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ORIGINAL ARTICLE

Mitral E-wave to stroke volume ratio displays stronger diagnostic performance to identify elevated left ventricular filling pressures than mitral E/e' during passive leg lift: A cross-sectional study employing simultaneous echocardiography and catheterization

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Abstract

Background: Elevated filling pressure is a hallmark of heart failure (HF) and portends poor prognosis. Accurate diagnosis is challenging, given that patients with normal filling pressure at rest develop disproportionate elevation with sudden preload increase. We aimed to test the accuracy of the ratio between mitral inflow velocity (E) and left ventricular stroke volume (SV) to identify patients with elevated filling pressure with passive leg lifting (PLL) and compare this with other echocardiographic surrogates of filling pressure.

Methods: Doppler echocardiography and right heart catheterization (RHC) were simultaneously performed in 37 patients (11 males, mean age 67 ± 12 years) with exertional dyspnea. Twenty-six healthy controls (14 males, mean age 60 ± 12 years) were added as reference. SV, cardiac output (CO), tricuspid regurgitation peak gradient (TRG), mitral E-wave (E) and early myocardial velocity (e') were obtained at rest and with PLL. E/SV, E/CO and E/e' were calculated and correlated with invasive pulmonary capillary wedge pressures (PCWP) with PLL.

Results: During PLL, E/SV (AUC = 0.94) displayed stronger diagnostic ability to identify PCWP >15 mmHg than E/e' (AUC = 0.81), mitral E/A ratio (0.76) and resting invasive PCWP (0.84). An E/SV cutoff of >1.0 showed 88% sensitivity and 75% specificity to identify elevated PCWP. Further, 10 patients (27%) were reassigned during PLL from normal to postcapillary pulmonary hypertension (postCPH), and 6 patients (16%) switched diagnosis from precapillary PH (preCPH) to postCPH.

Conclusion: The novel E/SV ratio identifies patients with elevated PCWP with PLL and displays stronger diagnostic performance than routinely utilized echocardiographic measures such as E/e' in addition to resting, catheterization derived PCWP.

KEYWORDS
Doppler, filling pressure, heart failure, passive leg lifting, pulmonary hypertension

1 | INTRODUCTION

Elevated left ventricular (LV) filling pressure is a hallmark of heart failure (HF) and portends poor prognosis.[1] Accurate assessment of LV filling pressure poses a diagnostic challenge irrespective of ejection fraction.[2] Furthermore, HF patients with normal filling pressures at rest often display a disproportionate pressure rise when provoked by exertional maneuvers, which include rapid preload increase during passive leg lift (PLL).

In routine clinical practice, right heart catheterization (RHC) is performed to assess intracardiac hemodynamics both at rest and with provocation to identify elevated filling pressure and differentiate pulmonary hypertension (PH) hemodynamic subgroups based on pulmonary capillary wedge pressure (PCWP). Echocardiography is a first-line diagnostic and displays multiple advantages owing to its noninvasive, nonionizing, widely accessible and portable nature. Echocardiographic variables that rule out elevated LV filling pressure during rest and with provocation are of immense value for screening, effective triage to invasive assessment and subsequent therapy regulation.

Based on a hemodynamic concept and by using pressure-volume loops, the ratio between mitral inflow velocity (E = pressure) and LV stroke volume (SV = volume) could be a method estimating dynamic changes in filling pressures (Figure 1) but its use by during PLL has not been adequately studied. We aimed to compare the accuracy of E/SV with conventional echocardiographic variables to assess elevated LVFP during PLL employing reference standard RHC.

2 | METHODS

Doppler echocardiography was performed simultaneous with RHC at rest and during PLL in 37 patients presenting with suspected HF exertional dyspnea (11 males, mean age 67 ± 12 years). For comparison we used 26 healthy controls (14 males, mean age 60 ± 12 years) with no cardiovascular risk factors or cardiovascular medication. Classification of PH was made based on invasive PCWP according to recent recommendations.[3]

Echocardiography. Echocardiographic examination was performed simultaneously with RHC using a Vivid 9 system (GE Medical Systems, Horten, Norway) equipped with an adult 1·5–4·3 MHz phased array transducer. Standard views from the parasternal long axis, short axis and apical four-chamber views were used. LA volume was measured from the apical four-chamber view using area-length method and was further adjusted for body surface area (BSA) (LAVI). Flow velocities were obtained using Pulse–Wave (PW) and Continuous-Wave (CW) Doppler techniques as proposed by the American Society of Echocardiography and European Association of Cardiovascular Imaging.[4,5]

All Doppler recordings were made at a sweep speed of 50–100 mm s^{-1} with a superimposed ECG (lead II). Offline echocar-

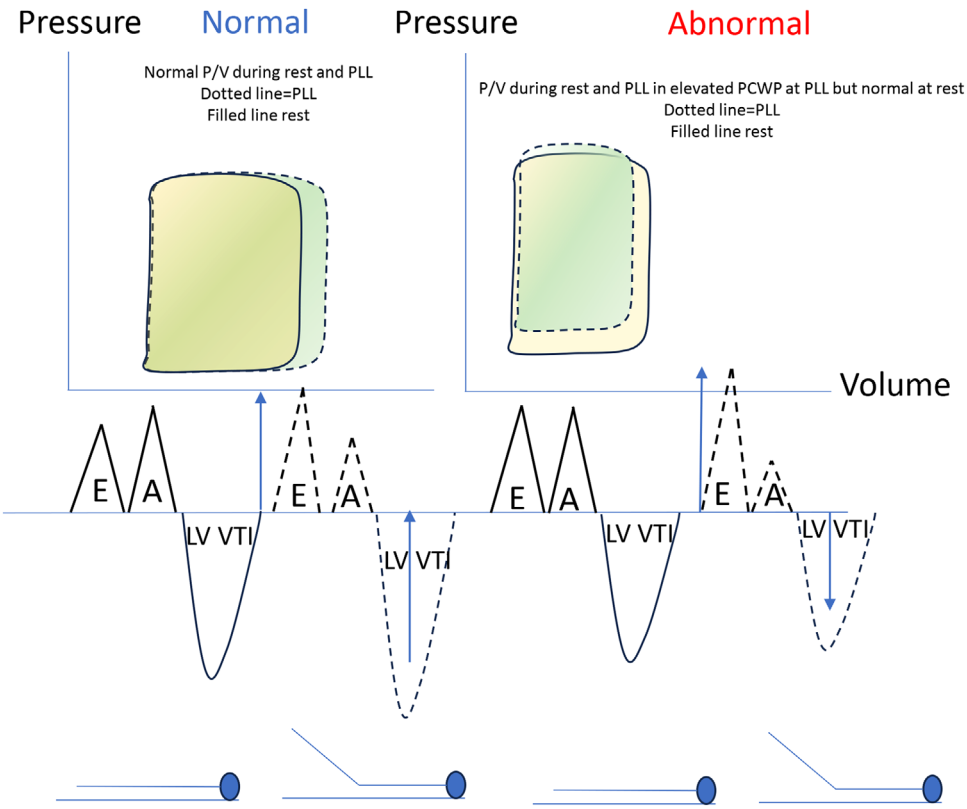

FIGURE 1 Hemodynamic pressure/volume and Doppler aspects on resting and leg raise in normal and patients(abnormal) with elevation in PLL filling pressures. PLL, passive leg lifting.

diographic analysis was made using commercially available software (General Electric, EchoPac version BT 13, 113.0, Waukesha, Wisconsin, USA), and means of three consecutive cardiac cycles were calculated. The study protocol was approved by the Regional Ethic Committee in Umeå, Sweden (Dnr 07–092 M), and all subjects provided informed consent to participate in the study.

Using DE ahead of standard measures we used SV, cardiac output (CO), tricuspid regurgitation peak gradient (TRG), early diastolic mitral velocities from blood flow (E) and myocardium (e'), all at rest and PLL. To calculate SV during rest, LV outflow tract (LVOT) diameter was assessed in the parasternal long axis view. A 5 mm sample volume was then placed in the LVOT of the apical five-chamber view to obtain the velocity time integral (VTI). SV was calculated as area of the LVOT multiplied by LVOT VTI. During PLL, LVOT VTI was reassessed. LVOT, however, was not re-evaluated during PLL to minimize any potential errors related to measurement. We assumed that LVOT was unchanged at PLL.

We then calculated E/SV, E/CO and E/e' and correlated all to PCWPs at rest and PLL. At ventricular systole left atrial reservoir strain (PALS) and left ventricular global longitudinal strain/GLS) were measured from apical four chamber view.

Right heart catheterization: Venous access was established by inserting a canula in a jugular, medial cubital vein or in the femoral vein. A retrograde catheterization was then performed using a Swan-Ganz Standard Thermodilution Catheters (Edwards Lifesciences). Mean right atrial pressure (RAP), systolic and end-diastolic right ventricular pressure (RVEDP), pulmonary artery systolic, mean and diastolic pressures (PASP, PAMP and PADP, respectively) and PCWP were all measured according to guidelines.[3]

PCWP pressures were calculated as mean of 10 consecutive beats. Blood samples for estimating oxygen saturation were drawn from the superior canal vein, pulmonary artery and femoral artery, and 8% was considered a significant step up between superior vena cava (SVC) and pulmonary artery. CO was determined by thermodilution technique. Pulmonary vascular resistance (PVR) was calculated using the equation PAMP−PCWP (transpulmonary gradient) divided by CO. PH was defined as mean pulmonary pressures >20 mmHg.

TABLE 1 Resting and PLL data.

	Rest	Passive leg lifting	*p*-value
Clinical characteristics			
Age, years	67 ± 12		
Sex, male/female	11/37		
BSA, m^2	1.9 ± .3		
SBP, mmHg	138 ± 18		
DBP, mmHg	81 ± 10		
NT-proBNP, ug/L	606 ± 535		
RHC			
PreCPH/postCPH/normal/CPH	8/23/0/6	5/12/2/18	
PCWP, mmHg	11 ± 3	16 ± 4	<.001
mPAP, mmHg	26 ± 10	31 ± 10	<.001
CO, L/min	4.8 ± 1.3	6.2 ± 3.5	Ns
PVR, WU	3.3 ± 2.2	–	
Doppler Echo			
LVDD, mm	47 ± 7	–	
IVSd, mm	12 ± 3	–	
LVEF, % (%HFpEF)	50 ± 10 (51%)	–	
MF E/A	1.3 ± .8	–	
E/e'(lateral)	10.0 ± 4.0	9.3 ± 4.8	Ns
E, cm/s	70 ± 23	76 ± 23	.006
SV, mL	72 ± 22	71 ± 15	Ns
CO, L/min	4.8 ± 1.2	5.0 ± 1.3	ns
GLS,%	−18.0 ± 4.6	–	
PALS,%	15.0 ± 8.2	–	
RV-RA, mmHg	35 ± 14	36 ± 12	ns
E/SV, cm/s/mL	1.1 ± .5	1.1 ± .4	ns
E/CO, cm/s/L/min	15.5 ± 5.6	16.6 ± 5.6	ns

Abbreviations: BSA, body surface area; CO, cardiac output; CPH, capillary pulmonary pressure; E and e', early diastolic blood and myocardial velocity; GLS, global longitudinal strain; LAVI, left atrial volume index; mPAP, mean pulmonary artery pressures; PALS, peak atrial longitudinal strain; RA, right atrial; RHC, right heart catheterization; RV, right ventricle; SV, stroke volume.

2.1 | Statistics

Continuous variables were presented as mean ± SD or median (IQR) and categorical variables as numbers and percentage. Data comparing resting and PLL data are done by parametric paired *t*-test. Results comparing patients with or without elevated PCWP during PLL were done using Mann–Whitney test. Correlations between reference standard invasive measurements and novel echocardiographic estimates were tested using Pearsons two-tailed test. Receiver operating characeteristics (ROC) analysis was performed to evaluate diagnostic performance of echocardiographic variables to assess elevated PCWP at PLL. Finally, regression analysis was done to study associations of Doppler echocardiographic with invasive measured PCWP at PLL. IBM SPSS statistics version 23.0 was employed for analysis. A p-value < .05 was considered statistically significant.

3 | RESULTS

Clinical, hemodynamic and echocardiographic characteristics are shown in Table 1. Of the 37 patients, 10 had heart failure with preserved ejection fraction (HFpEF) mainly due to hypertensive disease(n = 9) but one had ATTR cardiac amyloidosis, 11 had rheumatic disease (1 mixed connective disease, 9 had scleroderma and 1 systemic

lupus erythematosus (SLE), 6 had chronic thromboembolic pulmonary hypertension (CTEPH), 3 had chronic obstructive pulmonary disease (COPD), 4 had unclear dyspnea and 1 had hypertrophic cardiomyopathy (HCM), 1 had valve prothesis dysfunction and had idiopathic pulmonary arterial hypertension (PAH). No patients had significant (more than mild) valvular disease.

3.1 | Hemodynamic groups at rest and with PLL

Based on RHC measurements, 62% had precapillary PH, none had isolated postcapillary PH, 16% had combined pre- and postcapillary PH, and 22% normal hemodynamics. During PLL, 32% had precapillary PH, 5% had isolated post capillary PH, 46% combined pre- and postcapillary PH and 14% normal hemodynamics.

3.2 | Echocardiography

On echocardiography, patients demonstrated a significant increase in transmitral E wave velocity ($p = .006$) during PLL. When patients were subgrouped based on those that demonstrated elevated invasive PCWP (>15 mmHg) with PLL and those that did not, those that demonstrated a disproportionate elevation in PCWP displayed higher PCWP, PCWPv-wave and mPAP at rest ($p < .05$) . In addition, resting RV-RA peak systolic gradient was significantly different ($p < 0.05$) between groups. During PLL, E/SV was higher and SV lower ($p < .001$) lower in the group that displayed elevated PCWP, Figure 1. E/CO and E/ were higher ($p < 0.05$), in the elevated PCWP group, Table 2.

E/SV demonstrated a strong correlation with PCWP during PLL ($r = 0.73$, $p < 0.001$), Figure 2. Furthermore, patients displayed higher E/SV than controls during PLL ($1.13 \pm .42$ vs. $.81 \pm .15$, $p < 0.001$)

We used ROC analysis to test which variable demonstrated highest accuracy to identify PCWP >15 mmHg at PLL among those that demonstrated significant difference in Table 2. In addition, we also tested resting transmitral E/A and SV. In all patients ($n = 37$), E/SV PLL identified patients with PCWP >15 mmHg during PLL with an area under the curve (AUC) of 0.94. AUC for E/e' PLL was 0.81, for mitral E/A ratio at rest was 0.76, for resting PCWP and PCWPv were 0.84 and 0.81 respectively, Figure 3. E/SV at PLL > 1.0 had a sensitivity of 82% and a specificity of 81% and positive predictive and negative predictive values of 82% and 81% to identify patients with a PCWP >15 mmHg during PLL .

4 | DISCUSSION

In this study, the mitral E-wave to SV ratio displayed a strong ability to identify elevated LV filling pressure with PLL. Further, E/SV > 1 cm/s/mL displayed high sensitivity, specificity and greater accuracy when compared with mitral E/e' as well as resting E/A ratio and catheterization derived resting PCWP. Our findings suggest a role for this novel echocardiographic ratio as a strong adjunct to the assessment of filling pressures during PLL in the setting of suspected HF.

Estimation of left ventricular filling pressures at rest using Doppler Echocardiography is recommended as a part of clinical HF examinations but has its limitations.[6,7]

Echocardiographic algorithms take into consideration mitral E wave, mitral e' velocity, TR velocity and indexed left atrial volume (LAVI). We have recently demonstrated that adding left atrial strain to the diagnostic algorithm improves its accuracy in the setting of preserved EF.[8] Additional studies suggest a role for alternative algorithms and exercise echocardiography in LV filling pressure assessment.[9,10] Here, E/e' > 15 and TR velocity > 3.4 m/s were strong predictors for elevated PCWP. In another paper E/e' mean or E/e' septal wall > 15 or 14 and TR velocity > 2.8 m/s threshold for positive diastolic stress test. The mitral E/e' ratio is an important component of filling pressure assessment.[9] However, conventional variables demonstrate modest ability to predict elevated LVEDP with rapid preload increase or under provocation.

Despite new techniques and additional methods, patients with indeterminate filling pressure and those with normal resting pressures pose a diagnostic challenge. In RHC studies, we have found that incorporating a simple, easy-to-perform PLL to suddenly increase preload and test LV compliance identifies patients that develop high filling pressures due to reduced LV compliance.[11] However, noninvasive estimates of elevated PCWP under PLL using Doppler echocardiography were not exhaustively studied and has tremendous clinical value.[11]

Doppler echocardiography has shown ability to detect elevated LVFP during exercise by diastolic stress test.[12,13] Earlier studies suggest that markers for positive diastolic stress test (i.e., elevated filling pressures) were septal e′ > 7 cm/s, septal E/e′ at exercise >15 (>14 for mean E/e′) and TR velocity >2.8 m/s. None of these variables differed significantly between those that demonstrated normal and elevated PCWP during PLL in this study.

We suggest that the E/SV ratio may demonstrate value to identify patients with elevated PCWP under PLL. Earlier studies show a lack of increase in SV during PLL both in patients with PH[14] and in HFpEF.[15] We demonstrate similar findings, suggesting that the inability to augment SV may be owing to the LV operating on the plateau limb of the Frank–Starling curve. In this setting, preload reserve is likely exhausted, leading to an increase in LV filling pressure to preserve CO. Obokata and colleagues also showed that mitral E wave velocity increases both in HFpEF and in hypertensive controls without signs of HF during PLL.[15] Increase in transmitral flow in HFpEF can be attributed to a rise in filling pressure, while in controls, this may be owing to LV suction. The utilization of RHC as an invasive reference in our study further corroborates these findings.

Another striking finding in our results were the number of patients which needed reclassification to a different PH group during PLL.[16]

At rest 62% had precapillary PH which reduced to 32 % at PLL. None had isolated post capillary PH which increased to 5% at PLL. Twenty-two percent normal hemodynamics which reduced to 14% during PLL. Finally, 16% had combined pre and post capillary PH which increased to 46% at PLL. Thus, more patients were found having PH at PLL and

TABLE 2 Clinical data, resting and PLL echocardiographic data in patient subgroups with or without increased PCWP during PLL.

	Normal PCWP PLL ($n = 19$)	Elevated PCWP at PLL ($n = 18$)	p-value Mann-Whitney
Clinical characteristics			
Age, years	68 (20)	71 (15)	ns
Sex, male	4/17	7/9	ns
BSA, m^2	2.0 (.3)	1.8 (1.3)	ns
SBP, mmHg	133 (50)	135 (44)	ns
DBP, mmHg	80 (16)	77 (16)	ns
NT-proBNP, ug/L	344 (698)	457 (132)	ns
NT-proBNP ug/L lg(10)	2.5 (.7)	2.6 (.7)	ns
RHC rest			
PreCPH/postCPH/normal PAP/CPH	8/0/12/1	4/2/7/3	
PCWP, mmHg	8 (5)	13 (14)	<.001
PCWP v, mmHg rest	11 (6)	19 (8)	<.001
mPAP, mmHg	20 (35)	28 (27)	0.04
CO, L/min	5.3 (2.0)	5.0 (2.6)	ns
PVR,WU	2.6 (3.7)	3.0 (1.2)	ns
Doppler Echo			
Rest			
LVEF,% (%HFpEF)	51 (18) (69)	48 (13) (42)	ns
LAVI, mL/m^2	30 (21)	42 (17)	ns
MF E/A	1.0 (.3)	1.1 (1.5)	ns
E/e' (lateral)	10.2 (4.3)	10.0 (7.1)	ns
E, cm/s	74 (26)	74 (24)	ns
SV, mL	70 (18)	67 ± 13	ns
CO, L/min,	4.9 ± 1.4	4.4 ± 1.9	ns
GLS, %	−19.6 (6.3)	−17.5 (8.3)	ns
PALS, %	16.2 (12.1)	11.9 (10.7)	ns
RV-RA, mmHg	25 (22)	37 (23)	0.04
E/SV, cm/s/mL	1.0 (.5)	1.2 (.6)	ns
E/CO, cm/s/L/min	14.2 (8.1)	18.1 (7.2)	ns
PLL			
CO, L/min	5.2 ± 1.2	4.7 ± 1.3	ns
e' (lateral)	8.9 ± 2.8.	7.9 ± 1.8	ns
E/e' (lateral)	9.8 ± 6.2	11.4 ± 5.0	0.01
E/SV, cm/s/mL	.96 ± .42	1.36 ± .30	<0.001
SV, mL	79 ± 14	63 ± 12	<0.001
E/CO, cm/s/L/min	15.1 ± 1.6	18.4 ± 5.4	0.03
RV-RA, mmHg	35 ± 9	36 ± 12	ns
E, cm/s	75 ± 32	82 ± 17	ns

Note: Data presented as median (IQR).

Abbreviations: BSA, body surface area; CO, cardiac output; CPH, capillary pulmonary pressure; E and e', early diastolic blood and myocardial velocity; RV, right ventricle; RA, right atrial; SV, stroke volume; CO, cardiac output. GLS, global longitudinal strain; LAVI, left atrial volume index; mPAP, mean pulmonary artery pressures; PALS, peak atrial longitudinal strain; RHC, right heart catheterization.

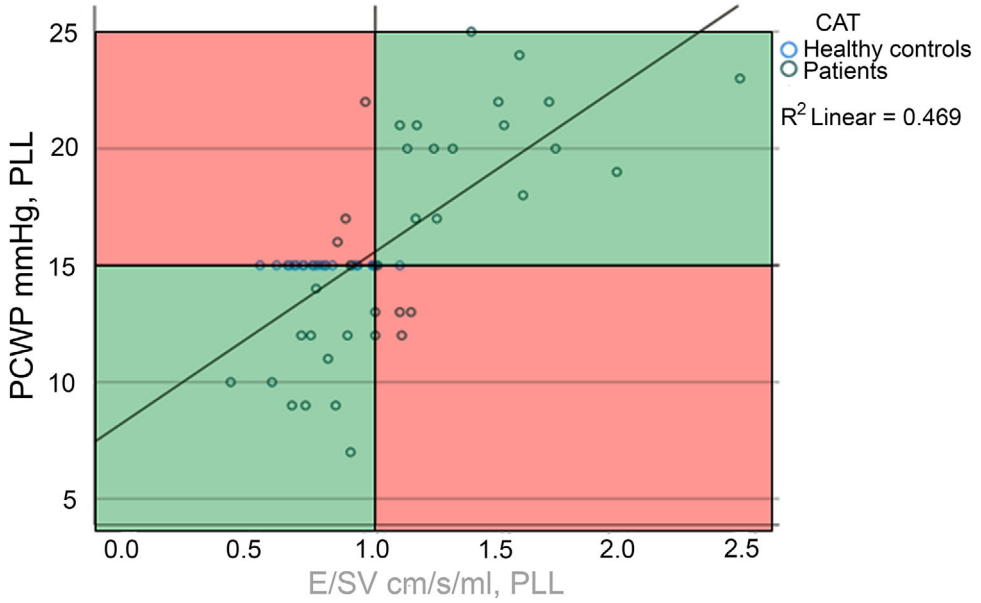

FIGURE 2 Scatterplott of the relationship between E/SV during PLL and PCWP during PLL. Circles are patients and cross are controls. Vertical line is 1.05 and horizontal line 15 mmHg. E, pressure PCWP, pulmonary capillary wedge pressures; PLL, passive leg lifting; SV, stroke volume.

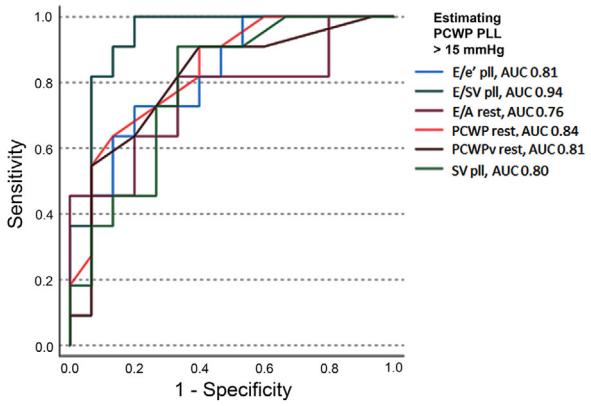

FIGURE 3 In all patients ($n = 37$) E/SV PLL identified patients with PCWP > 15 mmHg during PLL with an AUC of .94. AUC for E/e' PLL was .81, AUC for MF E/A rest was .76, AUC for resting PCWP and PCWPv .84 and .81. E/SV PLL > 1.0 had a sensitivity of 8% and a specificity of 76% identifying patients with a PCWP during PLL > 15 mmHg. AUC, area under the curve; E, pressure PCWP, pulmonary capillary wedge pressures; PLL, passive leg lifting; SV, stroke volume.

a significant number of patients developed pre and post capillary PH mainly because of increased PCWP at PLL.

Doppler echocardiography is related to limitation with low accuracy to estimate the filling pressures during a stress maneuver, i.e., preload stress or supine exercise.[17] However, an accurate measure for elevated filling pressures at cardiac stress is important as many patients with HF have normal filling pressures during rest which demasks during stress. Passive leg lifting (PLL) is a quick and easily performed maneuver which increases preload and tests the cardiac compliance. Therefore, performing PLL and Doppler echocardiography simultaneously might be an important diagnostic method to evaluate patients with stress induced increase in LV filling pressures.

4.1 | Limitations

Our study cohort comprised patients with precapillary PH and HF, and patient heterogeneity may be considered a major limitation in this study. However, a heterogeneous population permitted closer inspection of the transition of hemodynamic subgroups with PLL, which suggests that hemodynamic classifications at rest are misleading. We believe that this finding is of scientific interest and may not have been showcased if we had strictly defined populations. Additionally, the larger representation of heart failure with reduced ejection fraction (HFrEF) in those having elevated PCWP with PLL may limit the interpretation of our results. Finally, the relatively small number of patients in this cohort where echocardiography and catheterization were simultaneously performed is a limitation in this study. The novelty of these findings would, hence, need to be validated in larger groups.

5 | CONCLUSION

The novel E/SV ratio identifies patients with elevated PCWP during PLL and displays stronger diagnostic performance than routinely utilized echocardiographic measures such as E/e' in addition to resting invasive PCWP. However, its use in clinical practice needs to be validated in larger cohorts.

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CONFLICT OF INTEREST STATEMENT

PL has disclosed with Pfizer as consulting lecturer and as member of advisory board.

DATA AVAILABILITY STATEMENT

The majority of the data underlying this article can be shared upon reasonable request to the corresponding author.

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