressure-range analyses showed PASP most closely aligned with higher pPAP values and overvalued lower invasive pressures.

Conclusion:

In acute PE, TTE-derived PASP correlates poorly with invasive mPAP but aligns more closely with pPAP, supporting the physiologic premise that PASP reflects instantaneous systolic pressure rather than mean pulmonary vascular load. These findings highlight the limitations of using PASP alone to assess RV afterload in acute PE and underscore the need for multimodal risk stratification. Further studies comparing TTE and CDT pressures to clinical outcomes are warranted to better understand their prognostic value.

INTRODUCTION

Pulmonary embolism (PE) is a potentially fatal cardiorespiratory emergency that has the potential to elevate pulmonary vascular resistance and right ventricle (RV) afterload, resulting in RV strain and dysfunction. Assessment of RV function and hemodynamics in this context is beneficial to determine

risk stratification and the need for advanced therapies, like catheter-directed thrombectomy (CDT) or thrombolysis.

The European Society of Cardiology (ESC) and American Heart Association (AHA)/American College of Cardiology (ACC) provide similar risk stratification models for acute PE, incorporating clinical parameters, biomarkers, and imaging findings. By integrating hemodynamic stability, imaging evidence of RV dysfunction, and biomarkers, PE is classified into low, intermediate-low, intermediate-high, and high risk [1]. Intermediate risk PE poses a diagnostic and therapeutic challenge, as these patients are hemodynamically stable but exhibit imaging evidence of RV strain and/or biochemical evidence of myocardial injury. Current validated predictive models, such as the Pulmonary Embolism Severity Index (PESI), simplified (sPESI), and Bova score, help quantify 30-day mortality and PE-related complications, but are unhelpful in predicting the risk of acute decompensation [2–5]. The Pulmonary Embolism Progression (PEP) score and Composite Pulmonary Embolism Shock (CPES) score have been formulated to identify patients at higher risk for decompensation, utilizing multivariable data points across clinical, biomarker, and imaging inputs [6, 7].

In current practice, TTE serves as the first-line, non-invasive modality for assessing RV function and estimation of pulmonary artery systolic pressure (PASP), in conjunction with computed tomographic angiography (CTA) to assess RV size and clot burden. PASP, estimated by tricuspid regurgitant jet velocity and an assumed right atrial pressure, is considered a surrogate for RV afterload. PASP and tricuspid annular planar systolic excursion (TAPSE) are often cited variables of interest in risk stratification models, prognosis, and can inform the clinical decision-making process [8–10]. Prior studies evaluating the correlation between TTE-derived PASP and invasive measurements on right heart catheterization (RHC) have been conducted primarily in patients with chronic pulmonary hypertension or structural cardiac disease [11–19]. The agreement between PASP measured by TTE and RHC in this patient demographic has reported intra-class correlation coefficients (ICC) and Pearson correlation coefficients ranging from approximately 0.55 to 0.90 [11–13, 17, 18, 20]. Extrapolating these findings from RHC to acute PE is problematic, however, given the acute nature of PE, as well as the dynamic nature of right ventricular compliance. CDT has emerged as a key intervention, particularly in intermediate-risk PE, offering rapid removal of clot burden. It offers a unique way to obtain real-time, invasive pulmonary artery pressure (PAP) measurements in this population. Furthermore, it provides an opportunity to compare noninvasive (TTE-derived) and invasive (catheter-derived) hemodynamic data.

Based on prior studies, [11–13, 17, 18, 20] we expected poor-to-moderate agreement between TTE-derived PASP and CDT PAPs. This hypothesis is justified by the dynamic physiology of acute PE, where pulmonary pressures can fluctuate based on evolving right ventricular strain [1]. Understanding the degree and pattern of agreement between noninvasive and invasive (mPAP, pPAP) measurements will help clinicians interpret TTE data and understand the labile hemodynamic nature of acute PE within the pulmonary vasculature.

METHODS

Study Design and Setting

We conducted a retrospective cohort study at a 350-bed community-based tertiary teaching hospital. Data was obtained from the institutional Pulmonary Embolism Response Team (PERT) Registry between March 1, 2020, and July 31, 2025. The registry was approved by the institutional review board, with a waiver of informed consent.

PERT Registry Population

Our PERT registry is comprised of adult (≥ 18 years) patients diagnosed with acute PE confirmed by CTA Chest. Patients are identified through the inpatient discharge database via ICD-10 discharge codes for PE. De-identified data pertaining to the patients' hospital course are extracted from the electronic medical records, stored, and managed using REDCap, a secure, HIPAA-compliant web platform, in our institutional PERT registry.

Study Population

Included subjects were isolated from our PERT registry by identifying patients who underwent CDT during the same hospitalization and had a TTE with documented PASP estimation performed within 48 hours prior to CDT. Exclusion criteria included patients who underwent catheter-directed thrombolysis, patients whose initial TTE was obtained after CDT, and those with omitted pressure data on CDT.

Variables of Interest

Extracted variables of interest for our study included: 1) patient demographics (age, sex, body mass index (BMI)); 2) PE severity indices (PESI, sPESI); 3) TTE parameters (date/time of study, estimated PASP); and 3) catheter-based measurements (date/time of thrombectomy, pre-thrombectomy mPAP, and pPAP).

Outcomes

The primary outcome was the level of agreement between invasive mPAP and pPAP, and TTE-derived PASP. Two secondary outcomes were evaluated: agreement between invasive PAPs and TTE-derived PASP at different time intervals between TTE and CDT (≤ 12 hours and ≤ 24 hours) and agreement across PAPs at prespecified ranges (< 30 mmHg, 30–44 mmHg, 45–59 mmHg, and ≥ 60 mmHg) [16, 21].

Statistical Analysis

Descriptive statistics were calculated for all demographic variables. Agreement between invasive PAPs and TTE-derived PASP was evaluated using multiple modalities: 1) Bland-Altman analysis, displaying

bias (mean difference) and 95% limits of agreement between invasive PAPs and TTE-derived PASP; 2) intra-class correlation coefficient (ICC) to assess reliability (ICC < 0.5 indicates poor reliability, 0.5–0.75 moderate, 0.75–0.9 good, and > 0.9 excellent); 3) Pearson correlation coefficient (r), simplified kappa (κ), and weighted κ to quantify any linear association and assess any categorical agreement.

A prior sample-size estimation, based on an expected ICC = 0.55 ± 0.20, α = 0.05, and power = 0.80, indicated a minimum of 48 paired measurements were required to detect significant agreement [22].

Ethical Considerations

The study involved no direct patient interaction. All data were de-identified prior to analysis. No external funding was received, and the authors report no conflicts of interest.

RESULTS

Study Population

A total of 66 patients with acute PE who underwent CDT were identified within our institutional PERT registry (Table 1). Of these, 47 patients met inclusion criteria with a TTE performed 48 hours prior to CDT. Two patients lacked documented pPAP values, leaving 45 patients for pPAP analysis.

Table 1
Patient Demographics and Baseline Characteristics

Variable	TTE vs mPAP (n = 47)	TTE vs pPAP (n = 45)
Age, years (mean ± SD)	64.5 ± 15.1	64.4 ± 14.9
Body mass index (kg/m²)	32.3 ± 8.2	32.2 ± 8.3
Sex, male n (%)	21 (44.7%)	21 (46.7%)
Sex, female n (%)	25 (53.2%)	23 (51.1%)
Sex, other n (%)	1 (2.1%)	1 (2.2%)
PESI score median [IQR]	105 [80–135]	101 [80–132]
sPESI score median [IQR]	2.0 [1–3]	2.0 [1–3]

Table 1 shows the baseline characteristics of the cohort (n = 47). Included subjects had a mean age of 64.5 ± 15.1 years and a mean BMI of 32.3 ± 8.2 kg/m^2. The cohort was relatively equally divided among the sexes with 25 (53.2%) female, 21 (44.7%) male, and 1 (2.1%) other. The cohort reflected an intermediate- to high-risk PE population with a median PESI 105 [IQR 80–135] and sPESI 2 [IQR 1–3].

Comparison of Invasive mPAP and TTE-Derived PASP

As shown in Table 2 and Fig. 1, TTE PASP values were consistently higher than invasive mPAP values across the cohort. The mean TTE-derived PASP was 48.0 ± 15.5 mmHg versus a mean invasive mPAP of 35.3 ± 9.1 mmHg, corresponding to a mean bias = -12.8 mmHg ($p < 0.0001$, n = 46 due to one missing invasive pressure difference). Bland–Altman analysis demonstrated a wide 95% Limits of Agreement (LoA) -46 to + 20 mmHg, indicating substantial variability and a tendency to overestimate. The ICC between mPAP and PASP was ICC = 0.006, indicating no reliability, with a r = 0.15 (p = 0.32) (Table 2). Weighted κ demonstrated negligible categorical agreement; a κ = 0.03 overall, κ = 0.17 ≤ 12 h and κ = 0.13 ≤ 24 h (Table 2). Overall, 28 patients (60%) had higher PASP on TTE, 12 (26%) had equivalent values, 7 (15%) had lower PASP than mPAP (Table 2).

Table 2
Agreement and Correlation Between CDT PAPs and TTE-Derived PASP

Parameter	**mPAP vs TTE PASP (n = 47)**	**pPAP vs TTE PASP (n = 45)**
Mean TTE PASP (mmHg)	48.0 ± 15.5	48.4 ± 15.7
Mean Invasive Pressure (mmHg)	35.3 ± 9.1	51.0 ± 11.4
Mean Bias (Δ = Invasive – TTE)	-12.8 mmHg (n = 46*, p < 0.0001)	+ 2.6 mmHg (p = 0.21)
95% Limits of Agreement (mmHg)	−46 to + 20	−24 to + 29
Pearson Correlation (r)	0.15 (p = 0.32)	0.52 (p = 0.0002)
Intraclass Correlation Coefficient (ICC)	0.006 (p = 0.32)	0.49 (p < 0.001)
Weighted κ (Overall)	0.03 (p = 0.79)	0.31 (p = 0.03)
Weighted κ (≤ 12 h subset)	0.17 (n = 34, p = 0.33)	0.38 (n = 31, p = 0.02)
Weighted κ (≤ 24 h subset)	0.13 (n = 38, p = 0.36)	0.36 (n = 35, p = 0.03)
Proportion with TTE > Invasive	36/47 (76.6%)	23/45 (51%)
Proportion Equal	0	1/45 (2.2%)
Proportion TTE < Invasive	11/47 (23.4%)	29/45 (64.4%)
** n = 46 due to one missing invasive pressure difference*		

Pressure-range stratification demonstrated that agreement between invasive mPAP and TTE-derived PASP were highly variable (Table 3). When mPAP was < 30 mmHg (n = 11), TTE-derived PASP rarely fell within the same category; 40% PASP values < 30 mmHg matched invasive mPAP. The majority of mPAP cases < 30 mmHg had matched TTE-derived PASP falling into higher categories (81.81%). As mPAP increased to 30–44 mmHg (n = 26), category agreement improved modestly, with 6 of 14 PASP values matching invasive mPAP. In the 45–59 mmHg range (n = 9), 4 of the 21 PASP values corresponded in the same range. Only 1 patient had a mPAP ≥ 60 mmHg and no matched cases. Overall categorical

agreement remained poor, as reflected by simple κ = -0.0329 (95% CI -0.1923 to 0.1283) and weighted κ = 0.0305 (95% CI -0.1196 to 0.1806).

Table 3
Concordance Table Between CDT-Measured mPAP and TTE-Derived PASP

TTE-Derived PASP	CDT-Measured mPAP				
Frequency **Percent** **Row Pct** **Col Pct**	**1:<30**	**2:30–44**	**3:45–59**	**4:60+**	**Total**
1:<30	2 4.26 40.00 18.18	3 6.38 60.00 11.54	0 0.00 0.00 0.00	0 0.00 0.00 0.00	5 10.64
2:30–44	4 8.51 28.57 36.36	6 12.77 42.86 23.08	3 6.38 21.43 33.33	1 2.13 7.14 100.00	14 29.79
3:45–59	4 8.51 19.05 36.36	13 27.66 61.90 50.00	4 8.51 19.05 44.44	0 0.00 0.00 0.00	21 44.68
4:60+	1 2.13 14.29 9.09	4 8.51 57.14 15.38	2 4.26 28.57 22.22	0 0.00 0.00 0.00	7 14.89
Total	11 23.40	26 55.32	9 19.15	1 2.13	47 100.00

No significant association was identified between demographic characteristics and PE severity indices and the degree of agreement.

Comparison of Invasive pPAP and TTE-Derived PASP

Agreement improved when comparing invasive pPAP to PASP (Table 2; Fig. 2). TTE-derived PASP 48.4 ± 15.7 mmHg, compared with a mean invasive pPAP of 51.0 ± 11.4 mmHg, yielding a mean bias = + 2.6 mmHg, with 95% LoA of = -24 to + 29 mmHg. Correlation and reliability were also stronger: ICC = 0.49 (poor-moderate reliability) and r = 0.52 (p = 0.0002). Weighted κ value indicated fair categorical agreement (κ = 0.31 overall; 0.38 at ≤ 12 h; 0.36 for ≤ 24 h).

In comparison to invasive mPAP, agreement improved when assessing pPAP pressure ranges and TTE-derived PASP (Table 4). At pPAP < 30 mmHg (n = 1), TTE-derived PASP matched invasive category in 1 of 5 PASP readings. In the 30–44 mmHg range (n = 12), 6 of 13 PASP values matched. In the 45–59 mmHg pPAP group (n = 19), 10 of 20 PASP readings matched invasive values. When pPAP was ≥ 60 mmHg (n = 13), 5 of 7 PASP values remained in same grouping as invasive pPAP. Categorical agreement between invasive pPAP and TTE-derived PASP was substantially better than that of mPAP with a simple κ = 0.2570 (95% CI 0.0463 to 0.4677) and weighted κ 0.3150 (95% CI 0.1031 to 0.5268).

Table 4
Concordance Table Between CDT-Measured pPAP and TTE-Derived PASP

TTE-Derived PASP	CDT-Measured pPAP				
Frequency **Percent** **Row Pct** **Col Pct**	**1:<30**	**2:30–44**	**3:45–59**	**4:60+**	**Total**
1:<30	1 2.22 20.00 100.00	1 2.22 20.00 8.33	3 6.67 60.00 15.79	0 0.00 0.00 0.00	5 11.11
2:30–44	0 0.00 0.00 0.00	6 13.33 46.15 50.00	5 11.11 38.46 26.32	2 4.44 15.38 15.38	13 28.89
3:45–59	0 0.00 0.00 0.00	4 8.89 20.00 33.33	10 22.22 50.00 52.63	6 13.33 30.00 46.15	20 44.44
4:60+	0 0.00 0.00 0.00	1 2.22 14.29 8.33	1 2.22 14.29 5.26	5 11.11 71.43 38.46	7 15.56
Total	1 2.22	12 26.67	19 42.22	13 28.89	45 100.00

No significant association was identified between demographic characteristics and PE severity indices and the degree of agreement.

DISCUSSION

This study provides the first real-world comparative analysis of TTE-derived PASP, and invasive PAPs obtained during CDT in patients with acute PE. By evaluating agreement across mPAP and pPAP, as well

as time and pressure ranges, this study clarifies the performance characteristics of PASP in acute PE.

Relevance to PE Risk Stratification

Despite their widespread use, current PE risk-stratification tools such as the PESI, sPESI, and Bova score [2–5] provide limited ability to identify which intermediate-risk patients are at risk for early hemodynamic decompensation. The ESC framework offers a useful structural approach to categorize PE severity [1], but it similarly lacks the granularity needed to guide decisions about advanced therapies in intermediate- to high-risk PE. This gap is at least partially addressed by the 2026 AHA/ACC PE guidelines, which introduces acute PE clinical categories A-E to improve severity classification, risk stratification, prognostic assessment, and therapeutic decision-making [23]. This shift in classification captures the growing momentum in the PERT community to refine prognostic assessment by incorporating markers of RV strain and RV-PA coupling. TTE-derived metrics such as PASP, TAPSE, and TAPSE/PASP are increasingly studied for their potential to detect early RV maladaptation and stratify patients who may deteriorate [6–10]. Our cohort consisted primarily of intermediate- and high-risk PE patients, the population in which clinicians are most keen to determine values and scores to determine prognosis and risk of decompensation.

Interpreting CDT-derived PAPs and TTE-derived PASP

Our findings are supported by established principles of echocardiographic Doppler, which helps clarify why TTE-derived PASP demonstrated poor agreement with invasive mPAP yet better concordance with pPAP in acute PE. Across the cohort, TTE-derived PASP was consistently higher than invasive mPAP, demonstrated weak linear correlation, and showed negligible reliability on ICC and κ statistics. These findings support the interpretation that PASP does not accurately reflect integrated mean pulmonary vascular load in acute PE, where hemodynamics fluctuate rapidly and are influenced by clot location and overall burden, and the effects of systemic therapies. In contrast, agreement between pPAP and PASP was substantially better. PASP demonstrated stronger correlation, narrower Bland–Altman LoA, higher ICC, and fair categorical concordance. These findings align physiologically with the Doppler method of estimating PASP, which is based on peak tricuspid regurgitant velocity and therefore corresponds more closely to a peak systolic pressure measurement than to mean pulmonary pressure [24], which is estimated from the peak pulmonary regurgitant velocity, as the Doppler signal reflects the pressure gradient across the pulmonic valve [24]. Application of the Bernoulli equation provides an approximation of catheter-derived mPAP on RHC, which is not routinely assessed or reported in clinical TTE documentation [24].

Effect of Pressure and Time

Our pressure- and time-stratified analyses provide insight into the agreement among measurements, but also the variable performance of TTE-derived measurements in acute PE. When stratified by invasive mPAP categories, we observed that TTE-derived PASP were consistently higher than invasive mPAP at lower pulmonary pressures. In the lowest range (< 30 mmHg) only a minority (18.2%) of PASP values fell

within the same category, while the majority were categorized into higher categories. This is consistent with the mean bias seen in Bland Altman analysis. As invasive mPAP increased, categorical agreement can be observed to modestly improve. However, at the highest mPAPs of ≥ 60 mmHg no matching was observed on PASP, potentially reflecting a technical limitation of Doppler-derived estimates at extremes of mPAP. Agreement was more favorable when comparing PASP with invasive pPAP. PASP more closely aligned with pPAP as pressure increased. Agreement appeared strongest in the 45–59 mmHg and ≥ 60 mmHg groups, where PASP matched invasive categories in almost half of the cases. These findings support the physiologic basis that PASP, derived from peak tricuspid regurgitation velocity, reflects instantaneous systolic load or pressure [24]; thus, correlating better with pPAP and not mPAP, particularly in a labile hemodynamic milieu like that of acute PE.

Time-stratified analysis reinforced differences between TTE-derived PASP values and agreement with invasive mPAP and pPAP. Agreement between PASP and mPAP improved modestly when performed within 12–24 hours of CDT ($\kappa = 0.17 \leq 12$ h and $\kappa = 0.13 \leq 24$ h) compared to ≤ 48 h ($\kappa = 0.03$). Agreement between PASP and pPAP was highest when TTE and CDT were performed closer together, with κ values improving from 0.31 overall to 0.38 at ≤ 12 hours and 0.36 at ≤ 24 hours.

In conclusion, TTE-derived PASP agrees more closely with pPAP on CDT than with mPAP. Secondary analysis indicates a pressure-dependent relationship between CDT-measured pPAP and TTE-derived PASP: at lower pPAP levels, PASP tends to provide higher estimated values, whereas at higher pPAP levels, PASP tends to yield lower estimates. Our findings further suggest a time-dependence, with mildly improved agreement when imaging is temporally close to CDT. These pressure and time behaviors underscore the limitations of relying on a single set of TTE-derived variables in acute PE.

Clinical Implications

PASP alone is insufficient to quantify RV afterload or to guide escalation decisions in intermediate-risk PE. Rather, PASP and other TTE-derived values (e.g., TAPSE, TAPSE/PASP) should be interpreted within a multimodal framework that includes clinically relevant symptoms (e.g. syncope), vital signs, biomarkers and CTA imaging of RV size/strain to further stratify intermediate-risk PE patients.

Strengths and Limitations

Strengths of this study include the use of paired invasive and noninvasive measurements obtained during CDT in a real-world PERT registry, and the application of multiple agreement metrics. Limitations include our single-center retrospective design and modest sample size. Inherently, there is operator-dependent variability in both TTE and CDT measurements and potential confounding effects of sedation and anticoagulation on pulmonary vasculature hemodynamics. While the temporal distance between TTE and CDT is a limitation to our study when analyzing the agreement between TTE-derived PASP and CDT-measured PAPs, it is also the product of a real-world busy tertiary care center. Further prospective studies may be designed to assess the temporal relationship between TTE and CDT pressure measurements. While invasive measurements are often regarded as the reference standard, supported by RHC in chronic diseases [11–19], our findings do not establish a definitive gold standard in the setting

of acute PE. Further cohort studies would be needed to correlate CDT PAPs and TTE-derived PASP with post-PE outcomes – such as RV recovery, post-PE dyspnea, CTED, CTEPH – to clarify which measure has the most prognostic value and relevance.

Conclusion

TTE-derived PASP systematically overestimates invasive mPAP but aligns more closely with invasive pPAP in acute PE. These findings support the concept that PASP reflects instantaneous systolic load rather than mean hemodynamic burden. Further research correlating CDT and TTE pressures with clinical outcomes is warranted.

Declarations

Author Contribution

C.M. contributed to study conceptualization, interpretation of findings, and drafting of the main manuscript text. D.W. performed the statistical analysis and prepared Tables 3 and 4, and Figures 1 and 2. R.N., V.C., and B.E. contributed to study conceptualization, interpretation of findings, project supervision, and critical manuscript revision. All authors reviewed and approved the final manuscript.

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Figures

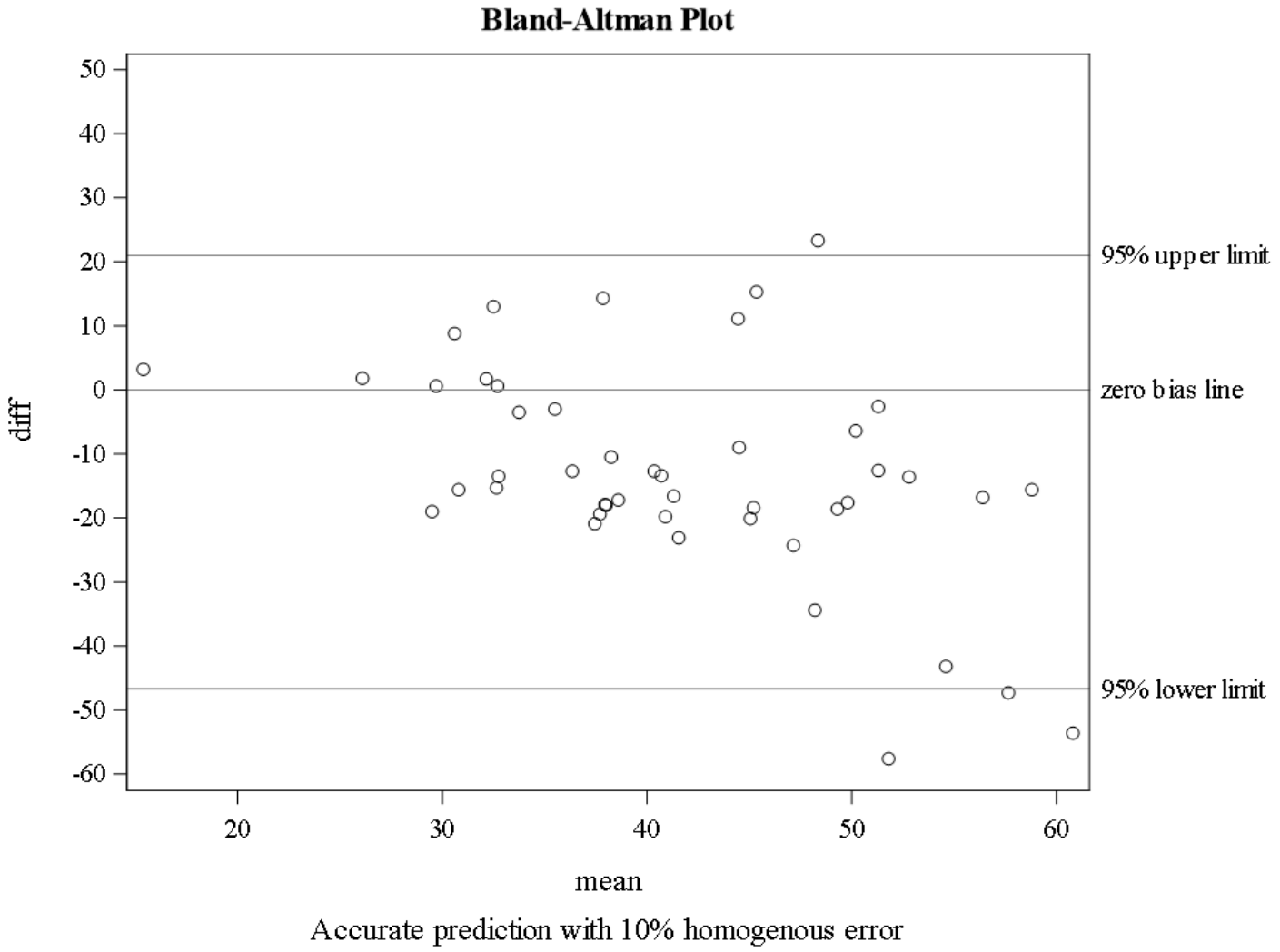

Figure 1

Bland-Altman Plot of CDT mPAP and TTE-Derived PASP

Description: Each point represents an individual patient (n = 46; one case excluded due to missing invasive pressure difference). The x-axis shows the mean of invasive mPAP and TTE-derived PASP, while the y-axis depicts their difference (mPAP – TTE). TTE-derived PASP was consistently higher than invasive mPAP with a mean bias of -12.8 mmHg ($p < 0.0001$). The 95% limits of agreement ranged from -46 to +20 mmHg, indicating wide dispersion and increasing variability at higher mean pressures. Most data points fall below zero, consistent with overestimation by echocardiography if invasive mPAP is inferred as reference.

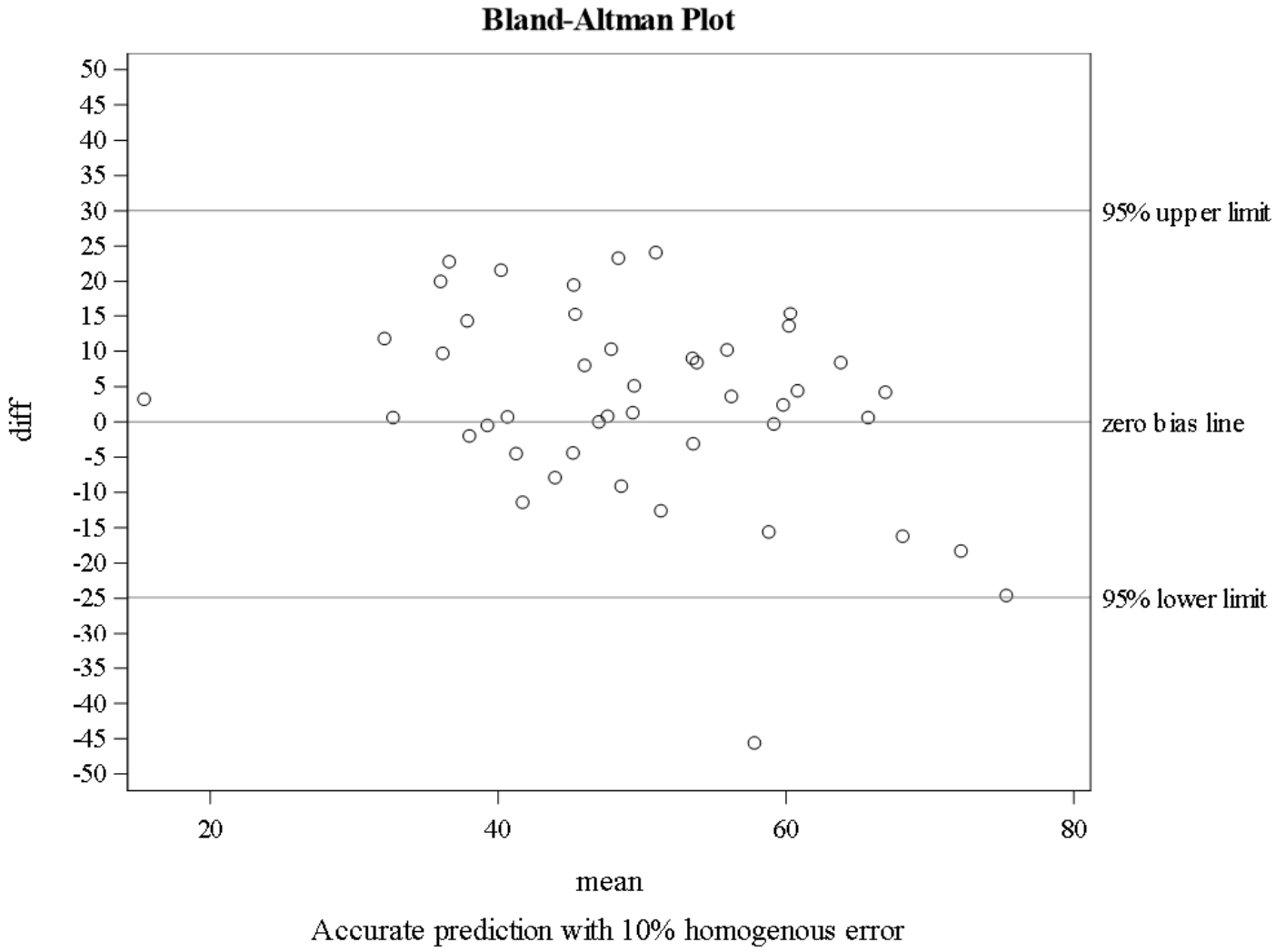

Figure 2

Bland-Altman Plot of CDT pPAP and TTE-Derived PASP

Description: Each point represents an individual patient (n = 45). The x-axis denotes the mean of invasive pPAP and TTE-derived PASP, while the y-axis represents their difference (pPAP – TTE). The plot demonstrates a small positive mean bias of +2.6 mmHg, with 95% limits of agreement -24 to 29 mmHg. Error distribution is bidirectional, with both over- and under-estimation across the range of pressures, suggesting improved alignment between noninvasive and invasive measures compared with the mPAP analysis.