

ORIGINAL RESEARCH

Left Atrial Strain Determinants During the Cardiac Phases



Georgiana-Grațela Mălăescu, MD,^{a,*}† Oana Mirea, MD, PhD,^{a,*}† Răzvan Capotă, MD,^{a,§}
Aniela Monica Petrescu, MD,^{a,||} Jürgen Duchenne, PhD, MSc,^a Jens-Uwe Voigt, MD, PhD^{a,b}

ABSTRACT

OBJECTIVES The present study investigated the determinants of left atrial (LA) strain in all phases of the cardiac cycle.

BACKGROUND LA strain by speckle-tracking echocardiography allows the assessment of LA function in each phase of the cardiac cycle. However, its determinants and its relation with left ventricular (LV) function have not yet been fully described.

METHODS The authors performed a retrospective analysis in 127 patients with different cardiovascular pathologies. Using 2-dimensional speckle tracking in 4- and 2-chamber apical views we derived both LA and LV strain curves. Strain-strain loops were reconstructed using LV strain and the corresponding, synchronized LA strain data. Linear regressions were calculated for the entire strain-strain loop as well as for the 3 phases of the cardiac cycle (systole, and early and late diastole). The association between LA strain parameters and LV systolic and diastolic parameters was studied. The prediction of cardiovascular events was evaluated for both measured and predicted LA strain and other parameters.

RESULTS LA and LV strain curves presented excellent correlations with an $R^2 > 0.90$ for the cardiac cycle, and $R^2 > 0.97$ for its phases. Moreover, the ratios of LV/LA maximal volumes and the slopes of the LA-LV strain-strain loops of the individual patients correlated well ($R^2 = 0.75$). In each phase of the cardiac cycle, LA strain parameters correlated well with the corresponding LV strain and the LV-LA volume ratio ($R^2 > 0.78$). No significant difference in predictive ability of cardiovascular events or atrial fibrillation between the measured and predicted LA strain was observed ($P > 0.05$ for both).

CONCLUSIONS In the absence of abnormal LA/LV volume exchange, LA strain is, to a large extent, determined by LV strain and further modulated by the ratio of LV and LA volumes. Nonetheless, measuring LA strain is of high clinical interest because it integrates several parameters into a single, robust, and reproducible measurement.

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From the ^aDepartment of Cardiovascular Sciences, Catholic University of Leuven, Leuven, Belgium; and the ^bDepartment of Cardiovascular Diseases, University Hospitals Leuven, Leuven, Belgium. *Drs Mălăescu and Mirea contributed equally to this work. [†]Dr Mălăescu is currently affiliated with Department of Cardiology, Clinical Emergency Hospital, Bucharest, Romania. [‡]Dr Mirea is permanently affiliated with the Department of Cardiology, University of Medicine and Pharmacy, Craiova, Romania. [§]Dr Capotă is currently affiliated with the Department of Cardiology, Institute of Cardiovascular Diseases “Prof. Dr. C.C. Iliescu,” Bucharest, Romania. ^{||}Dr Petrescu is permanently affiliated with the Department of Cardiology, University Medical Center of the Johannes Gutenberg University of Mainz, Mainz, Germany.

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ABBREVIATIONS AND ACRONYMS

AUC = area under the curve
DD = diastolic dysfunction
GLS = global longitudinal strain
HF = heart failure
LA = left atrial/atrium
LAP = left atrial pressure
LV = left ventricle/ventricular
ROC = receiver-operating characteristic

Left atrial (LA) volumetric indices are relevant prognostic markers in heart disease (1) and closely coupled with left ventricular (LV) systolic and diastolic function. The development of speckle-tracking techniques expanded the possibilities to quantify LA function, and atrial global longitudinal strain (GLS) emerged as a new parameter with diagnostic and prognostic value (2).

Most studies have focused on the “reservoir” or peak LA strain in different clinical scenarios, such as heart failure (HF) (3,4), myocardial infarction (5), diastolic function (6), valvular heart disease (7), or as a measure of LV filling pressure (8).

The independence of LA strain parameters from LV function has been challenged in several studies (5,9,10), whereas others found only LA strain during late-diastole to offer independent information beyond LV function assessment (11).

LV longitudinal strain and filling pressures together with LA volume have been identified as the main determinants of LA systolic strain (12). Nonetheless, a detailed analysis of other confounding factors and a comparison between LA and LV strain measurements during the same phase of the cardiac cycle has never been attempted.

The aim of this study was therefore to characterize the determinants of LA strain throughout the cardiac

cycle, to evaluate the relation between LA and LV deformation, and to determine to what extent LA deformation behaves independently from LV deformation.

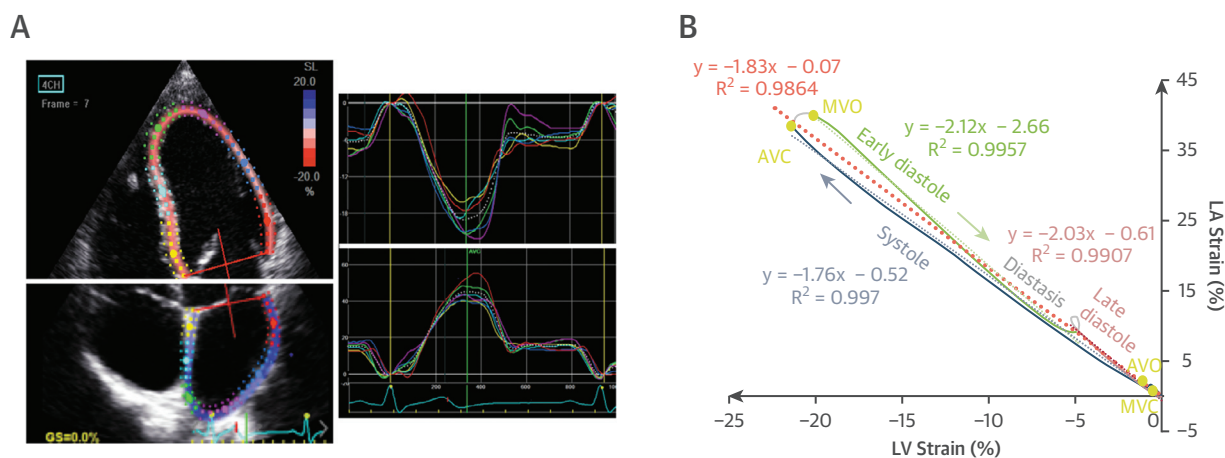
METHODS

STUDY POPULATION. We performed a retrospective analysis of echocardiographic examinations from the echo lab database of the University Hospital Leuven, acquired between November 2018 and July 2019. Images of patients >18 years of age were included if the image quality was suitable for both LA and LV strain analysis. This was discerned by visually inspecting whether the apical 4- and 2-chamber views had acceptable image quality, ie, if one could delineate the LV and LA wall in all segments, and acceptable image geometry, ie, if there was no relevant foreshortening of either of the chambers.

In order to investigate the undisturbed interaction between LA and LV, we excluded patients with pathologies that alter the physiological exchange of volume between these 2 chambers, such as more-than-mild mitral valve disease, more-than-mild aortic regurgitation, previous surgery for valvular heart disease, and congenital heart defects.

Additionally, patients with an excessively mobile interatrial septum, atrial fibrillation, and conduction abnormalities were also excluded from further analysis.

FIGURE 1 Representative Example of LV and LA Strain Tracking and Strain-Strain-Loop



(A) 2D speckle tracking of the left ventricle (LV) and left atrium (LA) in 4-chamber view. **(B)** Reconstruction of an LA-LV strain-strain loop and the corresponding regression lines for the entire loop (red dotted line) and per phase of the cardiac cycle (blue = systole, green = early diastole, and brown = late diastole). Note that each regression line has a slope and an R^2 . Yellow dots indicate aortic and mitral valve opening and closure (AVO, AVC, MVO, MVC). AVC = aortic valve closed; AVO = aortic valve open; MVC = mitral valve closed; MVO = mitral valve open.

The ethical commission of the hospital waived the requirement for a dedicated informed consent in retrospective studies.

STANDARD ECHOCARDIOGRAPHY. All patients underwent comprehensive 2-dimensional transthoracic echocardiography studies using Vivid E9 and E95 machines (GE Vingmed Ultrasound AS). The average acquisition frame rate was 76 ± 7 frames/s. Images were digitally stored and analyzed off-line (EchoPAC, Version 202, GE Vingmed Ultrasound AS). Standard measurements were performed according to current guidelines (13). LA and LV volumes were measured in end-diastole and end-systole using the semi-automated function of the analysis software (Auto-EF) in the apical 4- and 2-chambers views. Elevated mean left atrial pressure (LAP) was noninvasively defined according to current guidelines on diastolic function assessment (14). Diastolic dysfunction (DD) grades 2 and 3 were supposed to indicate elevated LAP.

STRAIN MEASUREMENTS. LA and LV strains were measured using EchoPAC Q-Analysis LV speckle-tracking software in both apical 4- and 2-chamber views according to current recommendations (9,15). Measurements were performed blinded to clinical data by a single observer (G.G.M.) experienced in speckle-tracking analysis. LA and LV strains were obtained from the same cardiac cycle. The QRS trigger was used as zero reference point for both strain curves. Tracking of the LV and LA was considered unsatisfactory if the tracking markers of the software did not accurately follow the motion of the underlying myocardium on visual inspection (16). As recommended, strain values were calculated as the difference between 2 points on the strain curve (16) and named according to the corresponding phase of the cardiac cycle (Figure 1A):

1. LA and LV systolic strain (as the difference of strain values at end of systole and the reference point);
2. LA and LV early-diastolic strain (as the difference between strain values at the start of LA contraction and the end of systole);
3. LA and LV late-diastolic strain (as the difference between strain values at the reference point and the start of LA contraction).

LA STRAIN-LV STRAIN LOOPS. For each patient, global LA and LV strain curves corresponding to the 4- and 2-chamber views were exported numerically, and strain-strain loops were reconstructed using LV strain values on the x-axis and the corresponding time-aligned LA strain values on the y-axis (Figure 1B). The regression equation was calculated,

TABLE 1 Clinical and Echocardiographic Characteristics of the Study Population

Age	59.3 \pm 18.8
Male	67 (52.7)
Normal hearts	26 (20.4)
Hypertensive heart disease	23 (18.1)
Ischemic cardiomyopathy	27 (21.3)
Dilated cardiomyopathy	18 (14.2)
Cardiac amyloidosis	15 (11.8)
Hypertrophic cardiomyopathy	8 (6.3)
Aortic stenosis	10 (7.9)
LV ED volume (ml)	113.1 \pm 38.7
LV ES volume (ml)	48.5 \pm 14.9
EF (%)	54.4 \pm 10.4
MAPSE average (mm)	12.4 \pm 3.1
LA ES volume (ml)	78.1 \pm 28.6
LA ED volume (ml)	41.9 \pm 26.8
E (cm/s)	78 \pm 21.5
A (cm/s)	63.9 \pm 25.3
E/A	1.5 \pm 0.9
e' average (cm/s)	7.9 \pm 3.3
E/e' average	10.6 \pm 5.7
a' average (cm/s)	7.5 \pm 3.1
TR peak velocity(m/s)	2.6 \pm 0.6
TAPSE (mm)	21.4 \pm 4.8
LA systolic strain (%)	27.4 \pm 11
LA early diastolic strain (%)	-14.4 \pm 6.8
LA late diastolic strain (%)	-12.3 \pm 6.3
LV systolic strain (%)	-15.5 \pm 4.1
LV early diastolic strain (%)	9.6 \pm 3.1
LV late diastolic strain (%)	4.9 \pm 2.2

Values are as mean \pm SD or n (%).

A = peak mitral inflow late velocity; a' = mitral annular late velocity; E = peak mitral inflow early velocity; e' = mitral annular early velocity; ED = end diastolic; EF = ejection fraction; ES = end systolic; LA = left atrium; LV = left ventricle; MAPSE/TAPSE = mitral/tricuspid annular plane systolic excursion; TR = tricuspid regurgitation.

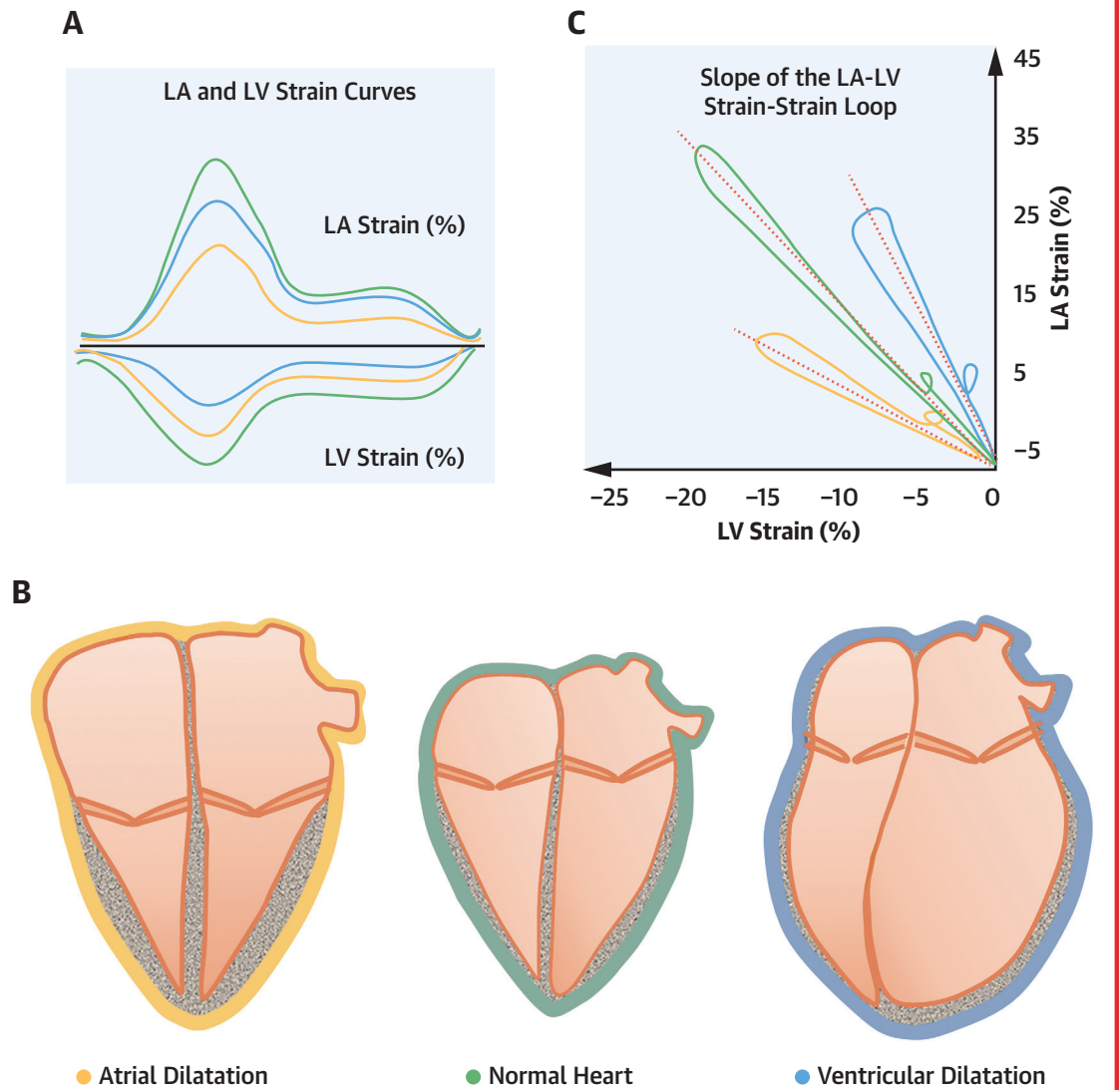
and the correlation between LA and LV strain was determined for the entire loop and for every phase of the cardiac cycle separately (Figure 1B). The area of each strain-strain loop was calculated and normalized by expressing it as a percentage of the area of its tangential circle (Supplemental Figure 1).

CLINICAL OUTCOME PREDICTION. In order to determine the prognostic value of different

TABLE 2 Correlation Between LA and LV Strain Curves From the Strain-Strain Loops

Strain-Strain Loop	R ²
Entire loop	0.91 \pm 0.05
Systole	0.97 \pm 0.02
Early diastole	0.98 \pm 0.02
Late diastole	0.97 \pm 0.03

LA = left atrium; LV = left ventricle.

CENTRAL ILLUSTRATION Left Atrial and Left Ventricular Strain Curves and the Respective Strain-Strain Loops in 3 Clinical Scenarios

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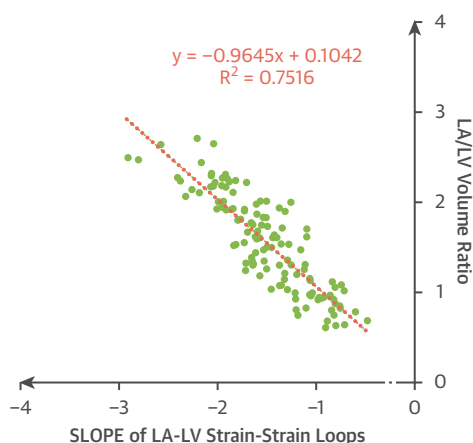
(A) Left atrial (LA) and left ventricular (LV) strain curves are obtained in 3 categories of patients. (B) Yellow = atrial dilatation; green = normal heart; blue = ventricular dilatation. (C) LA-LV strain-strain loops were reconstructed for each patient. Note the differing slopes (dotted red lines) of the LA-LV strain loops, reflecting the LV/LA volume relation.

parameters, data on clinical outcome were collected retrospectively from the hospital electronic records by an observer (O.M.) blinded to the patient's echocardiographic measurements at a time point 2 years after the index echocardiogram. Data could be obtained in 127 of the patients (100%). The mean follow-up period was 2.2 ± 0.5 years (between the baseline examination and May 2021). Cardiovascular events were defined as the first occurrence of any of the

following: acute coronary syndrome; myocardial revascularization (coronary artery bypass graft or percutaneous transluminal coronary angioplasty); hospitalization for HF; novel heart block; or new onset of atrial fibrillation. Furthermore, a new onset of atrial fibrillation served as a separate endpoint.

STATISTICAL ANALYSIS. Continuous variables were expressed as mean \pm SD; categorical variables were expressed as numbers (percentages). Differences

FIGURE 2 Correlation Between the Slopes of LA-LV Strain-Strain Loops and the Respective LV/LA Volume Ratios



For each patient, the ratio of the left ventricular (LV) over left atrial (LA) volumes was calculated and correlated with the slope of the LA-LV strain-strain loop. Note that the resulting regression line has a slope of nearly 1.

between 2 groups were tested using Student's *t*-test. Univariate regression models were used to analyze the relation between LA strain parameters and different echocardiographic parameters. Significant parameters were further included in stepwise multiple regression models. A receiver-operating characteristic (ROC) curve analysis was performed to assess the association of different echocardiographic measurements with cardiovascular events.

LA strain measurements were repeated at an interval of 2 weeks in 18 randomly selected cases. Both readers (G.G.M. and R.C.) were blinded to previous measurements. Bland-Altman statistics were used to assess the intra- and interobserver agreement. Statistical analysis was performed using IBM SPSS software version 25. A 2-sided *P* value of <0.05 was considered statistically significant for all tests. The comparison of areas under the curve (AUCs) was performed using the DeLong et al (17) method provided by MedCalc software.

RESULTS

STUDY POPULATION AND BASELINE CHARACTERISTICS.

We identified 671 cases with sufficiently high-quality image data for both LV and LA strain analysis. From those, 544 cases were excluded based upon the established exclusion criteria (31.3% caused by more-than-mild mitral regurgitation, 5.0% caused by more-than-mild aortic regurgitation, 26.8% caused by a

highly mobile interatrial septum, 8.6% caused by conduction disturbances, and 9.6% caused by previous cardiac surgery). Finally, echocardiographic studies of 127 patients (67 male) could be included.

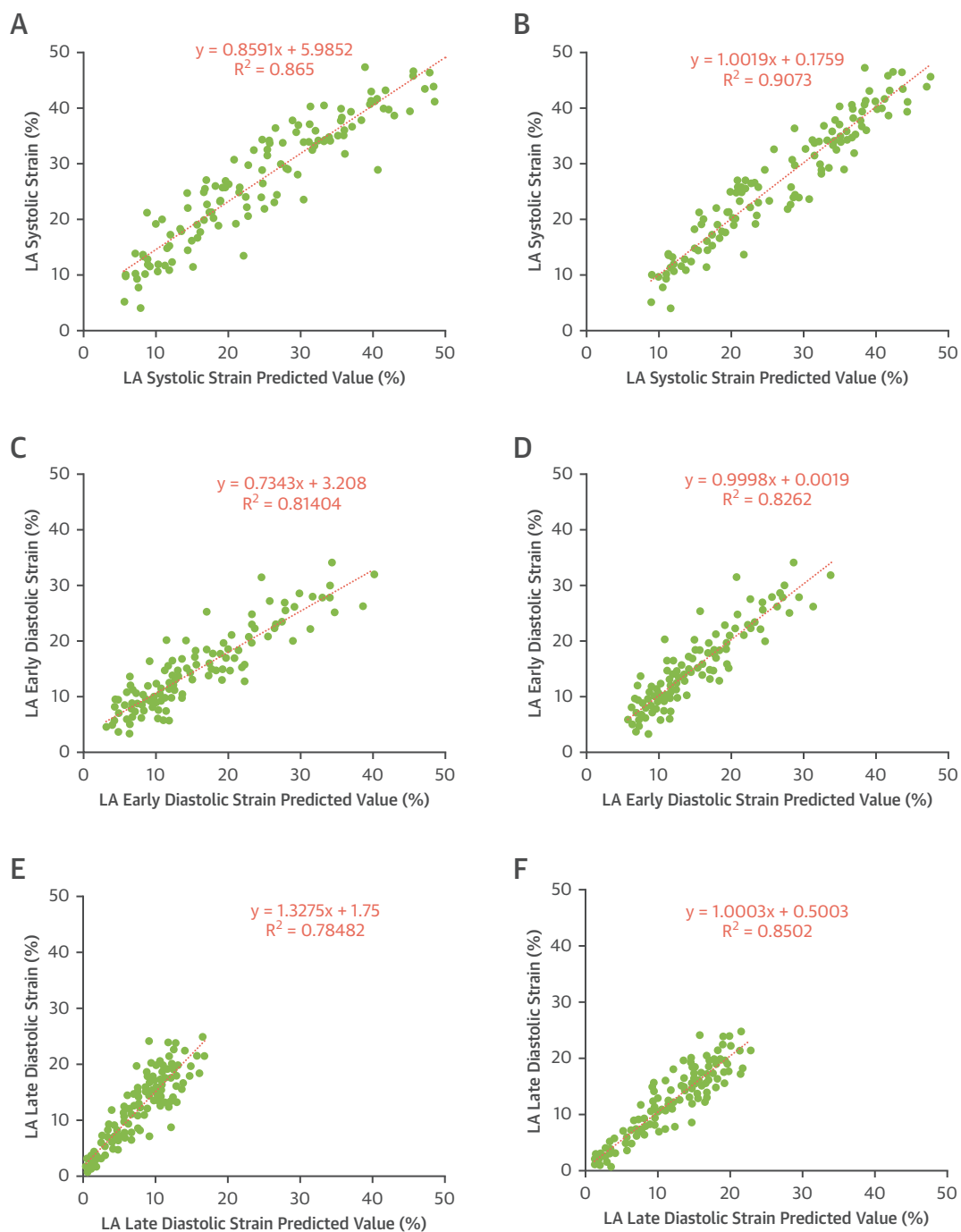
The study cohort had a mean age of 59.3 ± 18.8 years and comprised a wide range of cardiovascular pathologies. Ejection fractions ranged from 23.9% to 69.9%. Diastolic function assessment according to current guidelines (14) categorized 26 patients (20.4%) as having normal diastolic function, 35 patients (27.6%) as grade 1 DD, 38 patients (29.9%) as grade 2 DD, and 25 patients (19.7%) as grade 3 DD, leaving 3 patients (2.4%) with indeterminate diastolic function. The characteristics of our study cohort are summarized in Table 1.

SHAPE ANALYSIS OF STRAIN CURVES. LV and LA strain measurements could be obtained in all 127 patients. LA and LV strain curves showed similar—yet inverted—shapes throughout the cardiac cycle (Figure 1). Accordingly, most LA-LV strain-strain loops were narrow and close to a line, resulting in an average correlation factor R^2 of 0.91 (Table 2). Subjects with an elevated LAP had a slightly, but significantly, greater normalized loop area compared with those with normal LAP ($8.2 \pm 4.1\%$ vs $5.1 \pm 3.5\%$; $P = 0.004$) (Supplemental Figure 1).

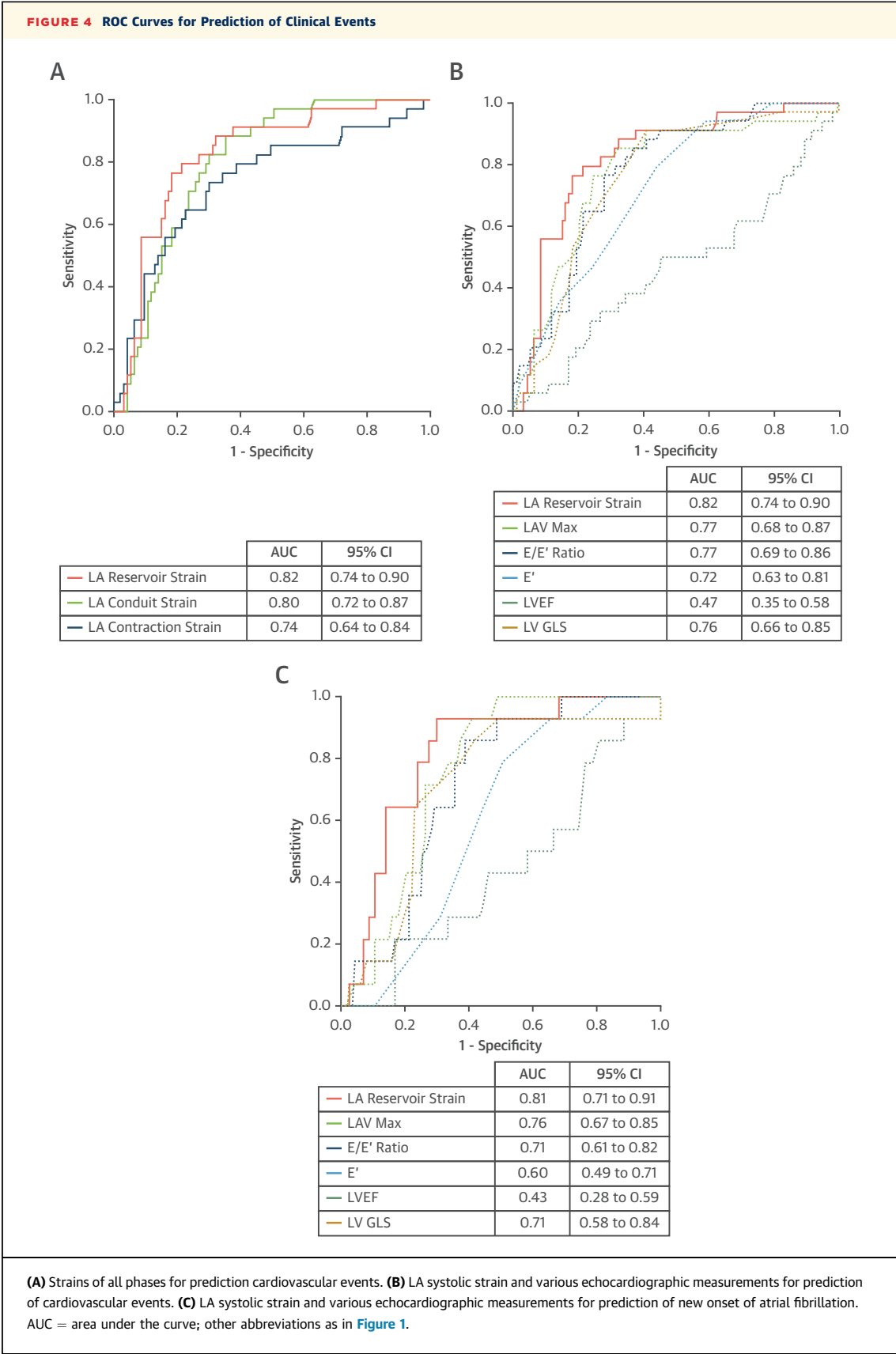
We observed in our cohort, that the slope of the regression line of an individual LA-LV strain-strain loop (ie, the LA/LV strain ratio of the entire cardiac cycle) was related to the respective LV maximum volume and inversely related to the respective LA maximum volume (Central Illustration). For the entire cohort, the individual LA/LV strain ratios correlated strongly with the individual LV/LA maximum volume ratios (slope = -0.96 , offset = 0.1 , $R^2 = 0.75$; $P < 0.0001$) (Figure 2).

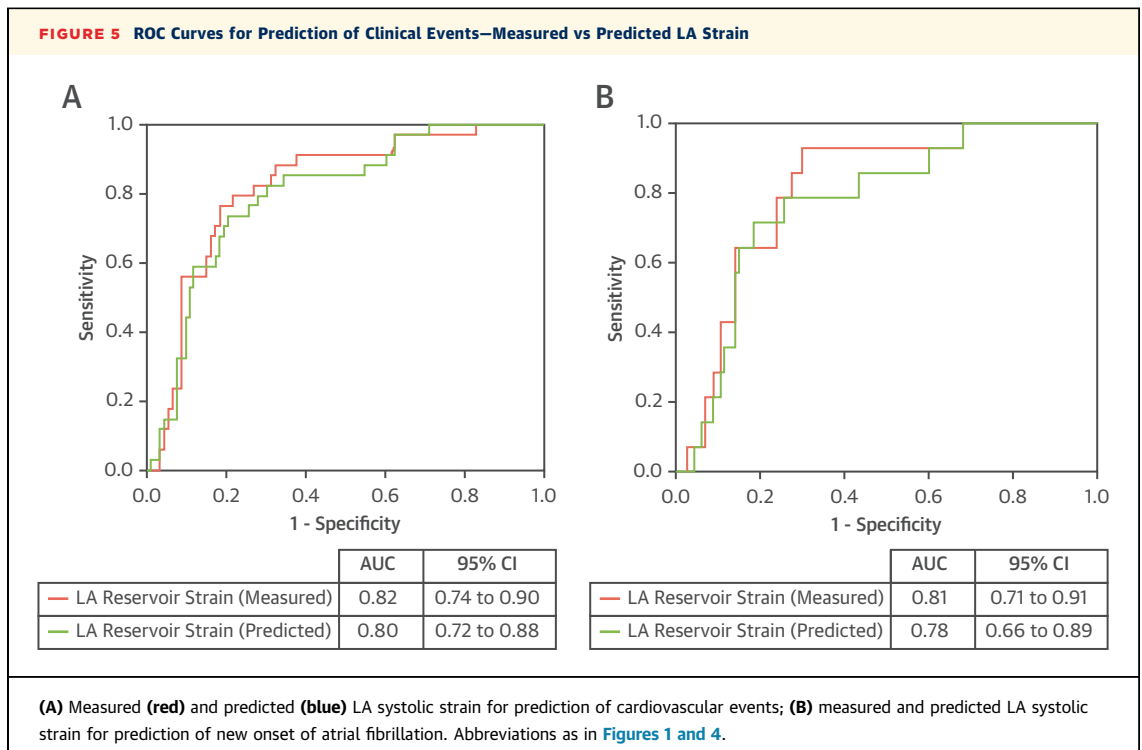
ASSOCIATIONS OF LA STRAIN WITH OTHER ECHOCARDIOGRAPHIC PARAMETERS. **LA systolic strain (LA reservoir strain).** LA systolic strain correlated significantly with LV systolic strain ($r = -0.78$; $P < 0.0001$), as well as with the ratio of LV/LA volumes ($r = 0.73$; $P < 0.0001$). In addition, other parameters of LV systolic and diastolic function showed moderate or good correlations with LA systolic strain (Supplemental Table 1).

A lumped regression model containing only LV systolic strain multiplied with the LV/LA volume ratio as the only parameter was able to predict the LA systolic strain with an R^2 of 0.87 ($P < 0.0001$) (Figure 3A). Adding all echocardiographic parameters that were significant on univariate analysis increased the predictive power of the model by 4% to an $R^2 = 0.91$ ($P < 0.0001$) (Figure 3B). Elevated

FIGURE 3 Correlation Between Predicted and Measured LA Strain Parameters

Left: Correlation between the measured left atrial (LA) strain parameter (systolic [A], and early [C] and late [E] diastolic strain, respectively) and the LA strain predicted from the simple model including only the left ventricular strain and LV/LA volume ratio. **Right:** The same (systolic [B], and early [D] and late [F] diastolic strain, respectively), but with the model including all significant parameters from the respective univariate regression analysis (see text for details).





LAP, together with mitral ring a' velocity and peak mitral inflow A velocity, were the only parameters that had a significant additional influence ([Supplemental Table 1](#)).

LA early-diastolic strain (LA conduit strain). LA early-diastolic strain correlated with LV early-diastolic strain ($r = -0.71$; $P < 0.0001$) and the ratio of LV/LA volumes ($r = 0.69$; $P < 0.0001$). The corresponding lumped regression model was able to predict LA early-diastolic strain with an R^2 of 0.81 ($P < 0.0001$) ([Figure 3C](#)). Adding all echocardiographic parameters to the aforementioned model improved its predictive power by 1.5% to $R^2 = 0.83$ ($P < 0.0001$) ([Figure 3D](#)). The only additional parameter with minor, but significant, additional influence was e' velocity ([Supplemental Table 2](#)).

LA late-diastolic strain (LA contraction strain). Similarly, LA late-diastolic strain correlated with LV late-diastolic strain, as well as the ratio of LV/LA volumes ($r = -0.81$ and $r = 0.51$, respectively; $P < 0.0001$ for both). The corresponding lumped regression model was able to predict LA late-diastolic strain with an R^2 of 0.78 ($P < 0.0001$) ([Figure 3E](#)). Adding all significant echocardiographic parameters improved the predictive value of the model by 7% to $R^2 = 0.86$ ($P < 0.0001$) ([Figure 3F](#)). The estimated presence of elevated LAP and the mitral ring a' velocity remained as additional parameters with significant influence ([Supplemental Table 3](#)).

Bland-Altman plots depicting the difference between the measured and predicted values of LA strain for all 3 phases are provided in [Supplemental Figure 2](#).

CLINICAL OUTCOME PREDICTION. We recorded 34 events during the follow-up period (14 HF hospitalizations, 14 new atrial fibrillation, 2 strokes, 3 coronary events, 1 new heart block). Systolic and early-diastolic LA strain showed both a good performance to predict cardiovascular events ([Figure 4A](#)) with an AUC of 0.82 (95% CI: 0.74-0.90), respectively 0.80 (95% CI: 0.72-0.87) ($P = 0.45$), which was higher compared with LA late-diastolic strain AUC = 0.74 (95% CI: 0.64-0.84; $P < 0.05$ for both). The AUCs of the different echocardiographic parameters ranged from 0.47 for LV ejection fraction to 0.82 for LA systolic strain ([Figures 4B and 4C](#)). The LA systolic strain showed a higher AUC compared with LV GLS to predict both cardiovascular events (AUC = 0.82 vs 0.76; $P = 0.05$) and atrial fibrillation (AUC = 0.81 vs 0.71; $P < 0.01$).

There was no statistical difference in prediction ability for cardiovascular events and atrial fibrillation between the measured and predicted LA systolic strain (AUC = 0.82 vs AUC = 0.80; $P = 0.27$ [[Figure 5A](#)]; and AUC = 0.81 vs AUC = 0.78; $P = 0.22$ [[Figure 5B](#)], respectively).

REPRODUCIBILITY. For intraobserver agreement, the bias $\pm 96\%$ limits of agreement were $-0.62 \pm 3.5\%$

(LA systolic strain), 0.44 ± 2.7 (LA early-diastolic strain), and $0.19 \pm 2.3\%$ (LA late-diastolic strain).

For interobserver agreement, the bias $\pm 96\%$ limits of agreement were $-0.75 \pm 4.6\%$ (LA systolic strain), $-0.06 \pm 3.6\%$ (LA early-diastolic strain) and $-0.55 \pm 4.8\%$ (LA late-diastolic strain).

The Bland-Altman plots for intraobserver and interobserver agreement are shown in [Supplemental Figure 3](#).

DISCUSSION

To the best of our knowledge, this is the first study that has systematically investigated the relation between speckle-tracking-derived LV and LA strain measurements in all 3 phases of the cardiac cycle. Our findings can be summarized as follows: 1) LA and LV deformation is closely coupled; 2) LA strain is in all cardiac phases highly dependent on LV strain of the same phase, and further modulated by the ratio of LV and LA volumes; 3) LA strain parameters do also integrate other parameters of diastolic function; and 4) LA strain is a good predictor of cardiovascular events.

RELATION BETWEEN LA AND LV STRAIN THROUGHOUT THE CARDIAC CYCLE. LV and LA are mechanically connected and share the motion of the mitral ring, which is reflected by the strong correlation of LA and LV strain curves throughout the cardiac cycle. However, because longitudinal strain describes a change in length relative to a given baseline length, the annular displacement is reflected differently in both strain curves, depending on the respective chamber dimensions. This close relation is proven by the good numeric correlation between the slope of the LA-LV strain-strain loops (ie, the LA/LV strain ratio) and the LV/LA volume ratio, which itself has a slope of almost 1 ([Figure 2](#)). LA shape changes between end-diastole and end-systole can explain an additional effect of LAP in the dynamic relationship between the strain curves ([Supplemental Figure 2](#)). Further, blood conduit and flow reversal into the veins may play a role.

The relation between LA and LV strains may be further modulated by pathology that interferes with the blood volume exchange between the 2 cavities, such as valve regurgitations. This, however, was not investigated in this study.

In the following, we therefore discuss, per cardiac phase, to what extent LA strain depends on LV strain of the same phase and the volume ratio of LV and LA, as well as other echocardiographic parameters.

LA systolic strain (reservoir strain). Most of the previous studies focused mainly on peak LA strain. They found good correlations with LV GLS and the indexed LA

volume, varying according to the selected population ([5,11,17](#)). In our study, similarly good correlations were found. The model including only LV systolic strain and the LV/LA volume ratio predicted LA strain numerically while explaining 86% of its variability ([Figure 3A](#)). Given a measurement variability in the range of 10% for LA strain and 6% for LV strain ([18](#)), very little of the strain variation remains unexplained. This is confirmed by the stepwise multivariate regression model, where only the presence of elevated LAP, mitral ring a' velocity, and peak mitral inflow A velocity remained as additional significant predictors. The beta-coefficients of these parameters, however, were rather low ([Table 1, Figure 3B](#)). The (minor) influence of elevated LAP might be explained by different shape changes of the LA during the cardiac cycle depending on the filling pressures. In that way, volume changes of the chamber could translate differently into strain changes ([Supplemental Figure 4](#)).

LA early-diastolic strain (conduit strain). Most of the previous studies correlated diastolic LA strain measurements with LV systolic strain, and found at best modest or no correlations ([11](#)). In early diastole, LV strain of the corresponding phase and the volume ratio of both chambers could explain 81% of the variance of LA strain ([Figure 3C](#)). This was improved in the stepwise multivariate model where also the e' velocity remained as a predictor ([Table 2, Figure 3D](#)). In this phase of the cardiac cycle, differential strain changes of both chambers can only be explained by a different degree of conduit volume passing through the LA into the ventricle without causing a deformation of the LA. In this sense, a high e' velocity might be a good representative of a high conduit volume.

Determinants of LA late-diastolic strain (contraction strain). In late diastole, 78% of the variance of the LA strain was explained by the corresponding standard model ([Figure 3E](#)). Adding classical echocardiographic parameters, elevated LAP and a' velocity remained and were associated with a 7% better prediction ([Supplemental Table 3, Figure 3F](#)). In this phase of the cardiac cycle, differential strain changes of both chambers can only be explained by the amount of retrograde volume flow into the pulmonary veins, which allows atrial volume reduction without increasing LV volume. Both an elevated LAP and a low a' velocity may be reflections of increased retrograde pulmonary vein flow.

PREDICTION OF CLINICAL OUTCOME. In our study, LA strain proved to be a good predictor of adverse cardiac events. For our population, LA systolic strain had the highest predictive accuracy for cardiovascular events and new onset of atrial fibrillation

(Figure 4). It outperformed LV GLS in predicting cardiac adverse events.

Interestingly, the LA strain values predicted by the lumped model comprising only LV strain and the LV/LA volume ratio and the directly measured LA strain showed no significant difference (Figure 5). This advocates that in subjects without pathology interfering with the volume exchange between LA and LV, a thorough assessment of LV function and conventional diastolic indices provides comparable information to LA strain measurements, although our limited sample size might prevent us from detecting meaningful differences.

Nevertheless, measuring LA strain in the clinical setting is likely of added value, because the parameter can integrate different information on LV systolic and diastolic function, chamber dimensions, and hemodynamics in a single measurement. Furthermore, it may be that LA strain provides particular added clinical value in the pathologies excluded from the study.

FEASIBILITY AND REPRODUCIBILITY. The feasibility of speckle-tracking-derived LA strain was reported to be good (6,19). In our study population, selected for high image quality to ensure a reliable tracking during the entire cardiac cycle, the feasibility for LV and LA strain was excellent (100%). This positive selection of good quality images may also have contributed to the good intra- and interobserver agreement of our measurement parameters, which are likely lower in clinical practice.

STUDY LIMITATIONS. Our study is a proof-of-concept study, and the patient population was highly selected for image quality and absence of pathology that could cause additional volume exchange between chambers. Although this selection allows an easier understanding of the interaction of hemodynamics and deformation, it limits the possibility to extrapolate our results to patients with additional pathology. Most importantly, we did not include patients with baseline atrial fibrillation in which LA strain has been previously shown to have the best prognostic value. In subjects with atrial fibrosis (such of those with atrial fibrillation), atrial stretching (distension) could be impaired and less dependent on the degree of LV function, which may account for a different relation between LV and LA strain. We used speckle-tracking software that had been optimized for LV strain analysis. We assume, however, that this impairment results only in random effects and not in bias toward higher or lower values.

CONCLUSIONS

Our data demonstrate that LA strain—in the absence of factors that interfere with the volume exchange between LA and LV—is in all phases of the cardiac cycle strongly determined by the deformation of the LV and the volume ratio of the 2 cavities.

Nevertheless, our results also showed that LA strain provides incremental prognostic information over LA volume and LV GLS, suggesting that the parameter integrates information of other echocardiographic parameters into a clinically valuable, single measurement.

The parameter might also have an added value in pathology that was excluded in this study.

CLINICAL IMPLICATIONS. First, we have shown that in subjects where the volume exchange between LA and LV is not disturbed by pathology, LA strain is strongly determined by the LV function and LV/LA volume ratio. A diversion from this relation could indicate the presence of atrial myopathy and help to identify patients who are at risk of developing atrial fibrillation and related complications (20).

Secondly, our results showed that LA strain is a good predictor of clinical events. The strength of LA strain lies in the simplicity of using a single measurement in clinical practice that integrates data on both LV and LA function and volumes. The newer developments of dedicated LA tracking tools may help LA strain to become clinically suitable by improving the feasibility and the robustness of LA strain measurements.

An understanding of the underlying hemodynamic mechanisms that drive LA strain changes, however, requires further research.

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ADDRESS FOR CORRESPONDENCE: Prof Dr Jens-Uwe Voigt, Department of Cardiovascular Sciences, KU Leuven, Department of Cardiovascular Diseases, University Hospitals Leuven, Herestraat 49, Leuven, Belgium. E-mail: jens-uwe.voigt@uzleuven.be.

PERSPECTIVES

COMPETENCY IN MEDICAL KNOWLEDGE: LA strain is mainly determined by LV function and the LV/LA volume ratio and only modulated by other factors, such as valve pathology, atrial myopathy, or filling pressures. Nonetheless, measuring LA strain is of clinical value,

because it integrates several parameters into a single, robust, and reproducible measurement.

TRANSLATIONAL OUTLOOK: Future studies with invasively measured left and right pressures and volumes might provide more insights into LA mechanics.

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APPENDIX For supplemental figures and tables, please see the online version of this paper.