THE ANATOLIAN JOURNAL OF CARDIOLOGY

Official journal of the TURKISH SOCIETY OF CARDIOLOGY

Which Diastolic Pressure Should Be Used to Assess Diastolic Function?

ABSTRACT

Background: Although high left ventricular filling pressures [left ventricular (LV) end-diastolic pressure or pulmonary capillary wedge pressure (PCWP)] are widely taken as surrogates for LV diastolic dysfunction, the actual distending pressure that governs LV diastolic stretch is transmural pressure difference (ΔP_{TM}). Clinically, preferring ΔP_{TM} over PCWP may improve diagnostic and therapeutic decision-making. We aimed to compare the clinical implications of diastolic function characterization based on PCWP or ΔP_{TM}.

Methods: We retrospectively screened our hospital database for adult patients with a clinical diagnosis of heart failure who underwent right heart catheterization. Echocardiographic diastolic dysfunction was graded according to the current guidelines. LV end-diastolic properties were assessed with construction of complete end-diastolic pressure–volume relationship (EDPVR) curves using the single-beat method. Survival status was checked via the electronic national health-care system.

Results: A total of 693 cases were identified in our database; the final study population comprised 621 cases. ΔP_{TM}-based, but not PCWP-based, EDPVR diastolic stiffness constants were significantly predictive of advanced diastolic dysfunction. PCWP-based diastolic stiffness constants were not able to predict 5-year mortality, whereas ΔP_{TM}-based EDPVR stiffness constants and volumes all turned out to have significant predictive power for 5-year mortality.

Conclusion: Left ventricular diastolic function assessment can be improved using ΔP_{TM} instead of PCWP. As ΔP_{TM} ultimately linked to right-sided functions, this approach emphasizes the limitations of taking LV diastolic function as an isolated phenomenon and underlines the need for a complete hemodynamic assessment involving the right heart in therapeutic and prognostic decision-making processes.

Keywords: Diastolic function, heart failure, hemodynamics, mortality, pressure–volume loop

ORIGINAL INVESTIGATION

Emre Aslanger[1]
Özlem Yıldırımtürk[2]
Dursun Akaslan[3]
Melih Öz[2]
Barış Güngör[2]
Halil Ataş[3]
Bülent Mutlu[3]

[1]*Department of Cardiology, Health Sciences University, Başakşehir Pine and Sakura City Hospital, İstanbul, Türkiye*
[2]*Department of Cardiology, Health Sciences University, Dr. Siyami Ersek Thoracic and Cardiovascular Surgery Training and Research Hospital, İstanbul, Türkiye*
[3]*Department of Cardiology, Marmara University, Pendik Training and Research Hospital, İstanbul, Türkiye*

Corresponding author:
Emre Aslanger
✉ mr_aslanger@hotmail.com

Received: August 02, 2023
Accepted: December 18, 2023
Available Online Date: January 24, 2024

Cite this article as: Aslanger E, Yıldırımtürk Ö, Akaslan D, et al. Which diastolic pressure should be used to assess diastolic function? *Anatol J Cardiol.* 2024;28(3):158-164.

DOI:10.14744/AnatolJCardiol.2024.3713

INTRODUCTION

End-diastolic wall stretch is an important hemodynamic variable that governs left ventricular (LV) systolic and diastolic functions and their integration.[1] According to the FrankStarling law, LV stroke volume (SV) increases with increasing end-diastolic wall stretch.[2,3] On the other hand, LV cannot be infinitely stretched, as LV end-diastolic pressure (LVEDP) exponentially increases while inherent elastic reserve of the LV is used up with further increases in LV volume (LVEDV). This end-diastolic pressure–volume relationship (EDPVR) characterizes LV diastolic function, and a normal diastolic function requires a LVEDP that (1) generates enough stretch for the delivery of adequate SV and (2) does not exceed a certain threshold that causes the transmission of increased pressure backward, resulting in pulmonary congestion. The current guidelines define this threshold as 15 mm Hg,[4,5] and many noninvasive surrogates of diastolic function were tested against this "gold standard."[6,7]

It is generally overlooked, however, that LVEDP or its more frequently used surrogate; pulmonary capillary wedge pressure (PCWP), is not the sole force acting on LV. There is also an outside pressure that prevents the distention of the LV and decreases its end-diastolic stretch. This external force is pericardial

pressure for the LV free wall and right ventricular (RV) end-diastolic pressure for the interventricular septum. Since RV end-diastolic pressure is the same as right atrial (RA) pressure, and pericardial pressure is very close to[8-10] and follows the changes in[10-12] RA pressure, RA pressure can be used as the pressure constraining the whole LV. Therefore, the real distending pressure that reflects LV stretch and governs EDPVR is not PCWP, but LV transmural pressure difference ($\Delta P_{TM} = PCWP - P_{RA}$) (Figure 1). This distinction is important because, according to this rationale, any assessment of LV diastolic function should also take right heart into account. Therefore, whether a ΔP_{TM}-based assessment would reflect diastolic function better than the PCWP-based one needs to be elucidated.

In this study, we aimed to compare these two approaches using a complete EDPVR analysis.

METHODS

Study Protocol

The study was conducted at Dr. Siyami Ersek Thoracic and Cardiovascular Surgery Training and Research Hospital, a tertiary center for heart failure (HF) and heart transplantation. A Local Ethical Committee approval was obtained, and the study was undertaken in accordance with the Declaration of Helsinki. We retrospectively screened our hospital database for adult patients with a clinical diagnosis of HF who underwent right heart catheterization (RHC) between 2015 and 2022. Exclusion criteria included incomplete RHC data, a PCWP less than 15 mm Hg, congenital heart disease with uncorrected shunts, and chronic kidney disease requiring dialysis.

The demographics and laboratory results were obtained via chart review and included complete blood count, kidney function tests, serum N-terminal pro-brain natriuretic peptide (NT pro-BNP) levels, echocardiographic, and RHC measurements. Echocardiographic data were obtained using ultrasound machines of the EPIQ series (Philips Medical Systems, Bothell, Wash, USA). Left atrial and LV volumes, and LV ejection fraction (LVEF) were calculated using the biplane Simpson's method. Mitral inflow pulsed-wave Doppler and lateral mitral annular tissue Doppler measurements were taken from the apical four-chamber view. Echocardiographic diastolic dysfunction was graded according to the guidelines,[6] using E to A wave ratio, e' velocity, Mitral E wave to e' velocity, LA volume index, and maximum tricuspid regurgitation velocity. Only the patients with a complete set of these variables were included in the comparison with EDPVR parameters.

Right heart catheterization was performed via the right jugular or femoral vein using a 7F balloon-tipped Swan-Ganz catheter (Edwards Lifesciences, Irvine, Calif, USA). Cardiac output was measured using the indirect Fick method. Pressure system calibration was checked with a square-wave test before the recordings were acquired. All pressure tracings were evaluated by visual exploration for physiological accuracy, and end-expiratory pressure values were taken. The LV transmural pressure difference (ΔP_{TM}) was calculated as PCWP minus RA pressure.

LV end-diastolic properties were assessed with construction of complete EDPVR curves using the single-beat method.[13] Briefly, the measured LVEDV was normalized by appropriate scaling, and a normalized EDPVR was constructed using the measured LVEDP pressure. LV volumes at zero pressure (V_0) and 30 mm Hg (V_{30}) were estimated based on the assumption of a relatively consistent relationship between the volume at a certain pressure and V_0. Then, the entire EDPVR was characterized as $LVEDP = \alpha(LVEDV)\beta$, where diastolic stiffness constants of α and β were calculated to force the curve through the measured LVEDP and LVEDV values, and the calculated V_0 and V_{30}.

All patients were managed according to the ESC guidelines for the diagnosis and management of heart failure (HF).[4,14] Survival status was checked via electronic national healthcare system.

Statistical Analysis

The SPSS statistics software (version 29.0; SPSS Inc., Chicago, Ill, USA) was used for all statistical analyses. Continuous variables were expressed as mean ± standard deviation or median (interquartile range [IQR]), while categorical variables were expressed in counts (percentages). The normality of continuous variables was assessed using Shapiro–Wilk's test and visual inspection of normal Q–Q plots. The diagnostic accuracy of PCWP and ΔP_{TM}, and PCWP- and ΔP_{TM}-based V_0, V_{30}, and diastolic stiffness constants (α and β) for echocardiographic diastolic dysfunction grade II or III and 5-year mortality was analyzed using receiver operating characteristics curve analysis. Area under curve (AUC) values for PCWP- and ΔP_{TM}-based values were compared pairwise using the method of DeLong et al.[15] For all statistical analyses, a P-value <0.05 was considered significant.

RESULTS

A total of 693 cases were identified in our database; 72 patients were excluded due to incomplete data (n = 21), a PCWP less than 15 mm Hg (n = 47), congenital heart disease with uncorrected shunts (n = 2), and a history of chronic

HIGHLIGHTS

- Left ventricular (LV) diastolic function is usually assessed by LV filling pressures.
- High LV filling pressures (LV end-diastolic pressure or pulmonary capillary wedge pressure) are used as a surrogate for diastolic dysfunction.
- However, LV diastolic function is also strongly influenced by right-heart pressures.
- Left ventricular diastolic function should be assessed using LV transmural pressure instead of isolated LV filling pressures.
- This perspective underlines the need for a complete assessment involving right heart in the therapeutic and prognostic decision-making processes about LV diastolic disorders.

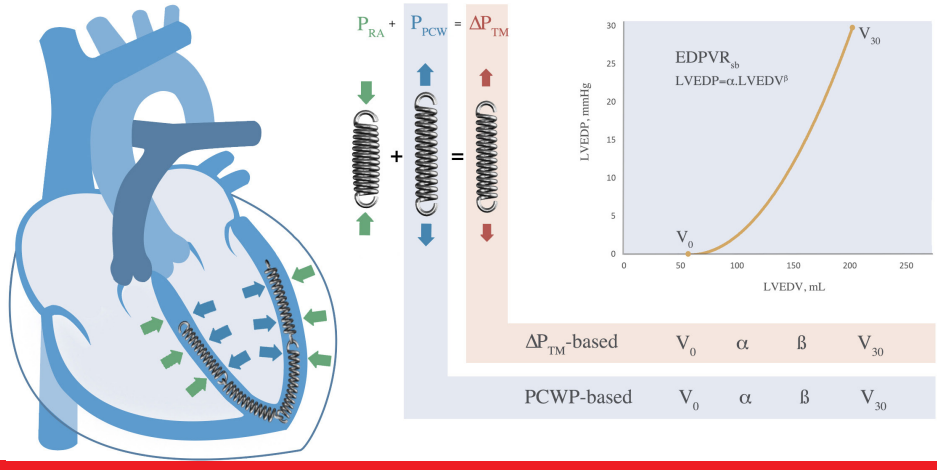

Figure 1. Left ventricular (LV) end-diastolic pressure–volume relationship (EDPVR) characterizes LV diastolic properties. The curve can be defined as LVEDP = α(LVEDV)$^{\beta}$, where LVEDP is left ventricular end-diastolic pressure, LVEDV is left ventricular end-diastolic volume, α and β are constants that define the steepness of EDPVR curve. V_0 and V_{30} are LV volumes at 0 and 30 mm Hg, respectively. Mechanistically, V_0 represents remodeling; higher values indicate a more dilated LV. V_{30} represents a measure of ventricular stiffness; higher values indicate a more compliant LV. ΔP_{TM}-based, but not PCWP-based, EDPVR diastolic stiffness constants were significantly predictive of grade II or III diastolic dysfunction. PCWP-based diastolic stiffness constants were not able to predict 5-year mortality, whereas ΔP_{TM}-based EDPVR stiffness constants and volumes all turned out to have significant predictive power for 5-year mortality. ΔP_{TM}, transmural pressure difference; $EDPVR_{sb}$, single-beat end-diastolic pressure–volume relationship; LVEDP, left ventricular end-diastolic pressure; LVEDV, left ventricular end-diastolic volume; PCWP, pulmonary capillary wedge pressure; P_{PCW}, pulmonary capillary wedge pressure; P_{RA}, right atrial pressure; SV, stroke volume.

kidney disease requiring dialysis (n = 2). Therefore, the final study population comprised 621 cases. Baseline characteristics are summarized in Table 1. Echocardiographic and invasive hemodynamic parameters are presented in Table 2.

Diastolic grading with a complete set of echocardiographic variables was possible in 39.4% (245/621) of the study cohort. Grade I, II, and III diastolic dysfunction was diagnosed in 93 (37.9%), 61 (24.8%), and 91 (37.1%) patients, respectively. Both PCWP (AUC, 0.702; 95% confidence interval (CI), 0.636-0.768; $P < .001$) and ΔP_{TM} (AUC, 0.674; 95% CI, 0.606-0.741; $P < .001$) were able to predict grade II or III diastolic dysfunction. However, only ΔP_{TM}-based diastolic stiffness constants α (AUC, 0.386; 95% CI, 0.313-0.459; $P = .005$) and β (AUC, 0.636; 95% CI, 0.563-0.710; $P = .001$) were significantly predictive of grade II or III diastolic dysfunction, whereas the predictive power of PCWP-based α (AUC, 0.545; 95% CI, 0.468- 0.622; $P = .269$) and β (AUC, 0.475; 95% CI, 0.398-0.552; $P = .548$) was not significant. The diagnostic accuracy of ΔP_{TM}-based α and β was significantly superior to PCWP-based ones (P for $\alpha = 0.002$; P for $\beta = .008$). This superiority was mostly preserved when analyses were limited to reduced (<40%) (for α AUC difference; 0.255, 95% CI 0.094-0.416, $P = .002$ and for β, AUC difference, 0.202; 95% CI 0.002-0.403, $P = .048$) and preserved (>50%) LVEF subgroups (for α AUC difference, 0.139; 95% CI 0.013-0.265, $P = .031$). The difference in ΔP_{TM}- and PCWP-based β was not significant in patients with preserved LVEF (AUC difference, 0.009; 95% CI −0.166 to 0.184, $P = .916$). V_0 (for V_0-PCWP; AUC, 0.538; 95% CI, 0.461-0.615; $P = .351$ and for V_0-ΔP_{TM}; AUC, 0.556, 95% CI, 0.479- 0.632; $P = 0.172$) and V_{30} (for V_{30}-PCWP; AUC, 0.555; 95% CI, 0.478- 0.632; $P = .179$ and for V_{30}-ΔP_{TM}; AUC, 0.547, 95% CI, 0.471-0.624; $P = .246$) values were unable to predict grade II and III diastolic dysfunction with either approach.

Median follow-up was 511 (832) days and 5-year mortality rate was 27.1% (173/621). Both PCWP (AUC, 613; 95% CI, 0.565-0.662; $P < .001$) and ΔP_{TM} (AUC, 557; 95% CI, 0.507-0.606; $P = 0.029$) are turned out to have a significant diagnostic power for 5-year mortality, but PCWP had a better accuracy compared to ΔP_{TM} (AUC difference, 0.057; 95% CI, 0.015-0.099; $P = .008$).

PCWP-based diastolic stiffness constants, α (AUC, 511; 95% CI, 0.455- 0.567; $P = .678$) and β (AUC, 473; 95% CI, 0.418-0.528; $P = .313$), were not able to predict 5-year mortality, whereas PCWP-based V_0 (AUC, 559; 95 CI%, 0.507-0.611; $P = .026$) and V_{30} (AUC, 567; 95 CI%, 0.515-0.618; $P = .013$) had a minor but significant predictive power. On the other hand, ΔP_{TM}-based diastolic stiffness constants; α (AUC, 421; 95% CI, 0.370- 0.472; $P = .003$) and β (AUC, 554; 95% CI, 0.503-0.606; $P = .042$), and volumes; V_0 (AUC, 575; 95% CI, 0.524-0.627; $P = .005$) and V_{30} (AUC, 569; 95% CI, 0.517-0.622; $P = .009$), all turned out to have significant predictive power for 5-year mortality.

When the predictive power of PCWP-based and ΔP_{TM}-based parameters were compared in terms of AUC values, ΔP_{TM}-based diastolic stiffness constant α had a significantly higher predictive power compared to PCWP-based α (AUC difference, 0.090; 95% CI, 0.017-0.163; $P = .016$). Similarly, ΔP_{TM}-based V_0 had a significantly higher predictive power compared to PCWP-based V_0 (AUC difference, −0.016; 95% CI, −0.026 to −0.007; $P = .001$). Despite showing a trend in this direction, the difference between ΔP_{TM}-based and

Table 1. Baseline Characteristics*

	n = 621
Demographics	
Age, years	52 ± 14
Female, %	186 (30)
BMI, kg m^{-2}	28.5 ± 5.7
Heart rate, beats min^{-1}	84 ± 17
SBP, mm Hg	125 ± 27
Comorbidities	
AF, n (%)	275 (44.1)
Hypertension, n (%)	249 (40.1)
Diabetes, n (%)	174 (28)
Dyslipidemia, n (%)	125 (20.2)
CAD, n (%)	245 (39.6)
CKD, n (%)	63 (10.1)
Laboratory parameters	
GFR, mL min^{-1} 1.73 m^{-2}	85 ± 33
Hemoglobin, g dL^{-1}	13.0 ± 2.1
AST, mg dL^{-1}	24.9 ± 10.0
ALT, mg dL^{-1}	28.0 ± 20.3
CRP, mg dL^{-1}	0.8 (2.6)
hs-cTnT, ng L^{-1}	8 (17.8)
NT-proBNP, ng L^{-1}	1485 (3072)
Functional class	
NYHA class I, n (%)	65 (10.4)
NYHA class II, n (%)	257 (41.3)
NYHA class III, n (%)	235 (37.8)
NYHA class IV, n (%)	58 (9.3)
Treatment	
Aspirin, n (%)	280 (44.1)
Anticoagulants, n (%)	274 (44.1)
Diuretics, n (%)	552 (88.9)
Beta blockers, n (%)	518 (83.4)
ACEI/ARBs or ARNI, n (%)	480 (77.3)
Spironolactone, n (%)	491 (79.1)
Statins, n (%)	143 (17.6)

*Values are mean ± standard deviation, median (IQR), or number (percentage).
ACEI/ARB, angiotensinogen converting enzyme inhibitors or angiotensin II receptor blockers; AF, atrial fibrillation; ALT, alanine aminotransferase; ARNI, angiotensin receptor-neprilysin inhibitor; AST, aspartate aminotransferase; BMI, body mass index; CAD, coronary artery disease; CKD, chronic kidney disease; CRP, C-reactive protein; GFR, glomerular filtration rate; hs-cTnT, high-sensitivity cardiac troponin T; IQR, interquartile range; NT-proBNP, N-terminal pro-brain natriuretic peptide; NYHA, New York Heart Association; SBP, systolic blood pressure.

PCWP-based diastolic stiffness constant β did not reach statistical significance (AUC difference, −0.081; 95% CI, −0.167 to 0.004; $P = .003$). The difference between ΔP_{TM}- and PCWP-based V_{30} was not significant (AUC difference, 0.003; 95% CI, −0.008 to 0.013; $P = .607$).

Complete EDPVR curves with both approaches are given in Figure 2.

Table 2. Echocardiographic and Hemodynamic Parameters*

	n = 621
Echocardiographic parameters	
LVEF, %	45 (40)
LVEF<40, n (%)	424 (68.3)
LVEF 40-50, n (%)	15 (2.4)
LVEF >50, n (%)	182 (29.3)
LVEDV, mL	129 (95)
LVESV, mL	66 (101)
LA area, cm^2	23 (10)
RA area, cm^2	21 (8)
LAVI, mL m^{-2}	37 (14)
Mitral E wave velocity, m s^{-1}	8.0 (0.4)
Mitral annulus e′ velocity, cm s^{-1}	8.0 (0.5)
Mitral E/A ratio	2.0 (2.0)
Mitral E/e′ ratio	12.8 (7.5)
TAPSE, mm	17 (7)
Peak TR V_{max}, m s^{-1}	3.4 (0.9)
Pericardial effusion, n (%)	89 (14.3)
PA systolic pressure, mm Hg	56 (25)
PA diastolic pressure, mm Hg	28 (15)
PA mean pressure, mm Hg	38 (17)
Ao systolic pressure, mm Hg	120 (37)
Ao diastolic pressure, mm Hg	72 (18)
Ao mean pressure, mm Hg	89 (21)
RA mean pressure, mm Hg	13 (9)
PCWP, mm Hg	25 (11)
PVR, Woods	3 (3.1)
SVR, Woods	21 (9.7)
SaO_2, %	97 (3)
MvO_2, %	59 (16)
CO, L min^{-1}	3.7 (1.6)
CI, L min^{-1} m^{-2}	1.9 (0.8)
SV, mL	44 (22)
SI, mL min^{-1}	23 (12)

*Values are median (interquartile range) or number (percentage).
Ao, aortic; CI, cardiac index; CO, cardiac output; LAVI, left atrial volume index; LVEDV, left ventricular end-diastolic volume; LVEF, left ventricular ejection fraction; LVESV, left ventricular end-systolic volume; MvO_2, mixed venous oxygen saturation; PA, pulmonary artery; PCWP, pulmonary capillary wedge pressure; PVR, pulmonary vascular resistance; RA, right atrium; RV, right ventricular; SaO_2, systemic oxygen saturation; SI, stroke index; SV, stroke volume; SVR, systemic vascular resistance; TAPSE, tricuspid annular plane systolic excursion; TR, tricuspid regurgitation; V_{max}, maximum velocity.

DISCUSSION

Several previous studies have questioned the interchangeable use of PCWP with end-diastolic myocardial stretch when interpreting LV diastolic function, and suggested the use of ΔP_{TM} instead of PCWP.[16-21] Despite these findings, PCWP or LVEDP is still being widely used as the sole surrogate for LV diastolic function. One reason for this might be that all previous studies were mechanistic, and no study has compared a PCWP-based approach to diastolic function

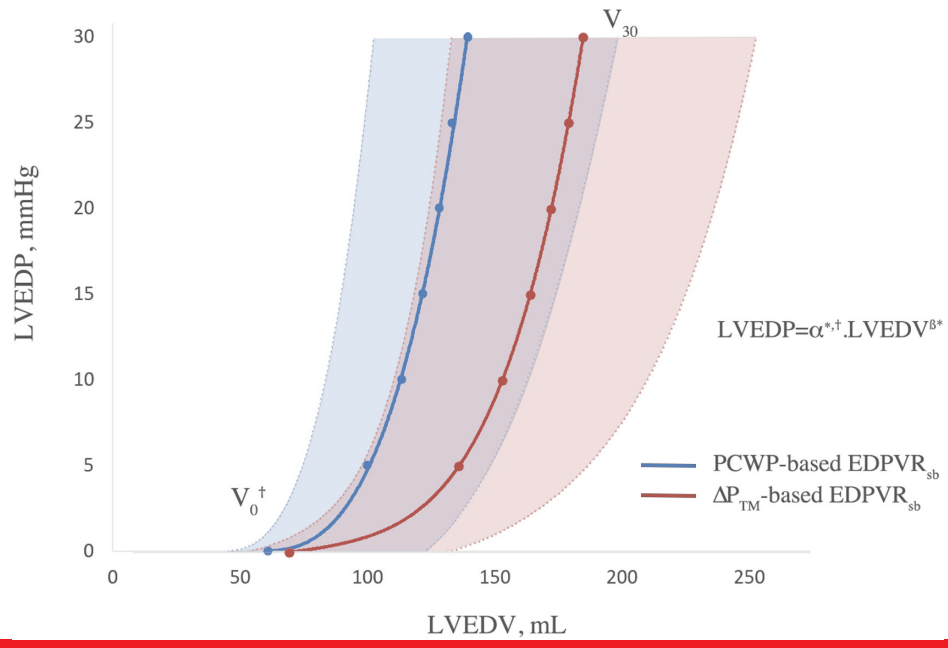

Figure 2. Median left ventricular (LV) end-diastolic pressure–volume relationship (EDPVR) curves with shaded areas representing interquartile ranges. The pulmonary capillary wedge pressure (PCWP)-based approach systematically estimates a stiffer LV compared to an LV transmural pressure difference (ΔP_{TM}) one. ΔP_{TM}, transmural pressure difference; $EDPVR_{sb}$, single-beat end-diastolic pressure-volume relationship; LVEDP, left ventricular end-diastolic pressure; LVEDV, left ventricular end-diastolic volume; PCWP, pulmonary capillary wedge pressure. *ΔP_{TM} significantly superior compared to PCWP in predicting echocardiographic grade II or III diastolic dysfunction. †ΔP_{TM} significantly superior compared to PCWP in predicting 5-year mortality.

with a ΔP_{TM}-based one in terms of hard clinical endpoints. Our study, to our knowledge, is the first to directly compare these two approaches in an HF population with a long-term mortality endpoint. Our results support the superiority of the ΔP_{TM}-based diastolic function characterization in predicting significant echocardiographic diastolic dysfunction in both reduced and preserved LVEF subgroups. More importantly, ΔP_{TM}-based diastolic function characterization was superior to the PCWP-based one in the prediction of 5-year mortality.

Our study has several important implications. First, our results indicate that any noninvasive parameter evaluating diastolic function should be tested against ΔP_{TM}, not PCWP. Secondly, the inclusion of RA pressure as an important factor in LV diastolic function adds the maintenance of optimal volume status and right-heart hemodynamics to the list of therapeutic targets in the management of "diastolic" HF. Even lowering ΔP_{TM} without reducing PCWP actually results in better diastolic function and can be coupled with increased contractile performance via an improved systolodiastolic integration, as shown in a sub-study of the Evaluation Study of Congestive Heart Failure and Pulmonary Artery Catheterization Effectiveness (ESCAPE) trial.[22] Thirdly, as diastolic stiffness is systematically overestimated when PCWP, instead of ΔP_{TM}, is used, increased right-sided pressures can cause LV diastolic dysfunction to appear exaggerated. Indeed, it has been estimated that the contribution of external constraint to the PCWP can be as high as 50%-80% in HF patients.[19] Therefore, diastolic function should be reassessed after adequate decongestion, otherwise, it may unnecessarily trigger a work-up for restrictive etiologies. Fourthly, at the extreme end of the spectrum, very high right-sided pressures can cause apparent diastolic LV dysfunction without any inherent pathology in LV relaxation. Although volume overload due to neurohormonal activation in HF usually causes both PCWP and RA pressure elevation, these can be discordant in one-fourth to one-third of the patients with HF.[23,24] This may be a problem especially in patients with pulmonary hypertension, and apparent LV failure due to right-sided HF may be possible in these patients, although the opposite, RV failure due to left heart disease, is the traditional concept. The presence of such a pathophysiological mechanism needs to be confirmed in further studies.

Lastly, it should be underlined that mechanistic and prognostic meaning of a parameter can be different. For example, the prognostic information contained in PCWP is not limited to diastolic function but also includes total volume status, which reflects neurohormonal activity and disease progression. On the other hand, ΔP_{TM} is at least partly independent of volume status, as both of its determinants, namely PCWP and RA pressure, are influenced by the same vascular volumes and therefore cancel each other.

Thus, ΔP_{TM} can characterize intrinsic diastolic function independent of volume status but does this at the expense of losing some prognostic information. This may explain why ΔP_{TM}-based diastolic stiffness coefficients were superior to PCWP-based ones, while ΔP_{TM} itself seems to be less

powerful compared to the PCWP measurement in mortality prediction.

Study Limitations

Our study has several strengths and limitations. To our knowledge, this is the largest mechanistic study in HF evaluating the pathophysiologic background of diastolic hemodynamics with a clinical hard endpoint. Although AUC values for both PCWP-based and ΔP_{TM}-based diastolic variables might seem low at first glance, it should be underlined that the main aim of this study was to prove the presence of a significant difference between these assessment approaches, not to explore their diagnostic powers. Moreover, as these two approaches were compared in the same population, the study design was not influenced by baseline differences and confounding factors. Lastly, we used the whole EDPVR, which characterizes diastolic properties of LV better than a single snapshot pressure measurement such as LVEDP or PCWP. On the other hand, single-beat estimation of EDPVR depends on complex mathematical calculations and several assumptions, which is a limitation. Furthermore, LV volumes and pressure measurements were not done simultaneously. The gold standard for acquiring EDPVR with simultaneous pressure and volume measurements is recording pressure–volume loops using a conductance catheter with changing venous return, but this would virtually be impossible on such a scale because of its prohibitive cost and cumbersome methodology.

CONCLUSION

Our results indicate that LV diastolic function is better characterized by ΔP_{TM} instead of PCWP. This approach underlines the importance of a complete hemodynamic assessment involving right heart in the therapeutic and prognostic decision-making processes of LV diastolic function evaluation.

Ethics Committee Approval: This study was approved by the Ethics committee of Dr. Siyami Ersek Thoracic and Cardiovascular Surgery Training and Research Hospital (Approval number: E-28001928-604.01.01, Date: 23.12.2022).

Informed Consent: No consent form was required.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – E.A., Ö.Y.; Design – E.A.; Supervision – B.M.; Resources – Ö.Y., M.Ö., B.G., H.A., E.A, D.A.; Materials – Ö.Y., M.Ö., B.G., H.A., E.A, D.A.; Data Collection and/or Processing – Ö.Y., M.Ö., B.G., H.A., E.A, D.A.; Analysis and/or Interpretation – E.A., D.A., Ö. Y.; Literature Search – E.A., D.A., Ö. Y. Writing – E.A.; Critical Review – E.A., H.A.,B.M.

Declaration of Interests: The authors have no conflicts of interest to declare.

Funding: The authors declare that this study received no financial support.

REFERENCES

1. Aslanger E, Yıldırımtürk Ö, Türer Cabbar A, Değertekin M. Cardiovascular disintegration: a conceptual, model-based approach to heart failure hemodynamics. *Turk Kardiyol Dern Ars*. 2021;49(4):275-285. [CrossRef]
2. Frank O. Zur Dynamik des Herzmuskels. *Z Biol* (English Translation, *Am Heart J* 1959;58: 282). 1895;32:3703.
3. Starling EH. *The Linacre Lecture on the Law of the Heart*. New York: Longmans, Green & Co, 1918:1-27.
4. McDonagh TA, Metra M, Adamo M, et al. 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure. *Eur Heart J*. 2021;42(36):3599-3726. [CrossRef]
5. Humbert M, Kovacs G, Hoeper MM, et al. 2022 ESC/ERS Guidelines for the diagnosis and treatment of pulmonary hypertension. *Eur Heart J*. 2022;43(38):3618-3731. [CrossRef]
6. Nagueh SF, Smiseth OA, Appleton CP, et al. Recommendations for the evaluation of left ventricular diastolic function by echocardiography: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. *J Am Soc Echocardiogr*. 2016;29(4):277-314. [CrossRef]
7. Mullens W, Damman K, Harjola VP, et al. The use of diuretics in heart failure with congestion - a position statement from the Heart Failure Association of the European Society of Cardiology. *Eur J Heart Fail*. 2019;21(2):137-155. [CrossRef]
8. Smiseth OA, Refsum H, Tyberg JV. Pericardial pressure assessed by right atrial pressure: a basis for calculation of left ventricular transmural pressure. *Am Heart J*. 1984;108(3 Pt 1):603-605. [CrossRef]
9. Traboulsi M, Scott-Douglas NW, Smith ER, Tyberg JV. The right and left ventricular intracavitary and transmural pressure-strain relationships. *Am Heart J*. 1992;123(5):1279-1287. [CrossRef]
10. Tyberg JV, Taichman GC, Smith ER, Douglas NW, Smiseth OA, Keon WJ. The relationship between pericardial pressure and right atrial pressure: an intraoperative study. *Circulation*. 1986;73(3):428-432. [CrossRef]
11. Boltwood CM Jr, Skulsky A, Drinkwater DC Jr, Lang S, Mulder DG, Shah PM. Intraoperative measurement of pericardial constraint: role in ventricular diastolic mechanics. *J Am Coll Cardiol*. 1986;8(6):1289-1297. [CrossRef]
12. Smiseth OA, Thompson CR, Ling H, Robinson M, Miyagishima RT. A potential clinical method for calculating transmural left ventricular filling pressure during positive end-expiratory pressure ventilation: an intraoperative study in humans. *J Am Coll Cardiol*. 1996;27(1):155-160. [CrossRef]
13. Klotz S, Hay I, Dickstein ML, et al. Single-beat estimation of end-diastolic pressure-volume relationship: a novel method with potential for noninvasive application. *Am J Physiol Heart Circ Physiol*. 2006;291(1):H403-H412. [CrossRef]
14. Ponikowski P, Voors AA, Anker SD, et al. 2016 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure: The Task Force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC)Developed with the special contribution of the Heart Failure Association (HFA) of the ESC. *Eur Heart J*. 2016;37(27):2129-2200. [CrossRef]
15. DeLong ER, DeLong DM, Clarke-Pearson DL. Comparing the areas under two or more correlated receiver operating characteristic curves: a nonparametric approach. *Biometrics*. 1988;44(3):837-845. [CrossRef]
16. Alderman EL, Glantz SA. Acute hemodynamic interventions shift the diastolic pressure-volume curve in man. *Circulation*. 1976;54(4):662-671. [CrossRef]
17. Sibbald WJ, Driedger AA, Myers ML, Short AI, Wells GA. Biventricular function in the adult respiratory distress syndrome. *Chest*. 1983;84(2):126-134. [CrossRef]
18. Belenkie I, Smith ER, Tyberg JV. Ventricular interaction: from bench to bedside. *Ann Med*. 2001;33(4):236-241. [CrossRef]

19. Moore TD, Frenneaux MP, Sas R, et al. Ventricular interaction and external constraint account for decreased stroke work during volume loading in CHF. *Am J Physiol Heart Circ Physiol*. 2001;281(6):H2385-H2391. [CrossRef]
20. Parasuraman SK, Loudon BL, Lowery C, et al. Diastolic ventricular interaction in heart failure with preserved ejection fraction. *J Am Heart Assoc*. 2019;8(7):e010114. [CrossRef]
21. Hieda M, Sarma S, Hearon CM Jr, et al. Increased myocardial stiffness in patients with high-risk left ventricular hypertrophy: the hallmark of Stage-B heart failure with preserved ejection fraction. *Circulation*. 2020;141(2):115-123. [CrossRef]
22. Drazner MH, Velez-Martinez M, Ayers CR, et al. Relationship of right- to left-sided ventricular filling pressures in advanced heart failure: insights from the Escape trial. *Circ Heart Fail*. 2013;6(2):264-270. [CrossRef]
23. Campbell P, Drazner MH, Kato M, et al. Mismatch of right- and left-sided filling pressures in chronic heart failure. *J Card Fail*. 2011;17(7):561-568. [CrossRef]
24. Drazner MH, Prasad A, Ayers C, et al. The relationship of right- and left-sided filling pressures in patients with heart failure and a preserved ejection fraction. *Circ Heart Fail*. 2010;3(2):202-206. [CrossRef]