Longitudinal Strain and Strain Rate for Estimating Left Ventricular Filling Pressure in Heart Transplant Recipients

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Traditional parameters have limited value to estimate left ventricular filling pressure (LVFP) in orthotropic heart transplant (OHT) recipients. We hypothesized that global longitudinal strain (GLS), diastolic, and systolic strain rate (SR) would be depressed in OHT recipients with elevated LVFP and could overcome the limitations of traditional parameters. We studied consecutively OHT patients at the time of endomyocardial biopsies and retrospectively pretransplantation studies conforming to the same protocol. Comprehensive echocardiography with strain measurements was performed. Results were compared with pulmonary capillary wedge pressure (PCWP) obtained from right heart catheterization that was performed just after the echocardiography study. In all, 74 studies were performed in 50 OHT recipients. Mean PWCP was 11.8 ± 4.3 mm Hg (range: 4 to 25 mm Hg). Several parameters, but not left atrial volume index, mitral inflow velocities, annular velocities, and their ratio (E/e'), were different between studies with normal (n = 47) and elevated PCWP (n = 27). Area Under Curve for GLS (0.932*), E/e'$_{SR}$ (0.849*), and systolic SR (0.848*) (*p <0.0001) were more accurate than traditional parameters for predicting PCWP>12 mm Hg. GLS, systolic SR and E/e'$_{SR}$ remained accurate regardless of LV ejection fraction and allograft vasculopathy. Meanwhile, E/e' was accurate to predict PWCP in native failing hearts before transplantation. Changes in GLS and E/e'$_{SR}$ tracked accurately changes in PCWP. In conclusion, traditional indices of diastolic function perform poorly in OHT recipients, whereas GLS and E/e'$_{SR}$ provide reliable means of LVFP, irrespective of ejection fraction and allograft vasculopathy. These parameters also track reasonably well the changes in LVFP. **© 2020 Elsevier Inc. All rights reserved.** **(Am J Cardiol 2020;137:63−70)**

Graft failure, despite insidious symptoms, is an important cause of mortality after orthotropic heart transplantation (OHT). Transthoracic echocardiography is the main imaging tool for the follow-up of OHT patients worldwide. Although hemodynamic evaluation is important for assessing the graft performance, accurate estimation of left ventricular filling pressure (LVFP) by echocardiography is challenging in OHT recipients.[1] Traditional parameters cannot be extended to this patient population due to several anatomical and functional particularities of the allograft[2]: Left atrial (LA) anatomy and function, consequently mitral inflow and pulmonary vein flow patterns are altered by anastomoses between donor and remnant recipient tissue.[3] Tricuspid regurgitation (TR) occurs irrespective of left ventricular (LV) diastolic function due to residual pulmonary vascular resistance and injury to tricuspid valve by repeated biopsies. Echocardiographic patterns of pseudodiastolic dysfunction are frequent and patients with apparently restrictive filling pattern do not necessarily have elevated LV end diastolic pressures.[4] Previous studies have shown that speckle tracking derived global longitudinal strain (GLS), systolic and diastolic strain rate (SR) could complement the assessment of LV diastolic function when traditional parameters were less accurate.[5−8] Accordingly, we hypothesized that GLS, diastolic and systolic SR would be depressed in OHT patients with elevated LVFP and could overcome the limitations of conventional parameters for the estimation of LVFP in these patients.

Methods

All OHT patients followed from January 2017 to December 2019 at our center were included consecutively at the time of surveillance endomyocardial biopsies regardless of symptoms according to our institutional follow-up protocol.[9] Patients were excluded if they had biatrial anastomoses (n = 2) or inadequate image quality for strain analysis (n = 2). All were in normal sinus rhythm, had no conduction abnormality, pericardial effusion or more than mild valvular disease. We excluded examinations within the first 3 months after OHT (n = 2) to allow allograft stabilization.[10] We included data obtained at 2 different time points with different hemodynamic status in 24 patients to assess the capability of the investigated parameters to track

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hemodynamic changes. These repeated studies were performed upon clinical suspicion of rejection or to check the resolution of rejection after treatment. In addition, we retrospectively studied 17 patients who underwent pre-transplantation work-up in our institution conforming the same protocol after excluding those with atrial fibrillation, frequent ventricular extrasystoles, respiratory or inotropic support, renal replacement therapy, ventricular assist devices, pacemaker rhythm, severe mitral regurgitation, prosthesis or valve repair and significant mitral annular calcification. The study protocol was approved by the Institutional Review Board and Institutional Ethical Committee for Human Research and complied with the Declaration of Helsinki. All patients gave written informed consent.

Traditional 2-dimensional echocardiography was performed by using Vivid E9 ultrasound system (GE Healthcare, Horten, Norway). Left ventricular (LV) and LA volumes were measured from the apical 4- and 2-chamber images using the biplane discs' summation method.[11] Early and late transmitral inflow (E,A) and TR jet velocities were measured from the apical 4-chamber view by using pulsed and continuous-wave Doppler, respectively. Pulsed-wave tissue Doppler velocities were acquired from the lateral and septal mitral annulus. The ratio E/e' (e', average of septal and lateral early diastolic mitral annular velocities) was calculated as recommended.[2] All Doppler measurements were averaged over 3 heart beats.

Speckle-tracking strain analyses were performed offline from digitally stored cine-loops at 40 to 90 frame/second, using the EchoPac software (version 113, GE Healthcare). Curved regions of interest were manually traced on the endocardial borders on apical 4-, 2-chamber and long axis images. Regional longitudinal strain and SR curves were obtained from 6 segments in each view after manual adjustment whenever necessary to optimize tissue tracking. GLS was derived from the automated software algorithm. Peak systolic and early diastolic SR (S_{SR}, e'_{SR}) were measured from the average SR curve of each apical view, then, the average of 3 apical views was computed to obtain global peak S_{SR} and e'_{SR}. Global E/e'_{SR} was also calculated. We did not measure SR during isovolumetric relaxation because the short lived period characteristic of the parameter is not suitable for the optimal frame rate of speckle tracking.

Invasive hemodynamic measurements were performed just after the echocardiography study by interventional cardiologists who were blinded to echocardiography findings. A traditional 6-F triple Cournand catheter and a 5F pigtail catheter were introduced into the femoral vein and artery, respectively, by the Seldinger technique. Right chamber pressures, PCWP as a means of LVFP, systolic and mean pulmonary artery pressures were measured. The wedge position was verified by observing the typical changes in waveforms, by fluoroscopy and by oxygen saturation if necessary to avoid erroneous damped pulmonary artery pressure recording. Acute rejection was determined according to the standardized International Society for Heart and Lung Transplantation definitions and nomenclature.[12]

Data are presented as mean values ± standard deviation for continuous variables, as percent for categorical variables unless otherwise mentioned. Least squares linear regression analysis was used to correlate PCWP measurements and echocardiographic parameters as continuous variables. One-way ANOVA test was used to compare continuous variables between independent groups. We tested several echocardiographic parameters in a logistic regression model to determine the predictors of elevated PCWP. Parameters with a p-value <0.1 in the univariate analysis were entered into a multiple regression model as covariates. To avoid multicolinearity between the univariate predictors, a correlation coefficient of <0.7 was set. Accordingly, S_{SR} and GLS as well as e'_{SR} and E/e'_{SR} were not evaluated together in multivariate model for their independent predictive value. We used receiver operating characteristics (ROC) curves and areas under the curves to study the diagnostic performance of the tested parameters, from where, cut-off values were determined by using maximum Youden Index. Differences between 2 observations from the same patients were calculated as (first observation value—second observation value) for PCWP, GLS and $E/e'_{SR..}$ Changes (Δ) in tested parameters (GLS, E/e'_{SR}) were plotted graphically against changes in PCWP to represent their relation. Intra- and inter-observer reliabilities of SR measurements were described by intra-class correlation coefficients (2-way mixed effects, absolute agreement, and single measure). Likewise, intra- and inter-observer reliabilities of strain measurements in OHT patients were previously reported from our center.[9] Two-tailed p values <0.05 were considered significant. We used a standard statistical software program (SPSS version 20; SPSS, Inc., Chicago, Illinois)

Results

A total of 74 data sets were obtained from 50 patients after OHT. Patients' characteristics are presented in Table 1. Twenty-four patients had 2 studies at different time points

Table 1
Patients characteristics

Variable	All Patients (n = 50)
Donor age (years)	32 ± 12
Recipient age (years)	36 ± 14
Women	16 (35%)
Time from transplantation (months)	62.9 ± 46
Fasting glucose (mg/dl)	112 ± 42
Creatinine (mg/dl)	0.89 ± 0.28
Systolic blood pressure (mm Hg)	122 ± 18
Diastolic blood pressure (mm Hg)	77 ± 10
Heart rate (beats/minute)	87 ± 10
Body mass index (kg/m^2)	25.2 ± 5.2
Beta blockers	26 (52%)
Calcium channel blockers	11 (22%)
ACEI/ARB	13 (26%)
Statins	26 (52%)
Diuretics	16 (32%)
Tacrolimus	40 (80%)
Sirolimus	8 (16%)
Mycophenolate	49 (98%)
Prednisone	40 (80%)

Abbreviations: ACEI = Angiotensin converting enzyme inhibitors; ARB = angiotensin receptor blockers.

Table 2
Echocardiographic measurements in groups with normal and elevated PCWP

Variable	Pooled Data (n = 74)	PCWP (mm Hg) ≤12 (n = 47)	PCWP (mm Hg) >12 (n = 27)	p value
E velocity (cm/s)	93 ± 20	92 ± 20	95 ± 22	0.53
E/A	2.02 ± 0.7	1.9 ± 0.7	2.2 ± 0.6	0.09
E deceleration time (ms)	140 ± 27	146 ± 27	130 ± 25	0.02
Average e' (cm/s)	11.3 ± 2.5	11.7 ± 2.2	10.7 ± 2.8	0.13
E/e'	8.6 ± 2.9	8.0 ± 2.3	9.5 ± 3.6	0.06
LAVi (ml/m^2)	33.2 ± 11.1	32.8 ± 10.5	33.8 ± 12.2	0.71
TR jet velocity (m/s)	2.4 ± 0.4	2.3 ± 0.3	2.5 ± 0.4	0.04
Ejection fraction (%)	54.6 ± 7.2	56.0 ± 5.3	52.2 ± 9.3	0.03
EDVi (ml/m^2)	47.8 ± 10.1	45.4 ± 7.7	52.0 ± 12.5	0.01
LV mass index (g/m^2)	61.7 ± 13.4	61.4 ± 11.7	62.1 ± 15.9	0.83
GLS (%)	−16.0 ± 3.5	−17.9 ± 2.5	−12.8 ± 2.5	0.0001
$S_{SR}(^{-1})$	−1.1 ± 0.33	−1.27 ± 0.27	−0.88 ± 0.27	0.0001
e'_{SR} ($^{-1}$)	1.53 ± 0.41	1.73 ± 0.37	1.18 ± 0.24	0.0001
E/e'_{SR}	65.2 ± 22.3	55.3 ± 15.7	82.5 ± 21.8	0.0001

Abbreviations: E = early mitral inflow velocity; e' = mitral annular early diastolic velocity; EDVi = end diastolic volume index; GLS = global ;longitudinal strain; LAVi = left atrial volume index; LV = left ventricle; PCWP = pulmonary capillary wedge pressure; S = systolic; SR = strain rate; TR = tricuspid regurgitation.

with different hemodynamic measurements. In all, PCWP was 11.8 ± 4.3 mm Hg (range 4 to 25), systolic PAP 27.2 ± 7.9 mm Hg (range 8 to 59), mean PAP 17.1 ± 5.9 mm Hg (range 7 to 47), right atrial pressure 5.7 ± 4.0 mm Hg (range 1 to 18). In the pooled dataset LV EF, GLS, S_{SR}, e'_{SR} were significantly decreased while LV end-diastolic volume index (EDVi), E wave deceleration time, TR velocity, and E/e'_{SR} were significantly increased in studies with PCWP >12 mm Hg in comparison to those with PCWP ≤12 mm Hg. However, E/A, e', and E/e' were not different

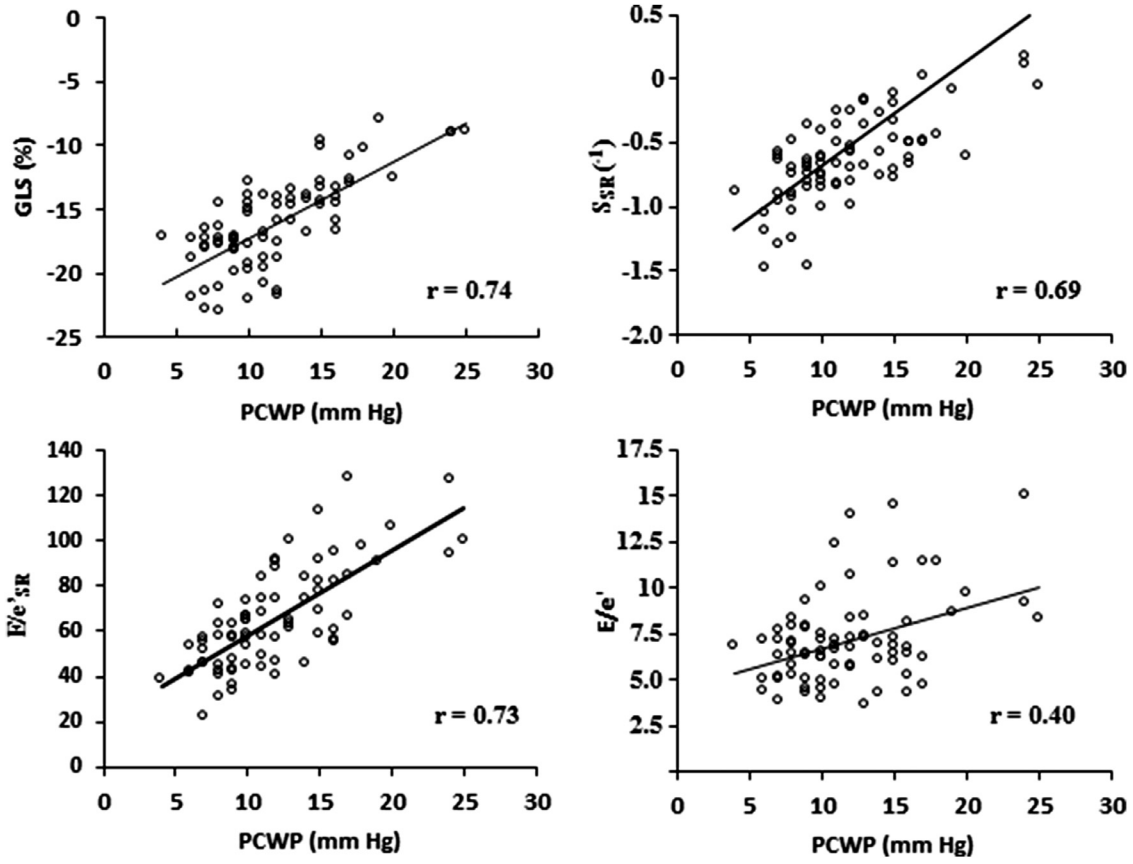

Figure 1. Correlations of PCWP with echocardiographic parameters: GLS, S_{SR}, E/e'_{SR}, and E/e'. Note the modest correlation of E/e' with PCWP (open circles; EF ≥50%, filled circles; EF<50%).

Table 3
Correlation of echocardiographic parameters with PCWP, univariate and multivariate determinants of PCWP

	Correlation Coefficient	p Value	Odds Ratio (95% CI)	p Value	Odds Ratio (95% CI)
E/A	0.34†	0.1	1.89(0.90−3.96)		
E deceleration time	−0.28*	0.03	0.98(0.96−1.00)	0.07	0.9(0.88−1.01)
E/e'	0.40‡	0.07	1.17(0.99−1.39)	0.19	0.7(0.37−1.22)
TR jet velocity (m/s)	0.24*	0.05	3.97(0.99−15.94)	0.26	0.1(0.01−7.80)
Ejection fraction (%)	−0.39†	0.03	0.93(0.86−0.99)	0.53	1.5(1.00−2.14)
EDVi (ml/m^2)	0.37†	0.01	1.07(1.02−1.13)	0.04	1.5(1.02−2.19)
GLS (%)	0.74‡	<0.0001	2.5(1.64−3.94)	<0.01	5.3(1.47−19.3)
S_{SR} ($^{-1}$)	0.69‡	<0.0001	2.2(5.44−10.17)	...	...
e'$_{SR}$ ($^{-1}$)	−0.65‡	<0.0001	0.02(0.00−0.03)	...	...
E/e'$_{SR}$	0.73‡	<0.0001	1.1(1.04−1.12)	0.013	1.3(1.05−1.53)

Abbreviations: E = early mitral inflow velocity; e' = mitral annular early diastolic velocity; EDVi = end diastolic volume index; EF = ejection fraction; GLS = global longitudinal strain; PCWP = pulmonary capillary wedge pressure; S = systolic; SR = strain rate; TR = tricuspid regurgitation.

S_{SR} with GLS, e'$_{SR}$ with E/e'$_{SR}$ are not evaluated together in the multivariate model because of their correlation coefficient ≥0.70 to avoid multicolinearity.

*: p <0.05
†: p<0.01
‡: p<0.001.

between the groups (Table 2). Figure 1 scrutinizes the correlation of PCWP with GLS, S_{SR}, E/e'$_{SR}$ and E/e' in studies with (n = 17) and without (n = 57) depressed EF.

The correlation of echocardiographic measurements with PCWP and the predictors of elevated PCWP are presented in Table 3. In the variables that correlated significantly with PCWP; E wave deceleration time, LV EDVi, EF, GLS, S_{SR}, e'$_{SR}$, and E/e'$_{SR}$ emerged as univariate determinants of elevated PCWP. In these, E/e'$_{SR}$ and GLS were independently associated with LVFP.

The performance of echocardiographic parameters to predict elevated PWCP in OHT patients are presented by ROC curve analyses. As observed, strain and SR parameters performed clearly better than traditional parameters to determine high LVFP in OHT recipients (Figure 2). The best fit cut-offs for GLS (−15%), E/e'$_{SR}$ (63), SRs (−1.12 s^{-1}), and e'$_{SR}$ (1.4 s^{-1}) had 85%, 79%, 82%, and 89% sensitivity and 85% 72%, 70%, and 83% specificity, respectively, for predicting elevated PCWP. Furthermore, GLS and E/e'$_{SR}$ performed equally good to predict elevated PCWP in subgroups with preserved (n = 57) or depressed (n = 17) LV EF and in subgroups with (n = 12) or without (n = 62) cardiac allograft vasculopathy of grade >1 (Figure 3).

Notwithstanding, a strong correlation between PCWP and E/e' (r = 0.74, p <0.001) was observed in

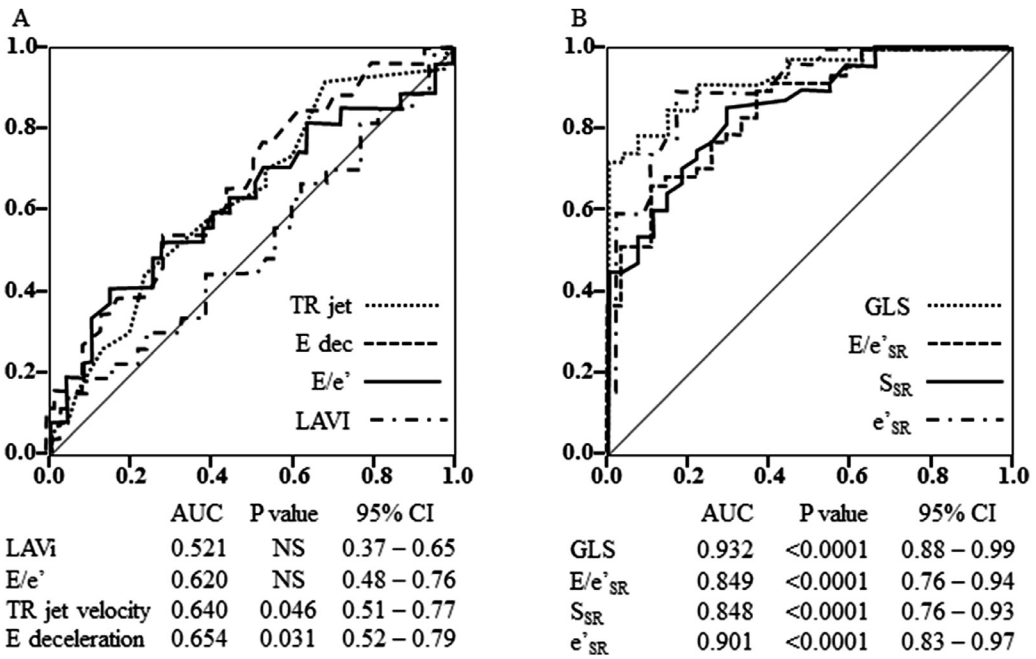

Figure 2. ROC curve analyses to determine PCWP >12 mm Hg. (*A*) Traditional parameters, (*B*) Deformation parameters

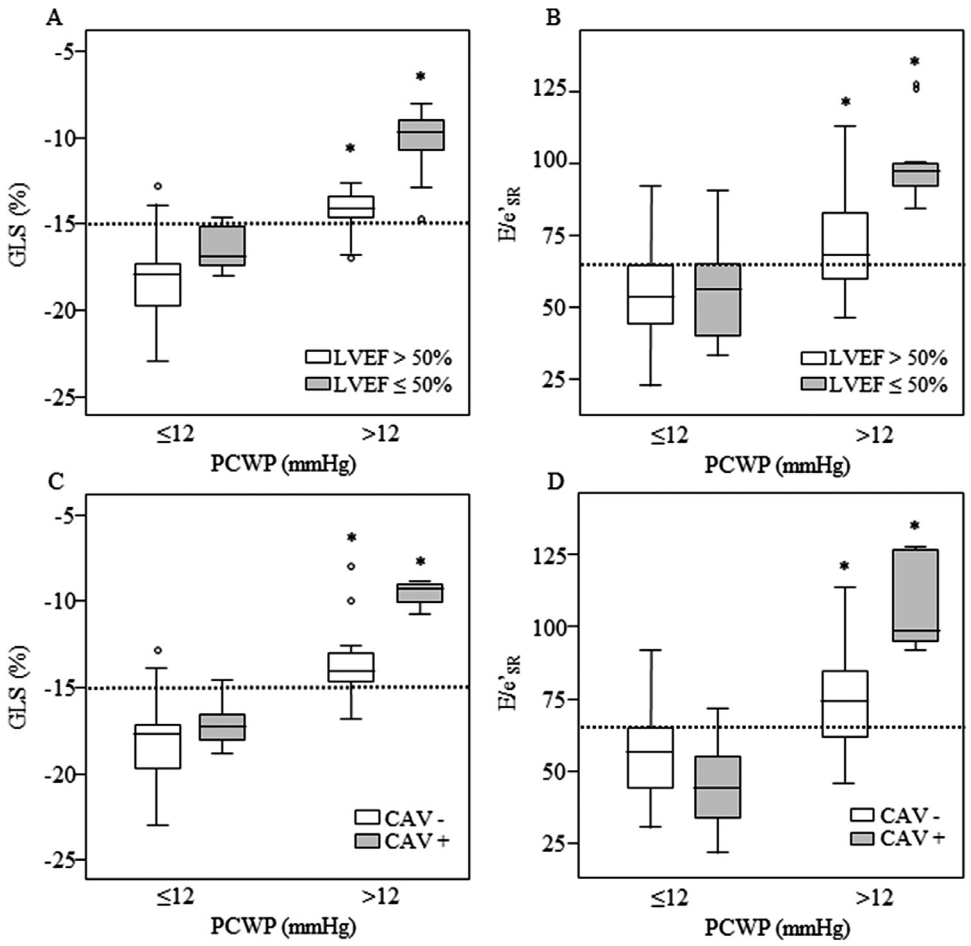

Figure 3. Box plots showing the discrimination between patients with normal and elevated PCWP by GLS and E/e'$_{SR}$. In relation to (*A*) EF and (*B*) cardiac allograft vasculopathy (CAV). Dotted lines indicate the cut-offs: -15% for GLS, 63 for E/e'$_{SR}$. (*: p<0.0001 vs PCWP ≤12 mm Hg).

pretransplantation measurements obtained from native failing hearts. In the pre-transplantation subgroup PWCP was 23.3 ± 8.9 mm Hg (range: 12 to 44), E/e' was 14.8 ± 2.9 (range: 10.3 to 21.0). No patient had PCWP below 12 mm Hg.

We found that in studies showing grade ≥2 rejection (n = 11) mean PCWP was significantly elevated (16.5 ± 4.7 mm Hg) as compared with those with grade 1 or no rejection (11.0 ± 4.7 mm Hg, p <0.001). Grade ≥2 rejection was observed in 8 out of 27 patients with PCWP >12 mm Hg in contrast to only 3 of 47 patients with PCWP ≤12 mm Hg (p <0.001). To test the ability of tracking changes in PCWP, we evaluated 24 patients having repeated studies at different time points (median 6 months, range 1 to 24 months apart) with different hemodynamic conditions: Importantly, the change in PCWP was well tracked by GLS and E/e'$_{SR}$ measurements (GLS; r = 0.77 and E/e'$_{SR;}$ r = 0.74, p <0.0001 for both) (Figure 4) As observed, changes in PCWP were classified correctly in 20 of 24 patients (overall accuracy 83%) by GLS, and in 19 of 24 patients (overall accuracy 79%) by E/e'$_{SR}$. However, changes in E/e' did not correlate with changes in PCWP in OHT recipients. Intra- and inter-observer intraclass correlation coefficients, performed in 10 patients for SR measurements, were 0.944 (95% CI: 0.842 to 0.986) and 0.911 (95% CI: 0.769 - 0.987), respectively.

Discussion

The main findings of the present study can be summarized as follows: S_{SR}, e'$_{SR}$, E/e'$_{SR}$, and GLS are worsening in OHT patients with elevated LVFP and have clearly stronger diagnostic performance than traditional parameters of diastolic dysfunction, even though E/e' performs well to predict elevated LVFP in native failing hearts of the same patients. Moreover, E/e'$_{SR}$ and GLS are associated with LVFP independently from LV EF, EDVi and allograft vasculopathy. Finally, changes in GLS and E/e'$_{SR}$ track accurately changes in PCWP.

Traditional parameters to evaluate LVFP have limitations depending on the studied population as underlined by the recommendation paper and recently, by the Euro-Filling study.[13,14] Although, E/e' is the most widely acknowledged noninvasive marker of LVFP, OHT recipients represent a particular challenge.[15,16] LA volumes almost always tend to be larger than normal limits, denervation of the allograft leads to relatively high heart rates with frequent fusion of mitral inflow waves, mitral annular velocities tend to be low in OHT recipients, as observed in this study. Even

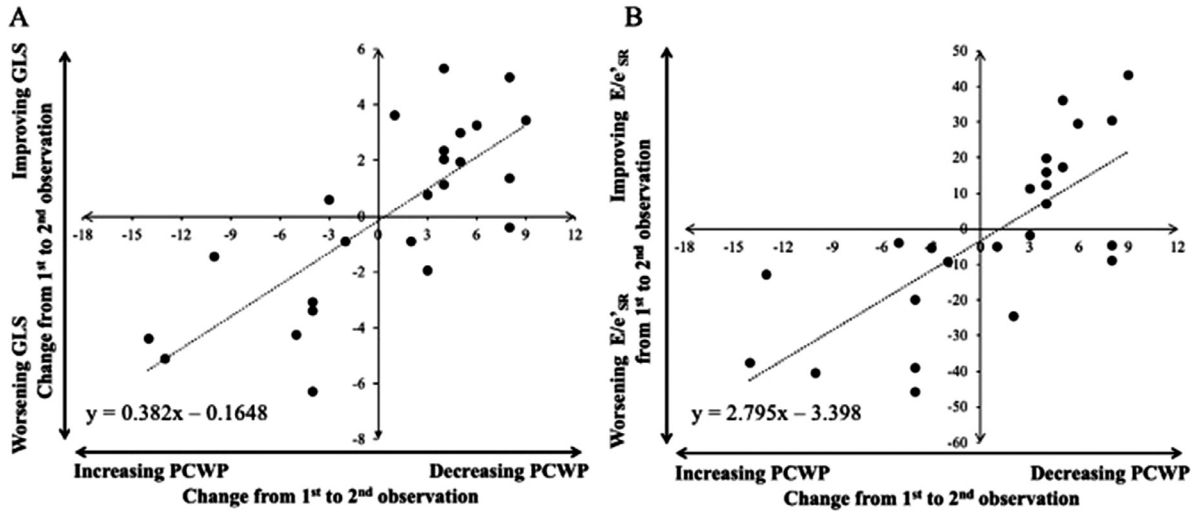

Figure 4. Changes in PCWP in relation to changes in strain and SR measurements. (*A*) Delta PWCP versus delta GLS and (*B*) Delta PWCP versus delta E/e'$_{SR}$.

though tissue Doppler parameters may recover over time in some OHT recipients,[17] they remain angle and load dependent and are subjected to translational motion of the transplanted heart. Both LA morphology and function are altered in OHT recipients by anastomoses and contribution of different proportions of donor and recipient tissue. This causes inability of LA to develop atrial forward force strong enough for atrial transport. Pulmonary flow pattern was shown to be altered in the allograft by the contraction of remnant recipient tissue.[3] TR occurs in association with both mild residual pulmonary vascular resistance of the recipient and leaflet injury by repeated endomyocardial biopsies. In addition, right ventricular mechanics are altered frequently after surgery possibly affecting TR jet velocity. Taken together, these characteristics limit the value of traditional markers of LVFP in OHT recipients.

Speckle tracking based deformation imaging is largely angle independent, not affected by translation, less load and heart rate dependent than Doppler based parameters.[18] In addition, particularly GLS is a highly reproducible and robust parameter.[19,20] Previous studies have shown that LV GLS and global SR are very sensitive to capture early myocardial damage due to allograft rejection or vasculopathy.[9,20,21]

Deformation parameters have been tested in previous studies as a means to complement traditional parameters for the assessment of LVFP. In the study of Kimura et al.[22] E/e'$_{SR}$ was more accurate than E/e' in intensive care unit patients to estimate PCWP. Wang et al.[6] showed that the ratio of E/SR$_{isovolumetric\ relaxation}$ could be an accurate predictor of elevated LVFP in patients who are in the gray zone by E/e' (8 to 15), particularly in patients with normal EF and in those with regional dysfunction. Our findings are important to fill a gap in evidence, by extending these findings to the prediction of elevated LVFP in OHT recipients. We found LV GLS and E/e'$_{SR}$ as the best correlates of PCWP and independent predictors of elevated LVFP in various echocardiographic indices including traditional parameters of diastolic function.

LV longitudinal deformation, although not an index of LV diastolic function per se, is a sensitive marker of myocardial performance. The strong association of GLS with LVFP might not sound confusing given the tight coupling of systolic and diastolic functions. Patients with heart failure and preserved EF (HFpEF) usually have depressed LV GLS and abnormal LVFP that varies in parallel with GLS. It is plausible to consider OHT recipients with preserved EF in the HFpEF category, given the well known attenuated GLS as compared with healthy controls at all times after transplantation.[11,23] Subclinical structural and functional alterations occur easily during the natural survival of the allograft, as a result of perioperative damage, undetected smouldering rejections, rejection-resolution episodes, infections, sympathetic denervation, immunosuppresive agents, micro and macrovascular allograft vasculopathy,[9,24–26] leading to increased LV stiffness and elevated LVFP. In addition, increasing LVFP causes increased wall tension, further depressing myocardial systolic deformation.[27] LV global longitudinal diastolic SR during the isovolumic relaxation period has also been shown to have a significant association with the time constant of LV relaxation.[6,8] In several disease states including HFpEF and ischemic cardiomyopathy, SR parameters in conjunction with mitral E velocity, presented encouraging results to estimate LVFP in accordance with our findings.[5,7,8,28] In addition, speckle tracking derived GLS and global SR have the advantage to encompass all the myocardial segments in contrast to sampling only the lateral and medial mitral annular sites that limits the use of tissue Doppler assessment in patients with ischemic cardiomyopathy and regional dysfunction.[6,20] This explains why the relation of GLS and E/e'$_{SR}$ remained strong with PCWP in the subgroup of patients with significant allograft vasculopathy in our study.

GLS and E/e'$_{SR}$ also tracked changes in PCWP which is particularly important in OHT recipients for 2 reasons: (1) Variation of myocardial performance in apparently stable OHT patients is higher than in healthy subjects, (2) GLS and SR measurements are often lower than accepted normal

cut-offs despite preserved EF.[9,23,29] Therefore, the ability of noninvasive parameters to track hemodynamic changes within a subject is highly desirable. Thus a baseline fingerprint study with GLS and SR imaging in a rejection-free, stabilized period around 3 to 6 months after transplantation, could be very useful to detect and track future hemodynamic changes and graft deterioration. However, we are cautious to underline that our results are not derived from acute hemodynamic alterations.

Our study has important clinical implications. Demonstration of elevated LVFP by noninvasive parameters is an important diagnostic target in OHT recipients given the alterations in myocardial function with several injurious factors, as mentioned above, despite silent or nonspecific symptoms. The recommendation paper underlines the importance and the prognostic value of evaluating diastolic dysfunction in OHT recipients despite lack of known salient confident parameters.[13] The present study provides evidence in favor of new, reproducible and clinically feasible noninvasive parameters in that regard. Of note, although serial assessment of LVFP by GLS and SR can be helpful for the detection of rejection, one should remember that it is unrealistic to expect prediction of rejection specifically by these parameters, as multiple other causes including donor age, pretransplant ischemic time, allograft vasculopathy, and perpetuating inflammation can alter systolic and diastolic function of the allograft.[30] Because of this continuous remodelling process in transplanted hearts strain cut-offs to determine rejection are time dependent. Serial follow-up with strain parameters, is reasonable to detect rejection, noninvasively instead of relying on single cut-offs.[9] Therefore the ability of GLS and E/e'SR to track changes in PWCP is of clinical importance. It seems clear that the use of GLS and SR are sensitive for monitoring allograft function by serial follow-up and should be incorporated into the echocardiographic examination of OHT recipients.

Some limitations are worth underlining. We did not assess pulmonary vein velocities because anastomoses at the level of pulmonary vein ostia alter pulmonary flow. We did not homogenize our population in terms of time from transplantation. We did not use high fidelity pressure catheters. Our results do not derive conclusions for LV relaxation properties of the allograft as we did not perform dedicated experimental measurements to assess the direct association of deformation parameters with tau. Also our findings need to be tested and validated for even higher PCWP values in other series. Inter-vendor variability in deformation measurements should be taken into account.

In conclusion, whereas traditional echocardiographic indices of LVFP perform poorly in OHT recipients, GLS and E/e'_{SR} provide reliable means of LVFP irrespective of LV EF and allograft vasculopathy and also track reasonably well the changes in LVFP.

Authors contribution

All authors have read and approved the manuscript. All authors have access to the full dataset. The authors have no conflict of interest related to this manuscript.

Dr Colak took part in reviewing echocardiographic data and drafting the manuscript. Dr Muderrisoglu contributed to the design and revision of the manuscript, Dr Eroglu and Dr Pirat took part in reviewing echocardiographic data, Dr Aydınalp took part in reviewing catheter data, Dr Sezgin took part in final approval and Dr Sade took part in the design, analysis, interpretation and drafting of the manuscript.

Disclosures

None of the authors have anything to disclose.

Declaration of Interest

The authors declare that they have no known competing financial interests or personal relations that could have appeared to influence the work reported in this study.

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