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Left atrial strain: a new parameter for assessment of left ventricular filling pressure

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Abstract In order to obtain accurate diagnosis, treatment and prognostication in many cardiac conditions, there is a need for assessment of left ventricular (LV) filling pressure. While systole depends on ejection function of LV, diastole and its disturbances influence filling function and pressures. The commonest condition that represents the latter is heart failure with preserved ejection fraction in which LV ejection is maintained, but diastole is disturbed and hence filling pressures are raised. Significant diastolic dysfunction results in raised LV end-diastolic pressure, mean left atrial (LA) pressure and pulmonary capillary wedge pressure, all referred to as LV filling pressures. Left and right heart catheterization has traditionally been used as the gold standard investigation for assessing these pressures. More recently, Doppler echocardiography has taken over such application because of its noninvasive nature and for being patient friendly. A number of indices are used to achieve accurate assessment of filling pressures including: LV pulsed-wave filling velocities (*E*/*A* ratio, *E* wave deceleration time), pulmonary venous flow (*S* wave and *D* wave), tissue Doppler imaging (E' wave and E/E' ratio) and LA volume index. LA longitudinal strain derived from speckle tracking echocardiography (STE) is also sensitive in estimating intracavitary pressures. It is angle-independent, thus overcomes Doppler limitations and provides highly reproducible measures of LA deformation. This review examines the application of various Doppler echocardiographic techniques in assessing LV filling pressures, in particular the emerging role of STE in assessing LA pressures in various conditions, e.g., HF, arterial hypertension and atrial fibrillation.

Keywords Echocardiography · Left ventricular filling pressure · Left atrium · Speckle tracking · Strain

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Background

Evaluation of left ventricular (LV) filling pressure is an essential step for accurate diagnosis, therapy and prognosis in patients with various cardiac diseases, particularly those with heart failure (HF). In recent years, increasing attention has been given to such patients particularly those with preserved ejection fraction (EF) (typically >50 %) and maintained LV cavity size, but still diagnosed with HF based on typical signs and symptoms in association with a diastolic dysfunction and a relevant structural heart disease (LV hypertrophy, left atrial enlargement) [1]. This subset of patients suffers from elevated morbidity and mortality [2, 3], not less than those with reduced LV EF [4].

Several studies have demonstrated that the incidence of HF with preserved ejection fraction (HFpEF) is increasing in the population and a significant proportion of hospitalized patients shows this variant of HF. They are mainly older women with a history of arterial hypertension [5], but obesity, CAD, diabetes mellitus, atrial fibrillation and dyslipidemia are also frequently met risk factors [6]. It has been suggested that these subjects might represent the largest cohort of patients with a cardiovascular disorder that is of substantial public health impact, who have not been

systematically studied [7]. The pathophysiological mechanisms that convert these patients from asymptomatic to symptomatic HF remain undefined [8]. Furthermore, patients who show a LV systolic dysfunction frequently have some elements of diastolic dysfunction as well [9], thus adding to the relevance of accurately defining diastolic disturbances that are characteristics for HF in its two entities.

The diastolic dysfunction includes altered LV relaxation, reduced compliance with or without raised filling pressures [10]. Regardless of the etiology, an abnormality in diastolic function can result in an increase in LV end-diastolic pressure (LVEDP), mean left atrial (LA) pressure and pulmonary capillary wedge pressure (PCWP). All these pressures are commonly referred to as LV filling pressures. During diastole, there is a direct continuity between the pulmonary capillary bed, the pulmonary veins, the LA and the LV. Therefore, a pressure rise in the LV leads to an increase in LA and pulmonary capillary pressures and eventually pulmonary venous congestion and HF symptoms. Consequently, accurate assessment of diastolic function can be used to estimate LV filling pressure [11].

Invasive estimation of LV filling pressure

The invasive left and right heart catheterization has been considered the gold standard investigation for confirming or refuting the presence of high LV filling pressures with a LVEDP greater than 16 mmHg or a mean PCWP greater than 12 mmHg suggestive of diastolic dysfunction [12]. In particular, PCWP turned out to be directly associated with functional capacity and prognosis in patients with HF [13–15]. However, the cardiac catheterization is a technique that is not without risks, often poorly accessible and utilizable especially in the acute setting and outpatients services. Therefore, it would be desirable to restrict its use to already diagnosed patients who developed unexplained symptoms or signs.

Noninvasive estimation of LV filling pressure

Currently, echocardiography has been shown the only noninvasive investigation that allows accurate estimation of LV filling pressures as well as evaluating LV and LA pathophysiological changes that associate diastolic dysfunction. Several echocardiographic indices have been proposed for this purpose (Table 1).

Pulsed-wave Doppler mitral flow velocity

The most widespread echocardiographic technique for assessment of LV filling pressure is the pulsed-wave Doppler of mitral flow velocities. It includes the following variables: peak early diastolic flow velocity (*E* wave, referred to as rapid filling), peak late diastolic flow velocity (*A* wave, related to atrial contraction), *E* wave deceleration time (DT) and the total duration of the *A* wave (A_{dur}). Normally with physiological filling pressures, a rapidly accelerating *E* wave is followed by a significantly smaller *A* wave with an *E*/*A* ratio greater than 1 and a DT between 160 and 240 ms. However, with normal aging, it is common for the *E* wave to be reduced due to a slowing of LV relaxation, resulting in equalization of early and late diastolic filling velocities in the sixth decade of life [16]. In patients with diastolic dysfunction, three different abnormal filling patterns are recognized: *impaired relaxation* (grade 1), characterized by a low *E* wave velocity and a high *A* wave velocity, causing *E*/*A* ratio to fall below 1 and *E* wave deceleration to prolong (>240 ms); *pseudonormal pattern* (grade 2), is associated with a normal appearance of the transmitral flow (*E*/*A* between 0.8 and 1.5, with normalized DT); *restrictive pattern* (grade 3), with marked elevation of *E* wave, low *A* wave, *E*/*A* usually greater than 2 and very shortened DT (<160 ms). LV filling is normally affected by preload, so the Valsalva maneuver represents an additional supplement for testing them: In patients with grade 2 pattern, the maneuver lowers LA pressure and unmasks impaired relaxation with *E*/*A* ratio <1. In subjects with early, still reversible, restrictive pattern, Valsalva maneuver can revert filling pattern to a pseudonormal or even impaired relaxation one. It should be remembered that in patients not in sinus rhythm, the *A* wave will be absent. Other conditions in which the *A* wave is not seen are the summation filling where a fusion between *E* and *A* waves occurs, which makes accurate identification of the predominant filling wave difficult. An example of the latter is patients with sinus tachycardia, prolonged PR interval and intraventricular dyssynchrony. It has been shown that restrictive LV filling pattern represents a powerful predictor of mortality, independent from LVEF and age, in patients with HF [2]. Moreover, studies have reported a strong correlation between DT and LV filling pressure, comparing Doppler mitral flow velocity and PCWP obtained by cardiac catheterization [17, 18], but only in patients with reduced EF [19].

Pulmonary venous flow

Another parameter, used to integrate LV filling velocities in assessing severity of diastolic dysfunction, is the pulmonary venous flow. Four main measurements are used: peak systolic velocity (*S*), peak diastolic velocity (*D*), peak atrial reversal flow velocity (PVa) and PVa duration (A_{dur}). Normally, pulmonary venous flow goes from the PV to the LA in systolic and early diastole, with a small component

Table 1 Echocardiographic parameters in the evaluation of LV filling pressure

Method	Parameter	Cutoff	Advantages	Disadvantages
PW Doppler mitral inflow	*E*/*A* ratio	Normal > 1 *Impaired relaxation* < 0.8 *Pseudonormal* 0.8–1.5 *Restrictive* > 2	Easy to obtain and interpret in most cases Strong predictor of mortality in HF, independent from EF and age.	All parameters are preload dependent Strong correlation demonstrated between DT and PCWP only in patients with reduced EF Values can be pseudonormal, requesting Valsalva maneuver.
	DT	Normal 160–240 ms *Impaired relaxation* ≥ 240 ms *Pseudonormal* 160–240 ms *Restrictive* < 160 ms		
Pulmonary venous flow	*S*/*D* ratio	Normal ≥ 1 Altered < 1	Useful to assess pseudonormal transmitral pattern.	Preload dependence Non easy to obtain, in dependence of patient habitus and operator dependence Similar *S*/*D* ratio in mitral regurgitation and AF.
Tissue Doppler imaging (TDI)	Septal E'	Normal ≥ 8 cm/s Altered < 8 cm/s	Preload independence Demonstrated correlation between E' and heart catheterization Tau.	Angle dependence Different values Affected not only by filling pressures but also by myocardial relaxation.
	Lateral E'	Normal ≥ 10 cm/s Altered < 10 cm/s		
	Averaged E/E' ratio	Normal < 8 Altered > 13	Strong correlation with diastolic dysfunction in several diseases.	Presence of a gray zone that requests integration with other parameters Low accuracy in advanced HF.
Left atrium evaluation	Volume index	Normal < 34 ml/m^2 Altered ≥ 34 ml/m^2	Reflects cumulative effects of LV filling pressure.	Elevated volume index in several other conditions: AF, atrial flutter, mitral valve diseases, high-output states like anemia.
Speckle tracking echocardiography (STE)	Left atrial longitudinal strain	Normal ≥ 18 % Altered < 18 %	Rapid and easy to perform Angle independence Less affected by reverberations, side lobes, dropout artifact Possibility of off-line processing Qualitative assessment of LA function.	Frame rate dependence Errors in epicardial/endocardial border tracing Absence of a dedicated analysis software.

AF atrial fibrillation, *DT* deceleration time, *EF* ejection fraction, *HF* heart failure, *LA* left atrial, *PCWP* pulmonary capillary wedge pressure, *PW* pulsed wave

reversing back during atrial contraction, in late diastole. When the LV and, consequently, LA pressure increase, most of the anterograde flow occurs in diastole with a reduction in *S* velocity, which becomes smaller than *D* velocity, thus reversing the *S*/*D* ratio. Furthermore, it has been demonstrated that a PVa greater than 35 cm/s with a duration longer than A_{dur} by 30 ms or more, is predictive of raised LVEDP greater than 15 mmHg [20, 21]. It must be mentioned that pulmonary venous flow recordings are not so easy to obtain in all patients though, because of habitus and operator experience. It may be also affected by other factors including significant mitral regurgitation and atrial fibrillation.

Tissue Doppler imaging

Tissue Doppler imaging (TDI) has been widely used as an accurate tool for the assessment of LV diastolic function noninvasively. It is obtained from the apical four- or two-chamber view by placing the pulsed-wave sample at the junction between LV wall and the mitral annulus, in septal (or medial) and lateral position. Three waves are displayed in each cardiac cycle: systolic wave (S'), early diastolic wave (E') and atrial contraction wave, at the end of diastole (A'). Normally, the lateral velocity is higher than the septal one, but the two values can be averaged. E' represents the main TDI parameter, and it is considered normal if the septal one is

≥8 cm/s and the lateral one ≥10 cm/s. If diastolic function is normal, E' is higher than A' and their ratio, like the transmitral flow velocities, decreases in mild dysfunction ($E'/A' < 1$), but E' continues to decrease with worsening segmental myocardial function. It must be remembered that in patients with normal filling pressures, E' determines transmitral flow pattern, and in patients with raised filling pressures, the E' wave remains small with respect to the spectral velocity [22]. E' has been shown to correlate with Tau, an invasive index of myocardial relaxation which is obtained from heart catheterization [23].

In addition to absolute measurement of E', the E/E' ratio may be used to predict LV filling pressures and has been shown to correlate with PCWP in a large variety of cardiac conditions [24–29]. In normal LV relaxation and normal LV filling pressure, both E and E' are elevated. With impaired relaxation and normal LV pressures, both E and E' are depressed, and in subjects with impaired relaxation and elevated filling pressures, E is high but E' is reduced. An E/E' ratio less than 8 is considered normal, while a ratio of 13 or greater represents an evidence of diastolic dysfunction. Values between 8 and 13 represent a gray zone in which more investigations are necessary, including mitral flow E/A ratio and its reduction with Valsalva maneuver, DT, pulmonary venous reversal flow duration compared with spectral A wave duration, as described above [30]. Dini et al. have proposed a model for noninvasive estimation of LV filling pressure using all these parameters, in patients with LV dysfunction and a wide range of EF. Averaged E/E' ratio ≥13 or E wave deceleration time <150 ms was closely associated with elevated LV filling pressures. Alternatively, prediction of PCWP > 15 mmHg requires E/E' ratio between 8 and 15, LA volume index (another parameter that reflects LV filling pressures, as explained later) >40 ml/m^2 or difference in duration of pulmonary venous and mitral flow at atrial contraction >30 ms and estimated pulmonary artery systolic pressure >35 mmHg [31]. Nevertheless, the E/E' ratio accuracy is still uncertain in patients with advanced HF and extensive LV remodeling, which frequently are not included in the studies. In fact, it has been demonstrated that, in advance decompensated HF, this parameter is less reliable in predicting filling pressures than previously reported in studies, especially with very low cardiac output and elevated LV volumes. Moreover, it is strongly influenced by the presence of cardiac resynchronization therapy or other treatments, like inotropic medications which may have independent effects on mitral annular motion [32]. It is well known how several conditions may distort the velocity of the E' value and of the E/E' ratio: mitral annular calcifications, surgical rings, prosthetic valves, several mitral regurgitation, atrioventricular or intraventricular conduction delay.

LA volume index

Transthoracic echocardiogram is the gold standard approach for assessing LA size. LA volume, and in particular LA volume index (LA volume indexed to body surface area), is a more robust marker of cardiovascular events over and above LA area or diameter [33]. The American Society of Echocardiography recommends the measurement of LA volumes by ellipsoid model and Simpson's method in four- and two-chamber apical views [34]. A LA volume index >34 mL/m^2 is considered abnormal and represents an independent predictor of mortality, HF, atrial fibrillation and ischemic stroke [35].

LA volume index has been shown also to correlate with the other parameters of diastolic function [36]. However, it must be remembered that while E/E' ratio is an instant measurement of LV filling pressure, LA volume index reflects the cumulative effect of the filling pressures over time. Therefore, since LA enlargement is seen in several conditions, even in the absence of ventricular disease (atrial flutter or fibrillation, mitral valve diseases, anemia and other high-output states, etc.), it has a low specificity. Therefore, it should always be considered in conjunction with patient's clinical status, other chambers' volume and Doppler parameters of LV relaxation [30].

Speckle tracking echocardiography: left atrial strain

Speckle tracking echocardiography (STE) is a new non-Doppler-based method for the objective quantification of myocardial deformation from standard bidimensional data acquisitions, which presents significant feasibility and reproducibility [37–40]. In contrast to Doppler-derived indexes previously analyzed, STE has several advantages being angle-independent and less affected by reverberations, side lobes and dropout artifacts. Although STE was originally developed to study ventricular function, it has been recently used to evaluate atrial chambers function as well: The atrial longitudinal strain represents an excellent parameter, useful for analysis of LA function in several conditions.

STE measurements

Two-dimensional strain imaging is an echocardiographic technique that utilizes standard B-mode images for speckle tracking analysis. The speckle pattern (acoustic backscatter generated by the reflected ultrasound beam) is followed frame-by-frame, identifying the best matching area by a statistical approach. The displacement of this pattern is considered to follow myocardial movement, and every

change between speckles is evaluated as a myocardial deformation [41].

For speckle tracking analysis, LA apical four- and two-chamber view images are obtained using conventional two-dimensional gray scale echocardiography, with an emphasis on the distinction between myocardium and extracardiac structures, during a brief breath hold and with a stable ECG recording. Three consecutive heart cycles are recorded and averaged. The recommended frame rate is set between 60 and 80 frames per second [37]. Recordings are processed using an acoustic-tracking software (Echo Pac, GE, USA), allowing off-line semi-automated analysis of speckle-based strain.

The LA endocardium surface is manually traced in both four- and two-chamber views by a point-and-click approach. An epicardial surface tracing is automatically generated by the system, and a region of interest (ROI) is identified. To trace the ROI in the discontinuities of LA wall, corresponding to pulmonary veins and LA appendage, the direction of LA endocardial and epicardial surfaces at the junction with these structures is extrapolated. The ROI can be manually adjusted in width and shape; then, the software divides it into six segments, and the resulting tracking quality for each segment is automatically scored as either acceptable or nonacceptable, with the possibility of further additional corrections. Segments, in which no adequate images quality can be obtained, are rejected by the software and automatically excluded from the analysis. In patients with adequate image quality, a total of 12 segments are then analyzed [37], and for each of these segments, the software generates the longitudinal strain curves together with a mean curve of all segments that reflects the pathophysiology of LA function.

Since the LA fills and stretches during the reservoir phase, the atrial strain increases, reaching a positive peak at the end of atrial filling, before the opening of the mitral valve. After opening of the valve, LA empties quickly and its volume falls so the strain initially decreases, up to a plateau corresponding to the phase of diastasis; then, it is followed by a second positive peak, less than the first, which corresponds to the period preceding atrial contraction (present only if patients are in sinus rhythm) and finally by a negative peak after the atrial contraction [37].

Peak atrial longitudinal strain (PALS) is measured at the end of reservoir phase, and peak atrial contraction strain (PACS) is measured just before the start of active atrial contractile phase (Fig. 1); the values obtained in four- and two-chamber views are then averaged (four- and two-chamber average PALS and PACS, respectively).

LA contraction strain index (CSI), representing in percentual values the contribution of the LA active contraction to the LV filling phase, can be calculated as: $(\text{global PACS}/\text{global PALS}) \times 100$. The time to peak longitudinal strain (TPLS) is also measured as the average of all 12 segments (global TPLS) and by separately averaging values observed in the two apical views (four- and two-chamber average TPLS) [42].

In the current ASE/EAE consensus [43], two techniques have been proposed to quantify atrial deformation by STE, regarding the measurement of peak atrial strain. They differ only by choice of frame from which processing software starts: The first takes the QRS onset as the reference point and measures the positive peak atrial longitudinal strain, corresponding to atrial reservoir. The second uses the *P* wave as the reference point, thus enabling the measurement of a first negative peak atrial longitudinal strain, corresponding to atrial systole, and a second positive peak atrial strain, corresponding to LA conduit function, and their sum (Fig. 2).

Estimation of LV filling pressures

LA longitudinal strain has been proposed as an alternative approach for LV filling pressure assessment (Fig. 3). PALS has been shown to have excellent sensitivity and specificity in predicting a PCWP of 18 mmHg or above in patients with HF, despite having no relationship between mean E/E' ratio and PCWP. In particular, a PALS cutoff value lower than 15.1 % results in the highest diagnostic accuracy [44]. Recently, a new study [45] explored the correlation between LA longitudinal function by STE, Doppler E/E' ratio and invasive measurements of LVEDP in patients, stratified for different values of EF. Between all echocardiographic parameters analyzed, global PALS, with a cut-off value of 18.0 %, had the greatest diagnostic accuracy in predicting a LVEDP above 12 mmHg. In subjects with preserved (>55 %) or mildly reduced (45–54 %) LV EF, mean E/E' ratio had good correlation with LVEDP, while in those with moderate (30–44 %) and severe (<30 %) LV EF, E/E' ratio poorly correlated with LV filling pressure. In contrast, global PALS showed a strong correlation in all four groups of patients, even with low EF. The potential mechanism for the inverse relationship between global PALS value and high LV filling pressure could be explained by the principle that LVEDP is the afterload of LA function; hence, LVEDP is elevated, the LA could be mechanically stressed, and its reservoir function reduced, hence inducing a progressive LA dilation [46–48].

Furthermore, it has been demonstrated that LA strain at the end of ventricular systole is closely related not only to LVEDP, but also to NT-pro-BNP plasma levels [49]: Natriuretic peptides and in particular BNP (brain or B-type natriuretic peptide) and his aminoterminal prohormone, are produced by atrial and ventricular cardiomyocytes in response to mechanical stress. Plasma concentration of BNP is well correlated with LVEDP in patients with LV

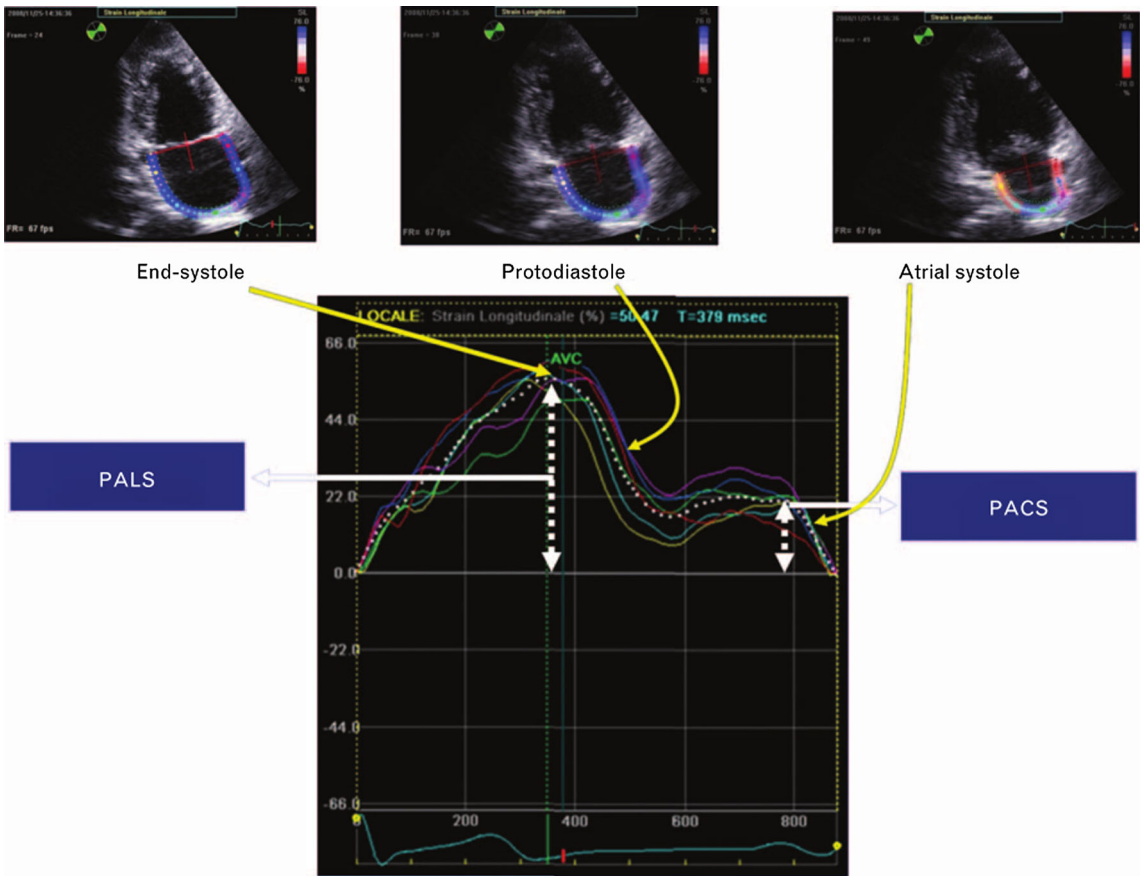

Fig. 1 Measurement of peak atrial longitudinal strain (PALS) at the end of reservoir phase and peak atrial contraction strain (PACS) before the start of atrial systole. The *dashed curve* is representative of the average atrial longitudinal strain during the cardiac cycle [65]

hypertrophy and systolic or diastolic HF [50, 51], so a combination of this parameter and LA strain could be used to increase the accuracy of noninvasive diagnostics of patients with HF.

LA stiffness index

The presence of atrial fibrosis [42, 52–54] and other ultrastructural alterations, often secondary to a chronic increase in LV filling pressure, likely influences the LA distensibility, increasing LA stiffness, and this finally could represent an anatomical substrate for the development of atrial arrhythmias [55] and a major risk of HF [56, 57]. Stiffness is defined as "the force required to displace a passive spring a unit length." Physiologically, it is the change in pressure required to increase the volume of a passive container a unit amount. The units of strain in physiology are mmHg/mL [58]. The ratio of invasively measured PCWP and left atrial systolic strain is used to estimate LA stiffness. Alternatively, E/E' ratio (related to LVEDP) can be used instead of PCWP in conjunction with atrial strain as a noninvasive measure of the same parameter. LA stiffness index is elevated in patients with diastolic dysfunction and is highest in subjects with HF [59].

Clinical implication of LA longitudinal strain

In the context of HF, a reduced atrial strain has been demonstrated to differentiate between subjects with diastolic dysfunction from those who already suffer from HFpEF [59]. In addition, it has been observed that, in patients with new-onset symptoms of HF, LA dysfunction is frequently present which could represent the *primum movens* of the clinical manifestations of HFpEF. Therefore, evaluation of LA strain in patients with new-onset dyspnea may be extremely useful for a correct diagnosis of HF and for the demonstration of a possible reversibility of LA dysfunction and symptoms with therapy [60]. In addition, LA strain has a strong negative correlation with NYHA class [61, 62].

Subjects who undergo heart transplant (HTX) are strictly monitored with periodically invasive measurements of PCWP. Recently, in pediatric HTX patients,

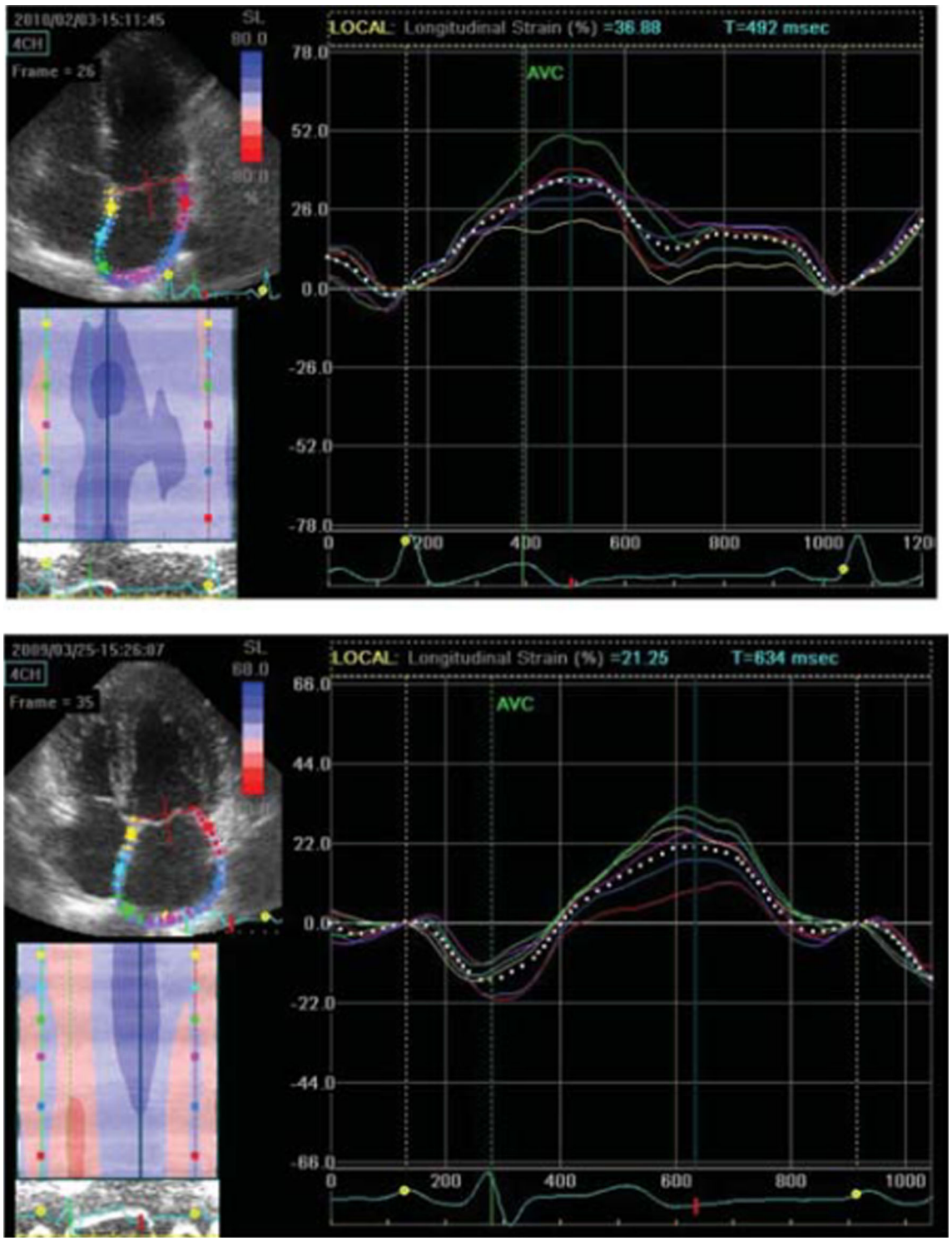

Fig. 2 Two different techniques and reference points (QRS, *above*, and *P* wave, *below*) proposed in ASE/EAE consensus for the measurement of global atrial deformation by STE [43]

PALS resulted to be a great noninvasive surrogate of cardiac catheterization, better than E/E' ratio, with a good reproducibility [63]. Moreover, it has been demonstrated how, in HTX with bicaval technique (in which the right atrium is whole donor tissue and LA is from both donor and recipient tissue), LA strain is reduced related to high PCWP and elevated LA volume [64].

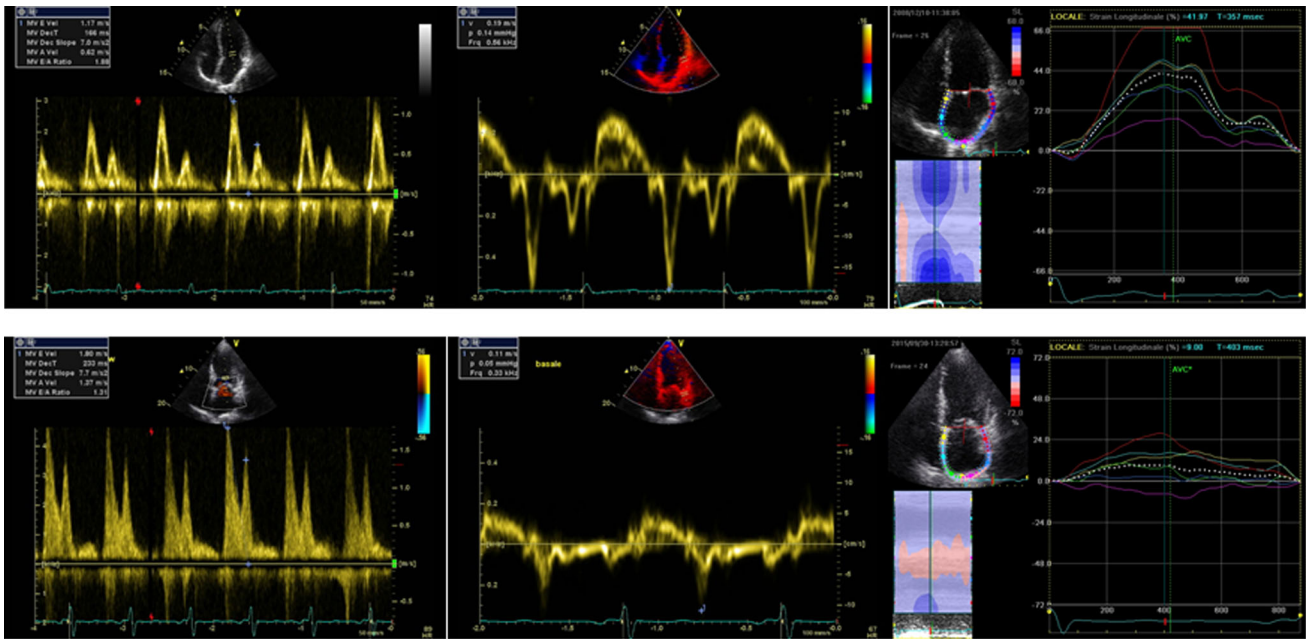

Fig. 3 Comparison between pulsed-wave Doppler of mitral flow velocities, tissue Doppler imaging (TDI) and left atrial longitudinal strain in a patient with normal LV filling pressures (invasively measured LVEDP = 9 mmHg, *above*) and in one with elevated LV filling pressure (LVEDP = 25 mmHg, *below*)

Studies have already shown early progressive reduction in LA strain in hypertensive patients [65] with diastolic dysfunction even before the appearance of LA enlargement and other structural alteration [66]. This adverse pattern is more evident in nondipper hypertensive patients (that is subjects in which daytime ambulatory systolic and diastolic blood pressure do not decrease by at least 10 % during the night) [67] and may reflect the raised LV pressure secondary to the systemic pressure overload that leads to increased LA stiffness during ventricular systole and to an abnormal LA–LV coupling [68], although other factors may also be involved. Similar aspect could be demonstrated in diabetic patients and even more in subjects with coexisting hypertension and diabetes [69–71]. Whether renin-angiotensin system (RAS) inhibitors have positive effect on LA myocardium in these hypertensive subjects is still unknown; however, patients treated with this class of drugs usually show improved atrial strain even after few months of therapy, whereas standard LA echocardiographic parameters (e.g., LA volumes, ejection fraction, active and passive emptying fraction) and other LV diastolic indices do not change [72].

LA strain has an important role also in predicting LV filling pressures and atrial dysfunction in ischemic and nonischemic cardiomyopathies. In patients with hypertrophic cardiomyopathy (HCM), LA is essential in maintaining an optimal cardiac output despite reduced LV compliance and elevated filling pressures, and undergoes important remodeling. LA function progressively decreases, and typical HF symptoms begin to appear. LA strain has been shown to be reduced in HCM, related to LV dysfunction [61, 73], and moreover allows to distinguish HCM from the nonpathological athlete's heart in which LA deformation parameters result similar to healthy controls [74]. Also in dilated cardiomyopathy (DCM), it is possible to find raised LVEDP due to diastolic and systolic LV dysfunction and consequently increased LA volume and pressures. PALS results to be particularly reduced in patients with idiopathic compared to ischemic DCM and closely associated with functional capacity during exercise [75]. LA strain is also an independent predictor of impaired LVEDP in these patients [76]. Lastly, LA myocardial dysfunction analysis by STE allows to differentiate restrictive cardiomyopathy (RCM), in which LA strain at septal segments is significantly reduced, from constrictive pericarditis, where the decreasing is placed mostly at superior and lateral segments [77]. In patients with cardiac amyloidosis, LA strain is impaired and, in particular, has resulted to be lower in those subjects with late gadolinium enhancement at atrial level showing a correlation with adverse atrial remodeling [78].

LVEDP results to be raised also in aortic stenosis (AS) where chronic afterload determines structural and functional LA changes. In these patients, all LA strain parameters are markedly reduced [79] and PACS is particularly affected by the severity of AS and LV diastolic function [80]. LA dysfunction is already observed in asymptomatic patients, and the reduction in strain is more pronounced in those who will develop symptoms during follow-up, besides normal LA volume [81]. Furthermore, patients who

undergo transcatheter aortic valve implantation (TAVI) show, in addition to a reduction in LA volume, a significant increase in PALS and PACS at 3-month follow-up, proving a reverse cavity remodeling [82].

Acute myocardial infarction (AMI) determines both systolic and diastolic LV dysfunction in surviving patients. In particular, the latter has been correlated with mortality and morbidity independently from systolic function [83], so in this setting, LA strain represents an important parameter, in addition to conventional ones, to detect subjects who are more likely to develop chronic HF. PALS decreases with reducing systolic and diastolic function in patients with AMI with ST elevation (STEMI) treating with percutaneous coronary intervention [84] and tends to increase in patients who undertake cardiac rehabilitation [85]. Also in AMI with non-ST segment elevation (NSTEMI), LA strain has demonstrated an essential prognostic role, being associated with the outcome in acute setting [86] and representing a valuable predictor of cardiovascular events over conventional echocardiographic parameters during follow-up [87].

Limitations and future perspectives

The measurement of LA strain, although feasible in most cases, needs adequate apical views and operator skills. A dedicated software for strain analysis has not been released yet, and currently, the software for LV is applied to the study of LA pattern strain. Moreover, different software uses various algorithms of analysis that could generate biases in comparisons between studies. The difficulty in obtaining the effective shape of LA in an accurate way, should as well as the possible contamination by signal components arising from structures placed around LA, also should be noticed. In patients with nonsinus rhythm is required an averaged measurement of almost five consecutive beats.

STE application is rapidly increasing. Development of dedicated and homogenous software and implementation of 3D-STE methods will allow an optimal and diffuse application of strain parameters. STE requires only a dedicated workstation: In addition to current use in diagnosis and prognostication, results of semi-automated analysis could be obtained and applied at the same time in acute settings like in hemodynamics laboratory and operating room, helping the decision-making process on the patient.

Conclusion

Noninvasive estimation of LV filling pressure is an essential step in the assessment of diastolic dysfunction in patients with HF, particularly in those with preserved EF. Most widespread echocardiographic indexes used for this purpose have limitations. The novel STE-derived parameter, left atrial longitudinal atrial strain, strongly correlates with invasive measurement of LV filling pressure and therefore could be easily utilized, in addition to the conventional parameters. Moreover, its ability of quantitative deformation measurements accurately reflects the degree of structural alterations, myocardial fibrosis and chamber stiffness.

Compliance with ethical standards

Conflict of interest Drs. Cameli, Mandoli, Loiacono, Dini, Henein and Mondillo have no conflicts of interest or financial ties to disclose.

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