International Journal of Multiphysics
Volume 18, No. 1, 2024
ISSN: 1750-9548

Role of Left Atrial Strain in Assessment of Mitral Stenosis

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Abstract:

Background: Mitral stenosis is a progressive valvular disorder characterized by narrowing of the mitral valve orifice, most commonly as a consequence of rheumatic heart disease. Echocardiography represents the principal noninvasive imaging modality for the diagnosis, grading, and follow-up of mitral stenosis. Two-dimensional echocardiography enables direct visualization of mitral leaflet morphology, commissural fusion, subvalvular involvement, calcification, and mitral valve area, while Doppler techniques provide essential hemodynamic information, including transmitral pressure gradients and pressure half-time. In addition, advanced echocardiographic techniques, particularly speckle-tracking echocardiography and velocity vector imaging, permit quantitative assessment of myocardial deformation and left atrial function. Left atrial reservoir, conduit, and contractile strain are frequently impaired in patients with mitral stenosis as a result of chronic pressure overload, atrial remodeling, fibrosis, and altered ventricular filling. These parameters may provide additional information regarding disease severity, functional status, procedural outcome, and risk of adverse cardiovascular events. Therefore, integrating conventional two-dimensional and Doppler echocardiography with deformation imaging may improve the comprehensive assessment of mitral stenosis and support clinical decision-making.

Keywords: Mitral stenosis; Two-dimensional echocardiography; Doppler echocardiography; Left atrial strain; Speckle-tracking echocardiography; Velocity vector imaging.

Introduction:

Echocardiography is the most powerful diagnostic tool in cardiology. Regarding its diagnostic accuracy, cost effectiveness, availability, and noninvasive nature it became the largest cardiovascular expense item in the Medicare budget. An integrated investigation of the MV includes an M-mode tracing, several two-dimensional views, a Doppler evaluation, and, if needed, TEE.

The posterior leaflet is somewhat less apparent than the anterior leaflet which is highly mobile and quite echogenic. By M-mode examination, an M-shaped pattern of MV motion is seen, reflecting first passive rapid filling and second atrial contraction, with near closure during diastasis, although blood may still pass from pulmonary veins to LV using the atrium as a conduit (1).

in the short-axis plane by two-dimensional imaging, the MV looks as an ovoid orifice. In the long-axis plane, it resembles clapping hands forming a stable coaptation plane in systole but moving freely in diastole. The MV annulus descends with the cardiac base to assist LA filling. Normal MV leaflets are thin (<2 mm), although somewhat thicker at points of chordal attachment the free margin (primary chordae) and leaflet body (secondary chordae). The papillary muscles can be detected in the short-axis view at 4 and 8 o'clock with highly variable anatomy. From the apical four-chamber view, posterior angulation (typically showing the coronary sinus) is necessary to show the papillary muscles and chordae. Normal chordae appear fragmented unless they are fused and thickened by fibrosis or calcification (1).

MS severity can be assessed on M-mode imaging by measuring the delay in diastolic closure (E-F slope). A normal value is greater than 60mm per second; a slope of less than 10mm per second indicates severe MS (1).

the leaflets dome into the ventricle throughout diastole by 2D echocardiography. In short axis, the MV orifice can be measured by direct planimetry, with an area

of less than 1 cm2 defining severe MS. chordal shortening, leaflet calcification, LA enlargement, LV under loading, and right-sided heart involvement (pulmonary hypertension) are the indirect signs of MS. although disease in patients with mild MS and aortic insufficiency progresses slightly faster but still it cannot be accurately predicted.

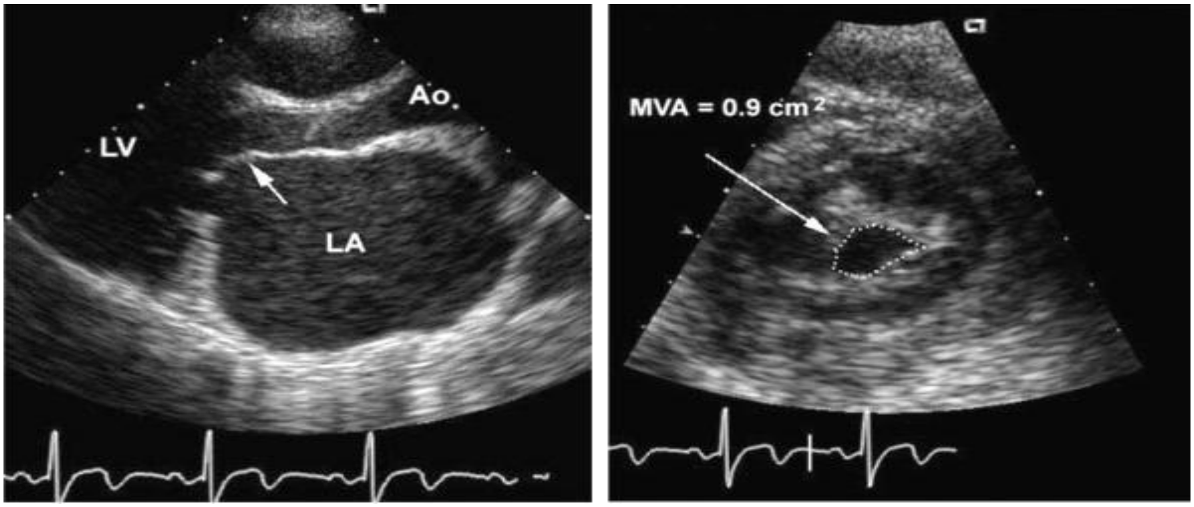


Figure (1): Parasternal long (left) and short (right) 2D echocardiographic views showing the characteristic findings in rheumatic mitral stenosis. Note the commissural fusion that results in doming of the leaflets in the long axis

view and in a decrease in the width of the mitral orifice in the short axis view. Ao: aorta, LA: LA, LV: left ventricle (From Otto CM: valvular heart disease. Elsevier, Philadelphia, 2004).
Doppler echocardiographic assessment of MS is done in the apical views. The mean gradient and the pressure half-time (PHT) should be calculated. PHT is calculated from the down slope of the E wave or the single diastolic wave in AF. The PHT in milliseconds is relatively robust against changes in cardiac output, heart rate, and the presence of mitral regurgitation; a useful empirical relationship exists with MV area (MVA, in cm2), given by MVA = 220/PHT (the Hatle formula) (2).

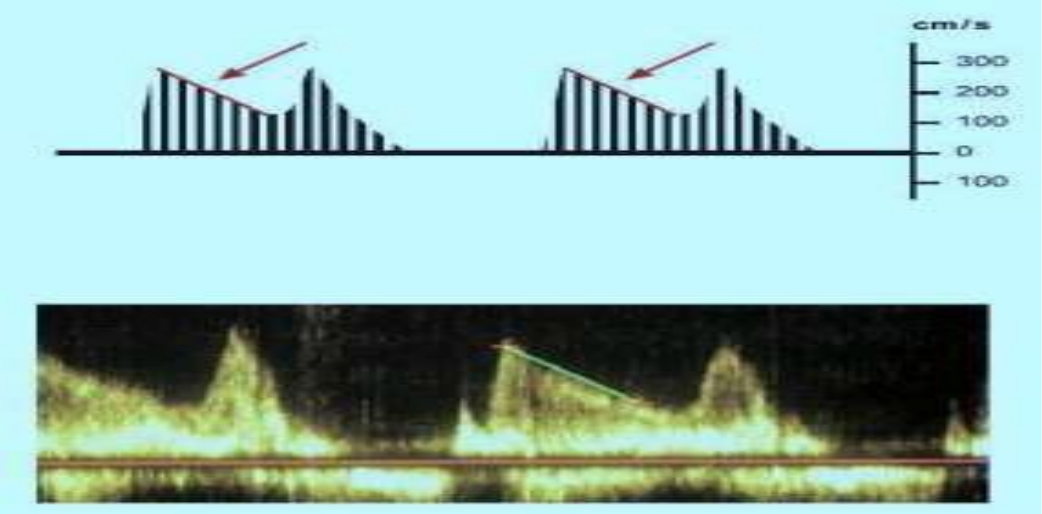


Figure (2): Measuring pressure half-time from Doppler flow envelope of

the mitral inflow (From: Valvular heart disease. In: Bohmeke, Dolvia R, eds. Pocket atlas of echocardiography. 1st ed. Stuttgart: Georg Thieme Verlag; 2006:95).

However, PHT also depends on LA and left ventricular compliance, the maximal pressure gradient, concomitant aortic regurgitation, the presence of an atrial septal defect, or the presence and type of a prosthetic MV and in these instances may be unreliable. Also, PHT method is less reliable immediately after balloon valvuloplasty as it underestimates the severity of mitral stenosis in this situation because PHT becomes shortened (2).

Other ways to calculate MVA from Doppler echocardiography (DE) data are to obtain cardiac output and to divide it by the time velocity integral of the mitral valve, or to use the diameters of the forward jet by color DE in the four-chamber view (D4CV) and the two-chamber view (D2CV) to approximate MVA by ellipse formula (3).

π* D4cv*D2cv/4

The proximal isovelocity surface area (PISA) method is based on the continuity principle and assumes that blood flow converging toward a flat orifice forms hemispheric isovelocity shells. It has been shown that the PISA method is accurate and reproducible (3).

Flow mitral (ml/s) = 2πr2 X (angle α/180) X Valias (cm/s) (3).

In symptomatic patients who do not clearly show severe MS It may be helpful to perform a Doppler hemodynamic assessment during bicycle exercise, after treadmill exercise, or during dobutamine infusion. A marked increase in the transmitral gradient, LA pressure, and pulmonary artery pressure occurs with the exercise-related increase in cardiac output and heart rate. A mean gradient greater than 15mmHg with exercise is considered severe MS. A dobutamine-induced mean mitral pressure gradient greater than or equal to 18mmHg predicted clinical events with 90% accuracy (4).

The comprehensive DE evaluation of MS should include determination of:

1. Peak mitral diastolic velocity by continuous wave DE

2. Mean PG and TVI by tracing mitral velocity

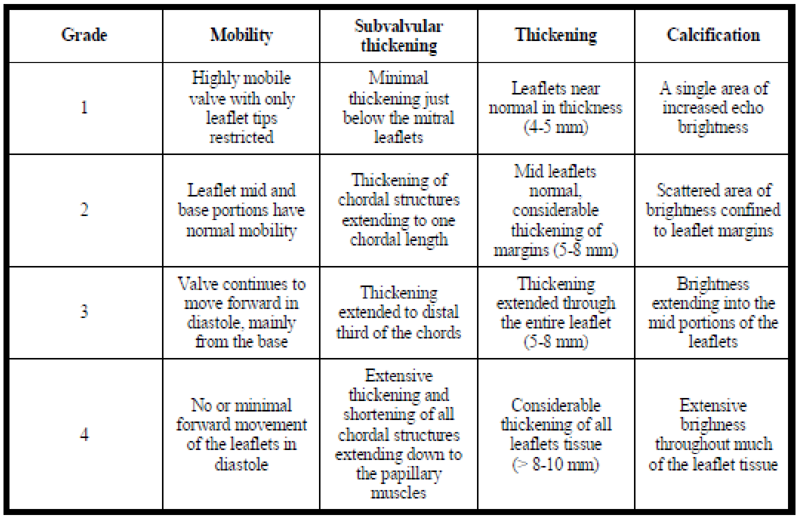
3. MVA by PHT method

4. MVA by the continuity equation and by the PISA

An echocardiographic score which was graded from 0–4 according to the

aforementioned criteria were derived from an analysis of the mitral leaflet mobility, valvular and subvalvular thickening & calcification. This gave a total score of 0–16 (4).

Table (1): Anatomical scores predicting outcome after percutanous mitral commissurotomy: Wilkins' MV morphology score:



Grade	Mobility	Subvalvular thickening	Thickening	Calcification
1	Highly mobile valve with only leaflet tips restricted	Minimal thickening just below the mitral leaflets	Leaflets near normal in thickness (4-5 mm)	A single area of increased echo brightness
2	Leaflet mid and base portions have normal mobility	Thickening of chordal structures extending to one chordal length	Mid leaflets normal, considerable thickening of margins (5-8 mm)	Scattered area of brightness confined to leaflet margins
3	Valve continues to move forward in diastole, mainly from the base	Thickening extended to distal third of the chords	Thickening extended through the entire leaflet (5-8 mm)	Brightness extending into the mid portions of the leaflets
4	No or minimal forward movement of the leaflets in diastole	Extensive thickening and shortening of all chordal structures extending down to the papillary muscles	Considerable thickening of all leaflets tissue (> 8-10 mm)	Extensive brighness throughout much of the leaflet tissue

From: Wilkins GT, Weyman AE, Abascal VM, Block PC, Palacios IF.

For decades, echocardiography has been the only imaging modality that allows dynamic imaging of the heart; it is only natural that new increasingly automated techniques for sophisticated analysis of cardiac mechanics have been driven by researchers and manufacturers of ultrasound imaging equipment (5).

Several such techniques have emerged over the past decades to address the issue of reader's experience and intermeasurement variability in interpretation. Some were widely embraced by echocardiographers around the world and became part of the clinical routine, whereas; others remained limited to research and exploration of new clinical applications (5).

Two such techniques have dominated the research arena of Echocardiography: (1) Doppler based tissue velocity measurements, referred to as tissue Doppler or myocardial Doppler, and (2) speckle tracking on the basis of displacement measurements.

Both types of measurements lend themselves to the derivation of multiple parameters of myocardial function. it is important to make a distinction between myocardial wall motion and wall deformation for a better understanding of different echocardiographic modalities available for the assessment of myocardial contractile function (5).

Whereas velocity and displacement characterize wall motion, strain and strain-rate describe wall deformation. Over time a moving object will change its position (displacement) but does not undergo deformation if all its parts move with the same velocity. If, however, different parts of the object move with different velocities, the object will undergo deformation and will change its shape. Thus, wall motion measurements (displacement and velocity) cannot differentiate between active and passive movement of a myocardial segment, whereas deformation analyses (strain and strain-rate imaging) allow discrimination between active and passive myocardial tissue movement (6).

Displacement: (d) is a parameter that defines the distance that a certain feature, such as a speckle or cardiac structure, has moved between two consecutive frames. Displacement is measured in centimeters.

Velocity: (v) reflects displacement per unit of time, that is, how fast the location of a feature changes, and is measured in centimeters per second.

Strain: (e) describes myocardial deformation, that is, the fractional change in the length of a myocardial segment. Strain is unitless and is usually expressed as a percentage. Strain can have positive or negative values, which reflect lengthening or shortening.

Strain rate: (SR) is the rate of change in strain and is usually expressed as 1/sec. (7).

Displacement and velocity are vectors; that is, in addition to magnitude, they have direction. so, one can examine their different spatial components along the x, y, and z directions, or alternatively, along the anatomic coordinates of the cardiac chambers, longitudinal, radial, and circumferential components, which are especially relevant for the characterization of myocardial mechanics (7).

Similar logic applies to strain and SR, which provide local information on myocardial deformation. The important advantage of strain and SR over displacement is that they reflect regional function independently of translational motion. moreover, deformation imaging cannot distinguish active from passive deformation (8).

The term “strain”, which in everyday language can mean “stretching”, is used in echocardiography to describe “deformation” (8).

The term ‘‘principal strain’’ describes the local magnitude and direction of the shortening or lengthening of the myocardium. The term ‘‘global strain’’ or, more precisely, ‘‘global longitudinal strain’’ or ‘‘global circumferential strain’’ usually refers to the average longitudinal or circumferential component of strain in the entire myocardium, which can be approximated by the averaged segmental strain components in individual myocardial wall segments. Strain values can be expressed for each Segment (segmental strain), as an average value for all segments (global strain mentioned above), or for each of the theoretical vascular distribution areas (territorial strain) (9).

However, the concept of strain is complex. Thus, for a one-dimensional (1D) object (i.e. an infinitesimally thin bar) the only possible deformation is lengthening or shortening and the linear strain (amount of deformation) can be defined by the Formula (2):

$$\varepsilon=\frac{L-L_0}{L_0}=\frac{\Delta L}{L_0},$$

Where ε = strain, L0 = baseline length and L = instantaneous

lengths at the time of measurement.

The instantaneous deformation is thus expressed relative to the initial length (Lagrangian strain). The deformation can be expressed relative to the length at a previous time instance (natural strain) and to be noted that in this definition of instantaneous strain the reference value is not constant over the time but changes during the deformation process. the Lagrangian and natural strain are approximately equal for small deformations whereas the difference between Lagrangian and natural strain are relevant for large deformations which can happen during ventricular contraction and relaxation. it is more appropriate to measure the natural strain for myocardial strain measurements because the measured values are less dependent on the definition of the initial length L0 (5).

the deformation is not limited to lengthening or shortening in one direction for two-dimensional (2D) objects. A 2D object can lengthen or shorten along the x or y axis (normal strain) and can also distort (shear strain) by the relative displacement of the upper to the lower border or the right border to the left border. Thus, the two dimensions strain has four components, two normal strains and two shear strains. three-dimensional (3D) objects are More complex in the deformation as myocardial segments. In this case there are three normal strains (along the x, y and z axes) and six shear strains. To completely define the deformation of 3D objects, all nine strain components must be defined, today, echocardiographic deformation imaging allows 1D measurements based on tissue Doppler imaging and 2D strain measurements based on speckle-tracking imaging (2).

The amount of deformation (positive or negative strain) is usually expressed in percentage (%). Positive strain values describe thickening, negative values describe shortening, of a given myocardial segment related to its original length. During myocardial contraction, as the wall shortens it also thickens and thus assessment of all parameters, radial thickening (positive strain), circumferential shortening (negative strain) and longitudinal shortening (negative strain), is useful for the evaluation of contractile function (2).

Strain rate (SR) is the rate by which the deformation occurs (deformation or strain per time unit). The unit of strain rate is s-1 and the local rate of deformation or strain per time unit equals velocity difference per unit length:

$$\varepsilon = \frac{\Delta\varepsilon}{\Delta t} = \frac{(\Delta L / L_0)}{\Delta t} = \frac{(\Delta L / (\Delta t)}{L_0} = \frac{\Delta V}{L_0},$$

Where HV is the velocity gradient in the segment studied

Thus, the velocity gradient (i.e. difference in velocities between two points of the myocardial wall) can be used for SR calculations. The SR has the same direction as the strain (negative strain during shortening and positive strain during elongation) (2).

The left atrium, contributing up to a third of cardiac output, has an important role in modulating left ventricular filling. Moreover, the left atrium has been identified as an important biomarker of cardiovascular disease and adverse cardiovascular outcomes. the role of LA function as a biomarker is increasingly being evaluated (10), both in combination with LA size and also independently (11). The LA function is known to be complex, comprising of three main components: reservoir function in systole when blood fills the left atrium, as a conduit in early diastole corresponding to passive left ventricular filling and as an active contractile chamber in late diastole.

There is no single parameter that best defines LA function as previously defined. Transmitral peak A wave velocity, its velocity time integral and atrial fraction are well described measures of LA contractile function. The LA ejection force incorporates peak A velocity and was used as a marker of LA function. Subsequently, tissue Doppler derived A' velocity was utilised as a less load dependent measure of LA contractile function, presenting good correlation with traditional Doppler and LA volumetric measurements. Colour tissue Doppler analysis was able to evaluate segmental LA function, showing temporal changes with improved LA function following cardioversion. Although, to use these measures the patient should be in sinus rhythm (SR) but, The LA function index (LAFI) was derived to evaluate LA function even in atrial fibrillation (AF). Additionally, volumetric measures including the LA ejection fraction (LAEF) and LA expansion index (LAEI) have been utilised, both in SR and AF (12).

More recent studies detected that strain analysis has been utilised for evaluation of LA function (13). Strain evaluates myocardial deformation while strain rate examines the rate of change in strain, and can be measured throughout the cardiac cycle, enabling the evaluation of LA reservoir function (in systole) and conduit and contractile function (in diastole).

evaluation of LA function using Strain and strain-rate imaging have several advantages over conventional echocardiography. Firstly, strain imaging is not evaluated relative to the transducer position, which allows discrimination between active and passive myocardial tissue movement. Strain parameters are relatively independent of tethering effects and is less load dependent compared to traditional parameters of LA function. Moreover, strain and strain rate parameters allow evaluation of phasic atrial function throughout the cardiac cycle.although There are no strain algorithms that have been developed yet for evaluation of LA function but several studies have utilised strain software that was developed for the left ventricle, with adjustments to the width of the ‘region of interest’ (ROI) to evaluate LA strain (14-15).

LA strain measurements can be obtained by tissue Doppler imaging (TDI), two-dimensional (2D) speckle tracking echocardiography (STE) and velocity vector imaging (VVI). For the latter two techniques, longitudinal strain and strain rate curves are generated for each of six atrial segments, obtained from the apical four and two chamber views (*Figure 3*). Heterogeneous segmental deformation of the LA has also been observed, with higher values noted in the regions adjacent to the mitral annulus (16).

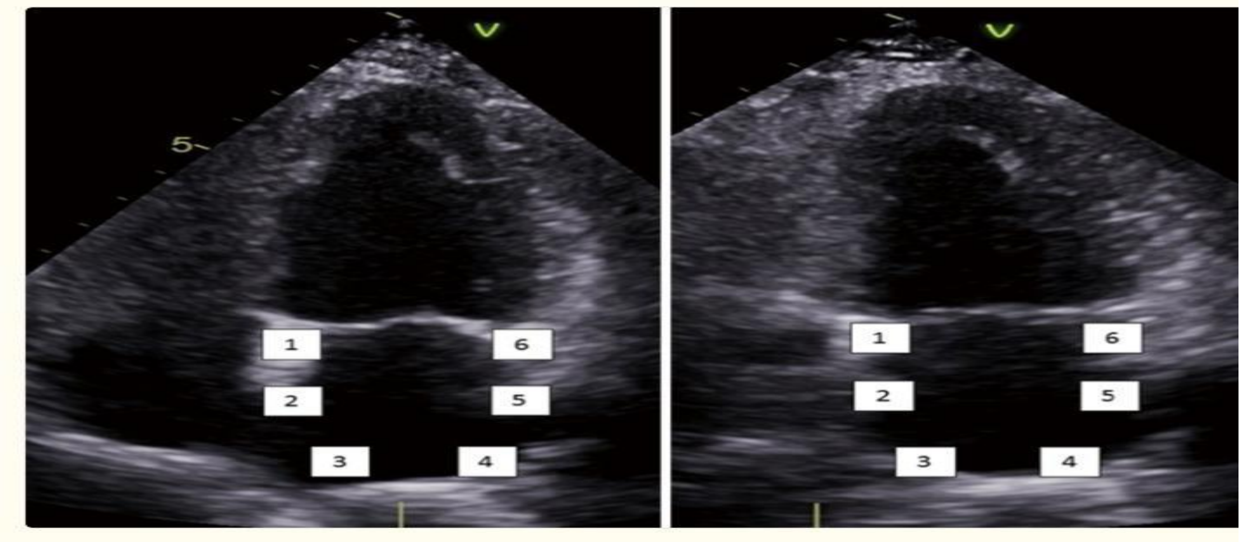
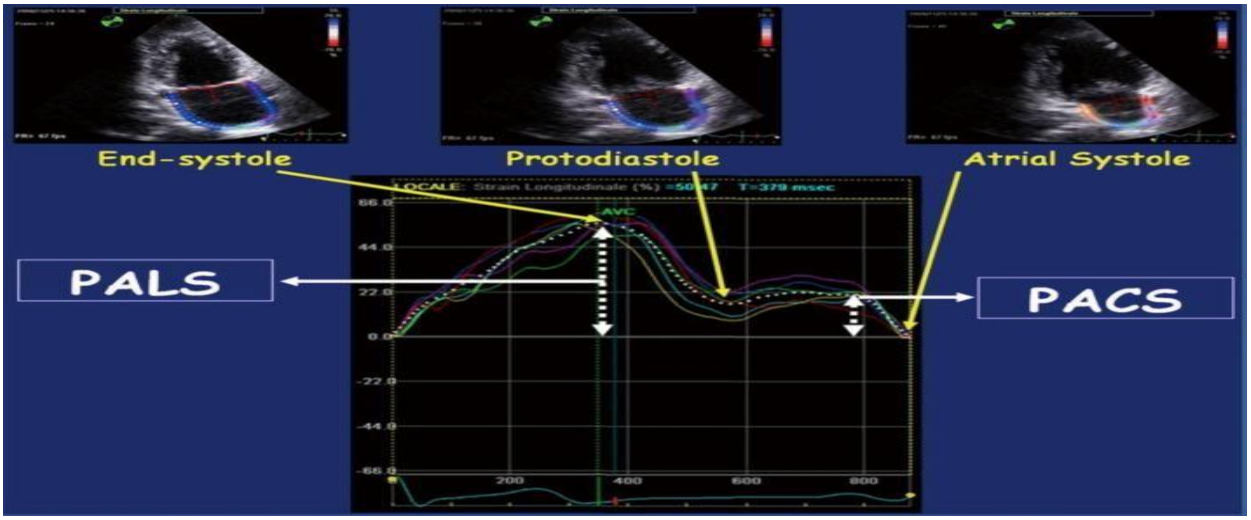


Figure 3: Apical four and two chamber six LA segments. LA, left atrial.

In the reservoir phase, as the LA fills and stretches, there is positive atrial strain that reaches its peak in systole at the end of LA filling, prior to opening of the mitral valve. Followed by passive LA emptying ensues with opening of the mitral valve resulting in decreased atrial strain with negative deflection of the strain curve up to a plateau period which is analogous to diastasis. A second deflection in the strain curve is then detected corresponding to atrial systole. at the end of the reservoir phase Peak atrial longitudinal strain (PALS) or LA systolic strain is measured. Peak atrial contraction strain (PACS) or late diastolic strain, is measured after the P wave and corresponds to active atrial contraction. LA function is affected by loading conditions and heart rate in all three phases. (*Figure 4*) (17).



Fig(4)Peak atrial longitudinal strain (PALS) and peak atrial contraction strain (PACS) (37). Modified from Cameli *et al*.

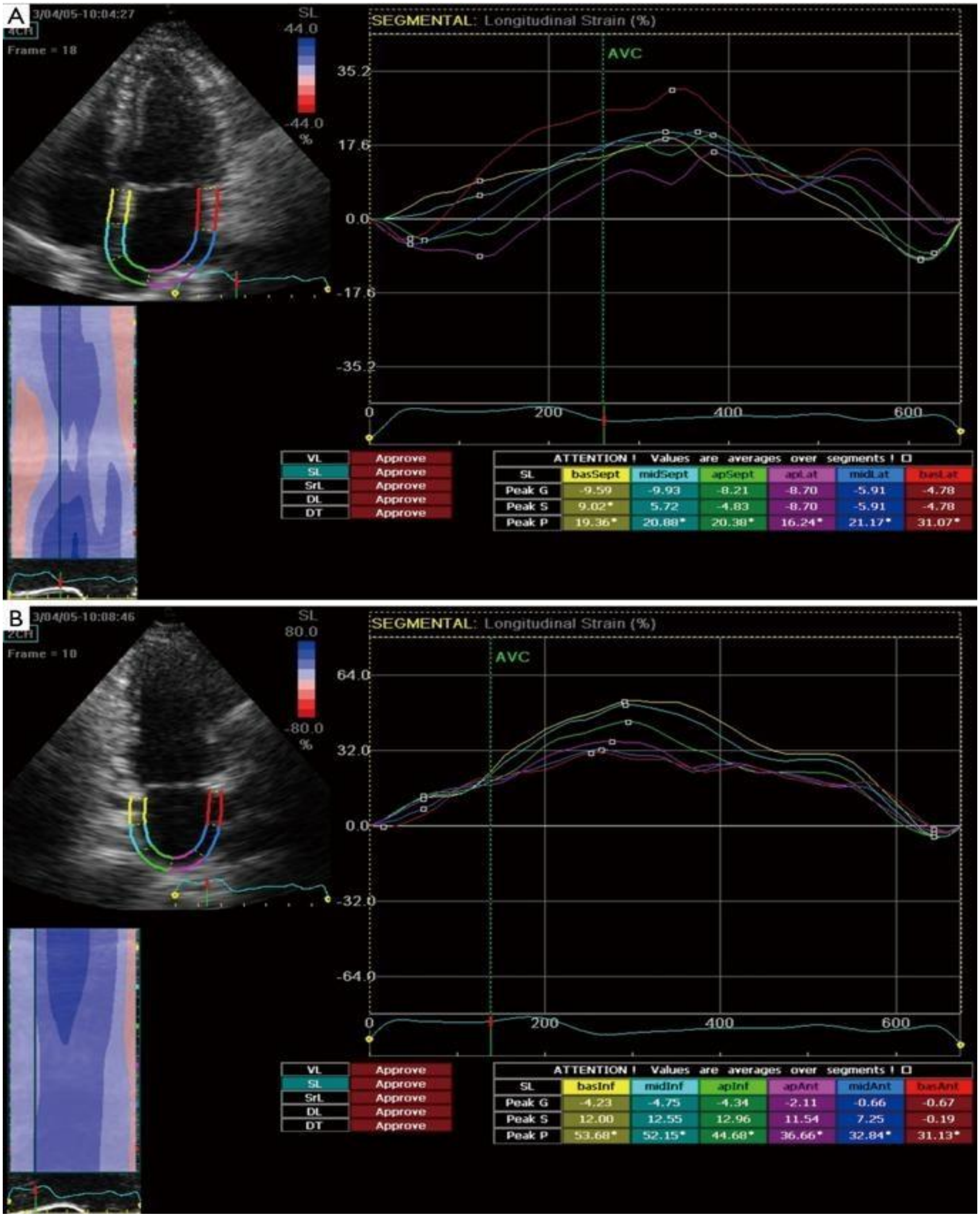
LA reservoir strain (LARS) predicts morbidity and mortality in the general population and has prognostic value in a variety of cardiovascular diseases (18-19). The anatomy and thin walls of the LA make this chamber susceptible to atrial remodelling from arrhythmic damage or pressure/volume overload. LA filling during the reservoir phase results in stretching of the atrial wall, that's why pathologies that cause a reduction in atrial compliance have a direct effect on LA reservoir phasic function. To sum up we can say that the degree of LA interstitial fibrosis correlates to LA reservoir function and this is evaluated by LA strain rather than alternative echocardiographic measurements.

LA contractile strain (LACS) and LA conduit strain (LAScd) have less clinical utility when compared to LARS and require more focused research to further explore their applications. LACS correlates to the active contraction of the atria and is affected by elevated LV filling pressures as well as reduced LA contractile function. LACS has predictive value in AF-reduced LACS is a predictor for the development of new AF, and preserved LACS predicts maintenance of sinus rhythm post DCR (20). Moreover, LACS may also help predict recurrence of AF post catheter ablation, even in patients with normal-sized LA (21).

LA conduit strain (LAScd) results from the pressure gradient between LA and LV with an open mitral valve then it is an indirect marker of LV relaxation. Impaired LAScd is associated with heart failure with preserved ejection fraction (HFpEF) and diastolic dysfunction. LACS has demonstrated prognostic value in specific populations such as patients with congenital aortic stenosis (22) and patients with ESRF (23), however further research is still required for wider application.

Technical Aspects of LA Strain Measurement

The EACVI/ASE/Industry Taskforce has released a consensus statement to standardize LA strain assessment and reduce inter-vendor variability (3). LA strain should be assessed using an optimized apical four-chamber view at a high frame rate (50–70 frames/min). Evidence supports the use of global rather than regional LA strain, and sampling over more of the LA wall probably provides less opportunity for sampling error than a single view. We use we don't use the apical long axis because of potential problems with inclusion of the pulmonary veins and left atrial appendage, only apical 4- and 2-chamber views (3). which may provide problems for automated tracking isn't preferable due to the thin LA and mobile interatrial septum as Manual correction to endocardial tracking is often needed (8). The recent development of specific LA-strain software (e.g. Auto Strain LA and LA Automated Function Imaging) has further consolidated a uniform approach to LA-strain measurement and allows for greater accessibility of these measurements by non-expert operators (13).



VL	Approve
SL	Approve
SrL	Approve
DL	Approve
DT	Approve

ATTENTION ! Values are averages over segments ! ☐

SL	basSept	midSept	apSept	apLat	midLat	basLat
Peak G	-9.59	-9.93	-8.21	-8.70	-5.91	-4.78
Peak S	9.02*	5.72	-4.83	-8.70	-5.91	-4.78
Peak P	19.36*	20.88*	20.38*	16.24*	21.17*	31.07*

VL	Approve
SL	Approve
SrL	Approve
DL	Approve
DT	Approve

ATTENTION ! Values are averages over segments ! ☐

SL	basInf	midInf	apInf	apAnt	midAnt	basAnt
Peak G	-4.23	-4.75	-4.34	-2.11	-0.66	-0.67
Peak S	12.00	12.55	12.96	11.54	7.25	-0.19
Peak P	53.68*	52.15*	44.68*	36.66*	32.84*	31.13*

Fig(5) 2D strain by speckle tracking echocardiography demonstrating segmental and global LA strain from the apical (A) four and (B) two chamber views. LA, left atrial.

Two temporal reference points may be used for tracking the LA border; QRS guided or P wave guided (Fig. 6) although The net displacement with each (ie reservoir strain) is similar. each option has specific considerations—for example QRS complex gating is more challenging in a bundle branch block and P-P gating is not possible in AF (3). Current LA-strain software automatically selects the upslope of the R-wave as the surrogate for end-diastole and generates a template for endocardial tracing. The MASCOT-HIT study took 26 expert centres and showed that both reference points for LA-strain assessment were reproducible, but QRS-guided LA-strain was more feasible and had a shorter analysis time (10).

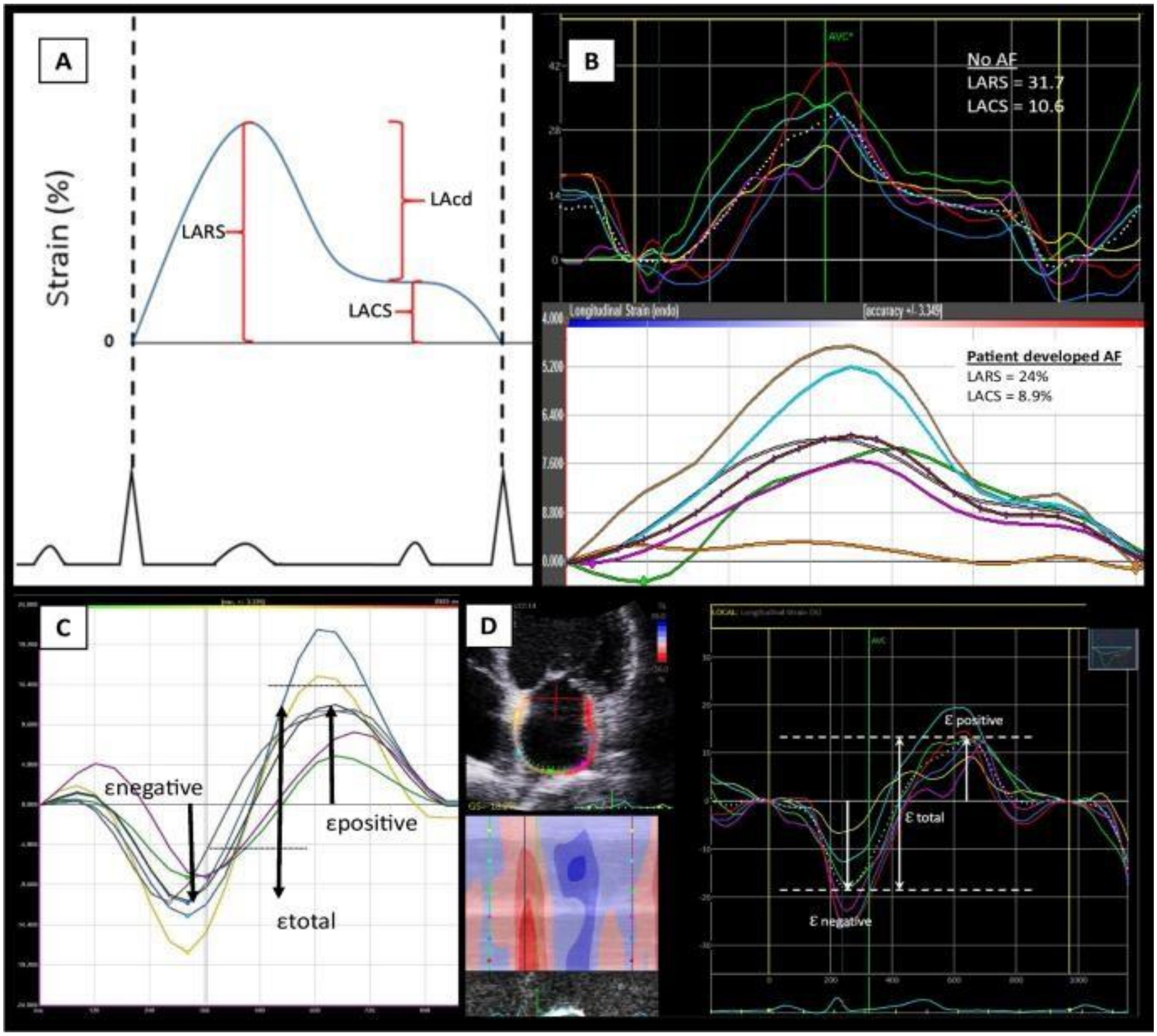
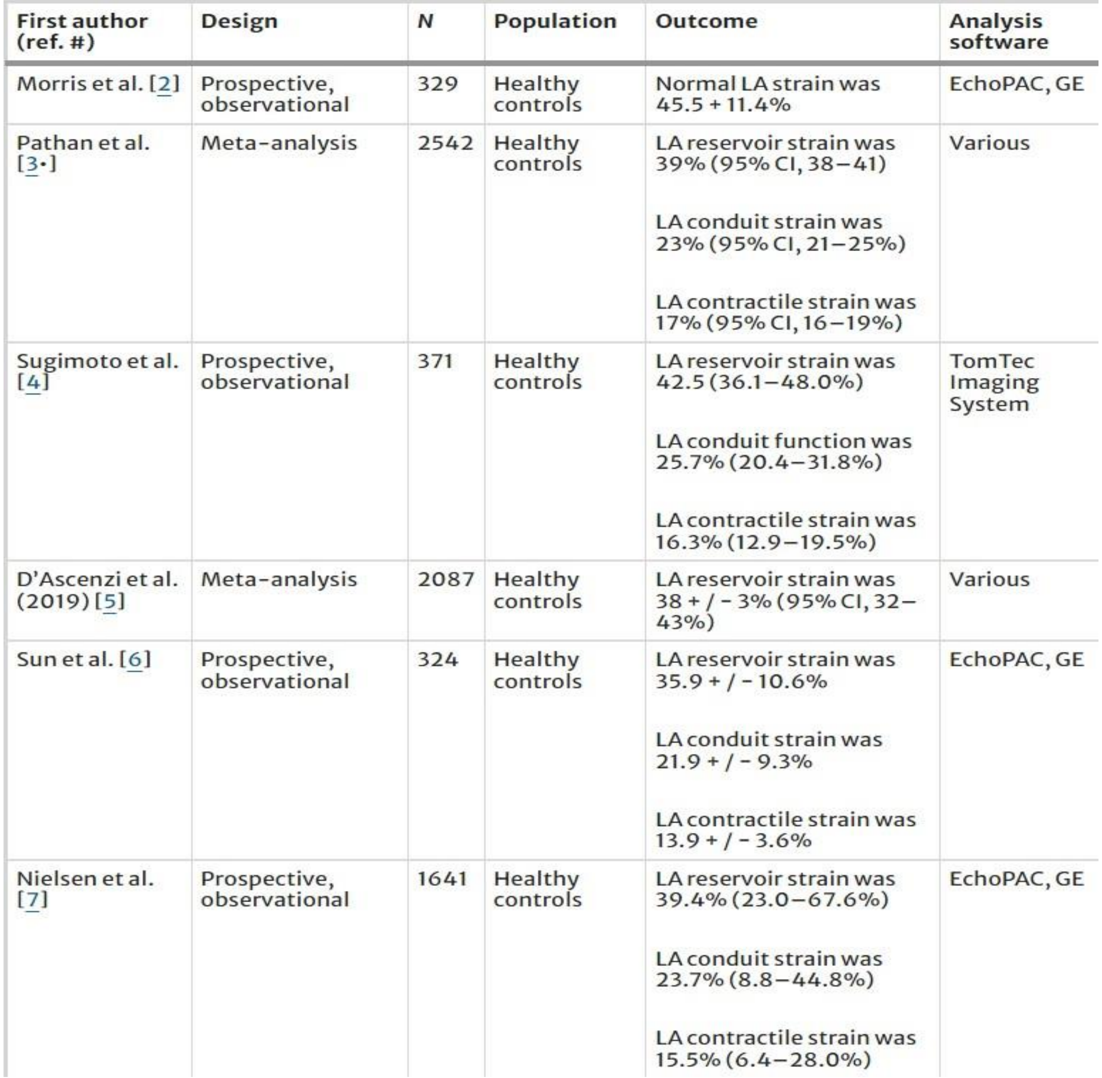


Fig (6) Left atrial strain. **A** R wave triggering. **B** Strain curve demonstrating changes to LA strain during AF (R wave triggering). **C** P wave triggering. **D** Strain curve demonstrating LA strain (P wave triggering) with tracing of LA border

Reference values for LA strain and strain rate have been reported. In a multicenter study involving 329 healthy subjects, Morris *et al.* reported LA systolic strain (i.e., PALS) to be 45.5%±11.4% and LA strain rate during late diastole (i.e., PACS) to be −2.11±0.61 s-1. The lowest expected values (using mean −2 SD) was 23.1% for LA systolic strain and −0.91 s-1 for A sr in late diastole (24).Normal values for LARS have been assessed in two meta-analyses (25-26) (Table 2). Variation of the “normal value” for LARS was influenced by differing ECG gating, sample size and vendor, as well as age, gender, and race. Increasing age correlated with deteriorating LA reservoir and conduit function (27) and this effect appeared to be more pronounced in women (28). The normal value for LARS is ≈ − 38%; however, studies have shown that a LARS cutoff of ≈ > − 22% identifies risk of adverse outcomes (29-30). This implies that LARS has a significant reserve.

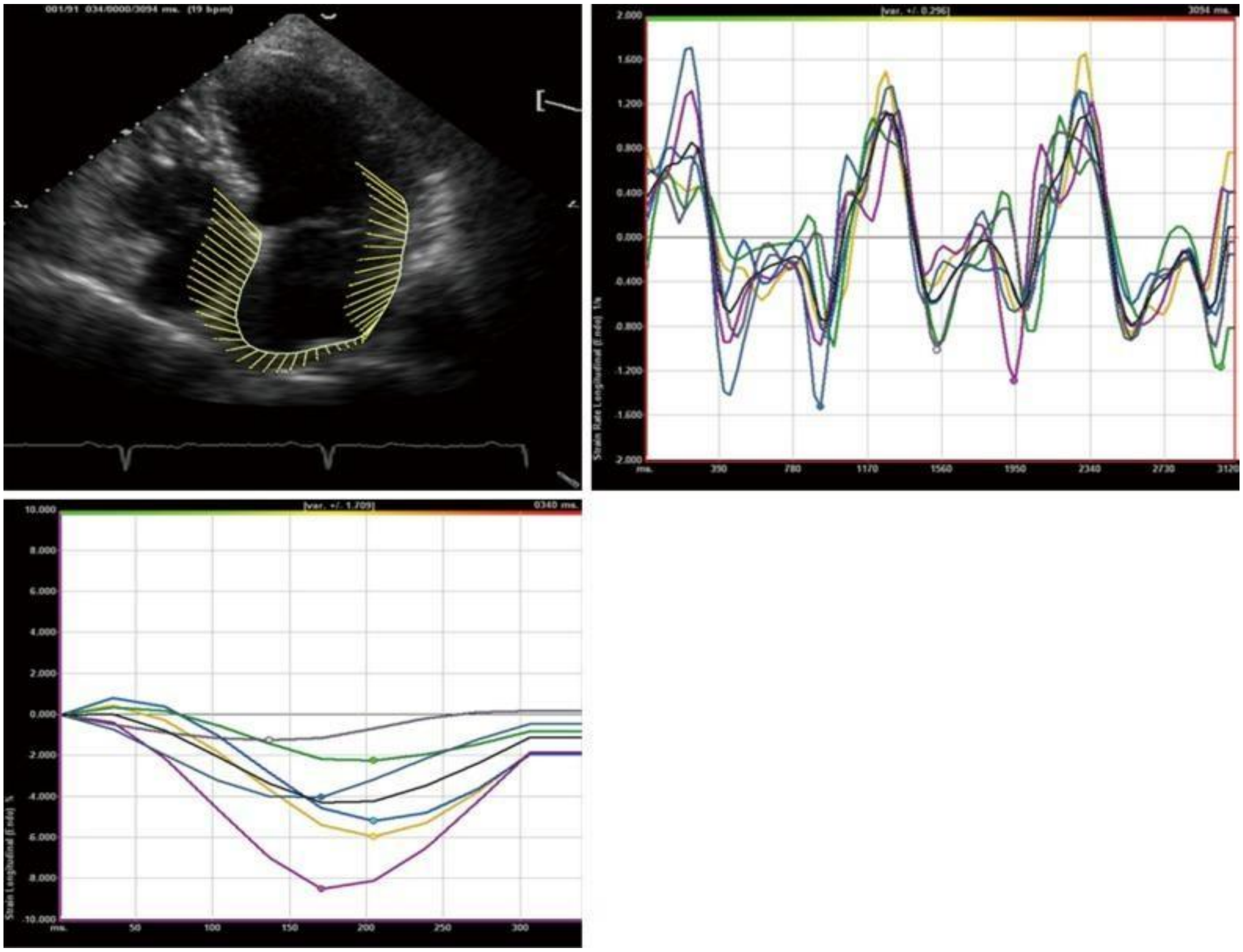
Table (2) LA strain by VVI



First author (ref. #)	Design	*N*	Population	Outcome	Analysis software
Morris et al. [2]	Prospective, observational	329	Healthy controls	Normal LA strain was 45.5 + 11.4%	EchoPAC, GE
Pathan et al. [3•]	Meta-analysis	2542	Healthy controls	LA reservoir strain was 39% (95% CI, 38–41) LA conduit strain was 23% (95% CI, 21–25%) LA contractile strain was 17% (95% CI, 16–19%)	Various
Sugimoto et al. [4]	Prospective, observational	371	Healthy controls	LA reservoir strain was 42.5 (36.1–48.0%) LA conduit function was 25.7% (20.4–31.8%) LA contractile strain was 16.3% (12.9–19.5%)	TomTec Imaging System
D’Ascenzi et al. (2019) [5]	Meta-analysis	2087	Healthy controls	LA reservoir strain was 38 + / - 3% (95% CI, 32–43%)	Various
Sun et al. [6]	Prospective, observational	324	Healthy controls	LA reservoir strain was 35.9 + / - 10.6% LA conduit strain was 21.9 + / - 9.3% LA contractile strain was 13.9 + / - 3.6%	EchoPAC, GE
Nielsen et al. [7]	Prospective, observational	1641	Healthy controls	LA reservoir strain was 39.4% (23.0–67.6%) LA conduit strain was 23.7% (8.8–44.8%) LA contractile strain was 15.5% (6.4–28.0%)	EchoPAC, GE

VVI is a novel echocardiographic method that combines speckle tracking and endocardial border detection. Similar to 2D STE strain imaging, VVI is angle independent but has additional advantages of simpler and faster tracking/processing times compared to conventional STE with the use of a continuously self-updating software and requires only a single frame tracing of the endocardial border.

With VVI analysis, 2D images of apical four and two chamber views are obtained with recommended frame rates between 70–100 Hz. The endocardium of the LA is traced manually in the four and two chamber views and velocity vectors are generated in cine loop format. The ROI is delineated and tracked. The displacement of LA endocardial pixels of the ROI and the velocity of deformation in every frame with the elongation or shortening of myocardium throughout the cardiac cycle, are the strain and SR measures which are calculated automatically (*Figure 7*). Special reference settings are applied, including valve annulus, chamber borders and tissue motion (31).



Velocity vector imaging.

in assessing LA volumes and function VVI has been shown to be feasible and less time consuming. In a study by Valocik *et al.* retrospectively assessing 100 transthoracic echocardiograms, LA volumes derived from VVI time volume curves had a good correlation with conventional LA volume assessment. A moderate level of correlation was noted with respect to LAEF. VVI led to a 62% reduction in measurement time in comparison to conventional 2D assessment. As Clinical applications of LA strain are broad but are most relevant to atrial fibrillation (AF), HFpEF, and valvular heart disease These findings were corroborated by Motoki *et al.* in a separate study involving 127 patients with AF. Measurement of LA strain and SR by VVI and 2D STE was noted to be feasible in a large proportion of patients with comparable strain and strain rate measurements using the two techniques (32).

LA strain assessed by VVI has shown clinical utility in patients with HF. Esmaeilzadeh *et al.* demonstrated that LA strain by VVI was significantly lower in patients with HFrEF compared to healthy subjects in a study involving 35 patients with LVEF <35% in SR. On multivariable analysis of diastolic parameters, a significant inverse relationship was identified between pulmonary arterial pressure and LA strain suggesting that systolic pulmonary artery pressure in HFrEF may be related to LA contractile dysfunction (33).

In mitral stenosis (MS), the combination of an increase in LA pressure and an intense atrial inflammatory response secondary to the underlying rheumatic carditis is accompanied by a progressive increase in interstitial fibrosis of the atrial wall with disorganization of atrial muscle bundles, LA dysfunction, and subsequently LA dilatation (34).

The correlation between LAS-r, New York Heart Association functional state, and MS has been confirmed (35). Regardless of the underlying rhythm (sinus or AF), all patients with severe MS showed a significant reduction of LAS-r (36).[Figure 8]. Using 2D speckle tracking, LAS in all phases was markedly reduced in pure severe MS with sinus rhythm (LAS-r 14.73% ± 8.59%, LAS-cd −7.61% ± 4.47%, and LAS-ct −7.16% ± 5.15%) compared with controls (44.11% ±10.44%, −32.45% ± 7.63%, and −11.85% ± 6.77%, respectively)

Reduced LAS-r ≤7.7% demonstrated a >80% accuracy in identifying patients in need of MV intervention according to the standard criteria in the guidelines (37). The collecting data demonstrated acute improvement of LAS within 24–48 h following balloon valvuloplasty more pronounced in patients with sinus rhythm with no significant difference in LA size. This immediate improvement of LAS-r post balloon valvuloplasty can be used as a good indicator of the procedure success in addition to MV area increase and transvalvular pressure gradient reduction (38) Roslan *et al*. described an improvement of LAS-r from 11.23% ± 6.83% prevalvuloplasty to 16.80% ± 8.82% at 6 months postvalvuloplasty (39).

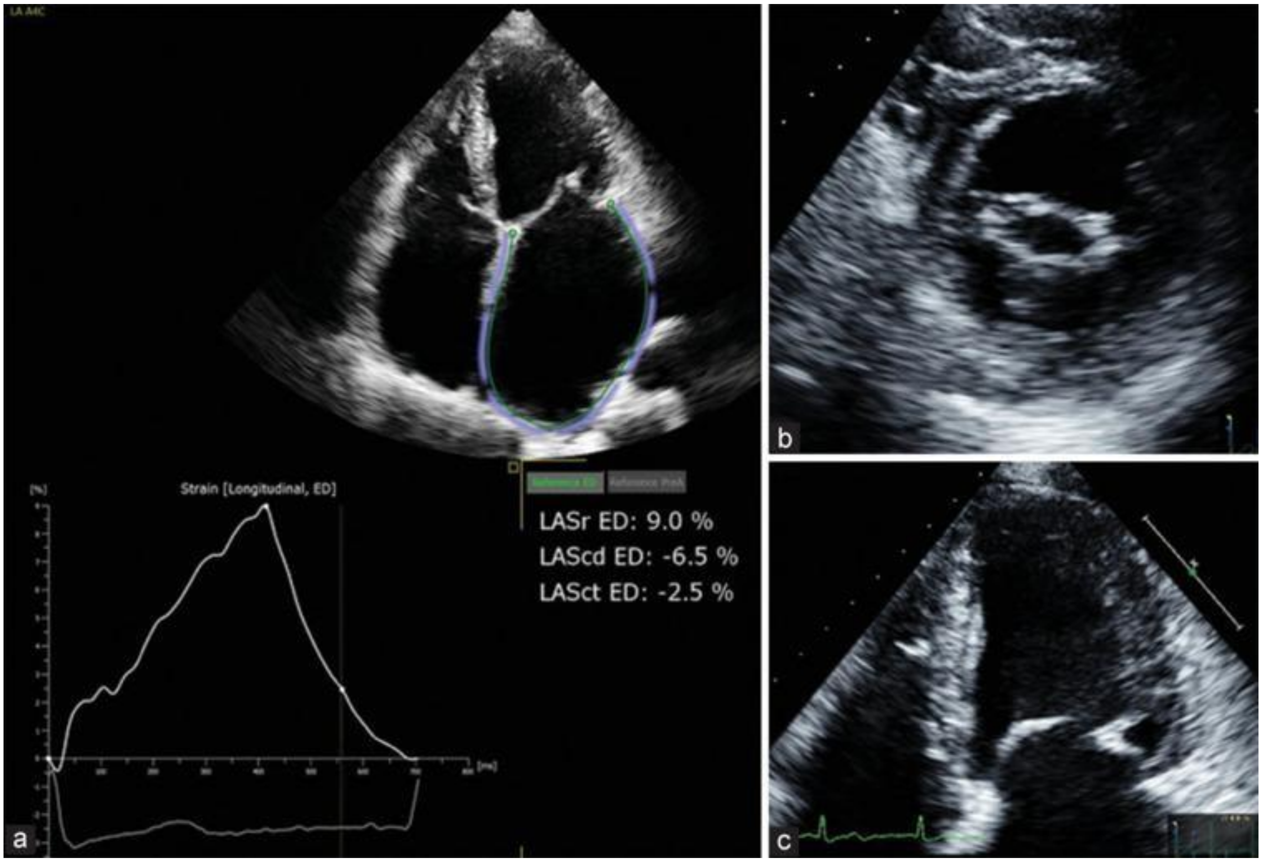


Fig (8) Example of patient with moderate-to-severe mitral stenosis; (a) Left atrial strain (LAS) curve and values in all phases showed marked reduction of LAS-reservoir, LAS-contractile, and LAS-conduit, (b) Two-dimensional echo showing mitral valve orifice in short-axis view, and (c) Apical four-chamber view showing doming of both mitral valve leaflets with restricted opening. LAS-r = Left atrial strain reservoir, LAS-cd = Left atrial strain conduit, LAS-ct = Left atrial strain contractile

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