

# Left atrial mechanical functions in patients with anterior myocardial infarction: a velocity vector imaging-based study

Selen Yurdakul, Saide Aytakin

Florence Nightingale Hospital, Cardiology Division, Istanbul, Turkey

## Abstract

**Background:** The contribution of the left atrium (LA) to left ventricular (LV) function increases in myocardial infarction (MI).

**Aim:** To evaluate LA function by using volume measurements and a novel strain imaging method, namely velocity vector imaging (VVI), in patients with acute anterior MI.

**Methods:** Twenty-four patients with previous anterior MI (aged  $63.8 \pm 4.2$  years, 56% men) and 30 healthy controls (aged  $60.7 \pm 5.3$  years, 60% men) were enrolled. LA volume measurements and VVI-derived LA peak systolic strain (S), strain rate (SRs), early diastolic (ESRd) and late diastolic strain rate (LSRd) were measured. LV diastolic function was analysed by pulsed wave-Doppler and tissue velocity imaging.

**Results:** LA maximum volume index was increased in patients compared to controls ( $28.83 \pm 7.2$  vs.  $19.72 \pm 6.27$  mL/m<sup>2</sup>,  $p = 0.0001$ ). As LA active emptying volume index and fraction were increased ( $6.16 \pm 0.7$  vs.  $5.46 \pm 0.99$  mL/m<sup>2</sup>,  $p = 0.009$  and  $22.16 \pm 3.07$  vs.  $16.78 \pm 2.93\%$ ,  $p = 0.0001$ , respectively), passive emptying volume index and fraction were decreased in the patient group ( $6.09 \pm 0.57$  vs.  $7.57 \pm 0.61$  mL/m<sup>2</sup>,  $p = 0.0001$  and  $45.76 \pm 6.86$  vs.  $56.45 \pm 5.36\%$ ,  $p = 0.0001$ , respectively). However, total emptying volume index of the LA was similar between the two groups. VVI-derived LA peak systolic S, SRs and ESRd were impaired in the patient group. LA LSRd was similar between the groups. LA active emptying fraction was positively correlated with LV diastolic dysfunction and negatively correlated with LV systolic dysfunction.

**Conclusions:** We demonstrated increased LA booster function and decreased LA conduit and reservoir functions in patients with prior anterior MI. Improvement in LA booster function correlated with the degree of LV systolic and diastolic dysfunction, suggesting a compensatory response of the LA.

**Key words:** anterior myocardial infarction, left atrium, velocity vector imaging

Kardiol Pol 2013; 71, 12: 1266–1272

## INTRODUCTION

The left atrium (LA) plays a major role in left ventricle (LV) performance. LA function is a surrogate marker of LV diastolic dysfunction. LA mechanical dysfunction occurs in LV systolic and diastolic dysfunction, coronary artery disease, myocardial infarction (MI), hypertension, aortic stenosis and cardiomyopathies. In the MI process, the contribution of the LA to LV function may increase if the cardiac myocytes are not affected by direct ischaemia. However, myocyte necrosis or ischaemia of the LA or significant LV systolic/diastolic dysfunction affects this booster effect. Assessment of LA size and

function provides prognostic data for the outcome of patients with MI or ischaemia [1].

Many studies have failed to yield a 'gold standard' method for evaluating LA functions. Despite being angle- and load-dependent, conventional echocardiographic and tissue velocity imaging (TVI) derived parameters are widely used to evaluate LA dysfunction. Two-dimensional (2D) strain imaging has emerged as a new technique for the assessment of subclinical myocardial dysfunction. It has the advantages of being independent from cardiac angle and tethering effects [2]. Velocity vector imaging (VVI) is a novel 2D strain imag-

### Address for correspondence:

Saide Aytakin, MD, Florence Nightingale Hospital, Abide-i Hurriyet street. No: 290, Postal code: 34381, Caglayan, Istanbul, Turkey, tel: +0090 212 224 49 50, fax: +0090212-2244982, e-mail: saideaytekin@gmail.com

Received: 17.09.2012 Accepted: 20.03.2013

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ing technique which may provide detailed information on regional cardiac functions [3].

In the present study, we evaluated changes in LA mechanical functions by using volume measurements and deformation parameters in patients with anterior MI. Additionally, we aimed to assess the clinical utility of VVI-derived strain imaging parameters for the quantitative analysis of LA function.

## METHODS

### *Study design and patient population*

The study included 24 patients (aged  $63.8 \pm 4.2$  years and 56% male) with previous anterior MI (defined as presentation with chest pain, echocardiographic changes and an increase in cardiac troponin T [4]), and 30 age- and sex-matched healthy subjects (aged  $60.7 \pm 5.3$  years and 60% male). All the patients were evaluated and included in the study six months after anterior MI. Exclusion criteria were: 1) previous percutaneous intervention or coronary artery bypass graft operation; 2) > 70% stenosis in coronary arteries other than left anterior descending artery (LAD); 3) functional capacity class III or IV according to the classification of the New York Heart Association (NYHA); 4) LV ejection fraction (EF) < 40%; 5) more than a mild degree of any valve disease; 6) atrial fibrillation; 7) atrioventricular conduction abnormalities; and 8) low quality echocardiographic image for TVI or VVI analysis.

The study protocol was approved by the local Ethics Committee of our institute, and a detailed written informed consent was obtained from each patient. The study was carried out according to the Declaration of Helsinki.

### *Echocardiographic measurements*

Patients underwent a transthoracic echocardiography (Siemens, Sequoia, C256; Mountainview, CA, USA) by using a 2.3–3.5 MHz transducer. LV end-diastolic and end-systolic diameters, interventricular septum and posterior wall thickness were measured from the parasternal long-axis view by using M-mode [5]. From an apical four-chamber view, LV stroke volume and EF were calculated using the modified Simpson's method [5]. Peak early (E) [m/s] and late diastolic (A) [m/s] transmitral flow velocities were also analysed. Deceleration time (DT) and isovolumic relaxation time (IVRT) were also calculated. LA horizontal systolic diameter was measured from an apical four-chamber view. Maximal (max), minimal (min) and pre-atrial LA volumes were determined by the biplane area-length method [6]. LA maximal volume (Vol max) was measured in ventricular end-systole just before mitral valve opening; minimal volume (Vol min) after mitral valve closure and pre-atrial volume (Vol pre) at the onset of the 'p' wave on electrocardiogram (ECG). LA volume measurements were indexed by dividing by body surface area. In addition, by using LA volumes, the following LA dynamic volumes were also calculated:

— LA reservoir volume (total emptying volume) (LA RV): Vol max-Vol min

- LA passive emptying volume (LA PEV): Vol max-Vol pre
- LA passive emptying fraction (LA PEF): LA passive emptying volume/Vol max
- LA conduit volume (LA CV): LV total stroke volume-LA reservoir volume
- LA active emptying volume (LA AEV): Vol pre-Vol min
- LA active emptying fraction (LA AEF): LA active emptying volume/Vol pre.

### *Tissue velocity imaging*

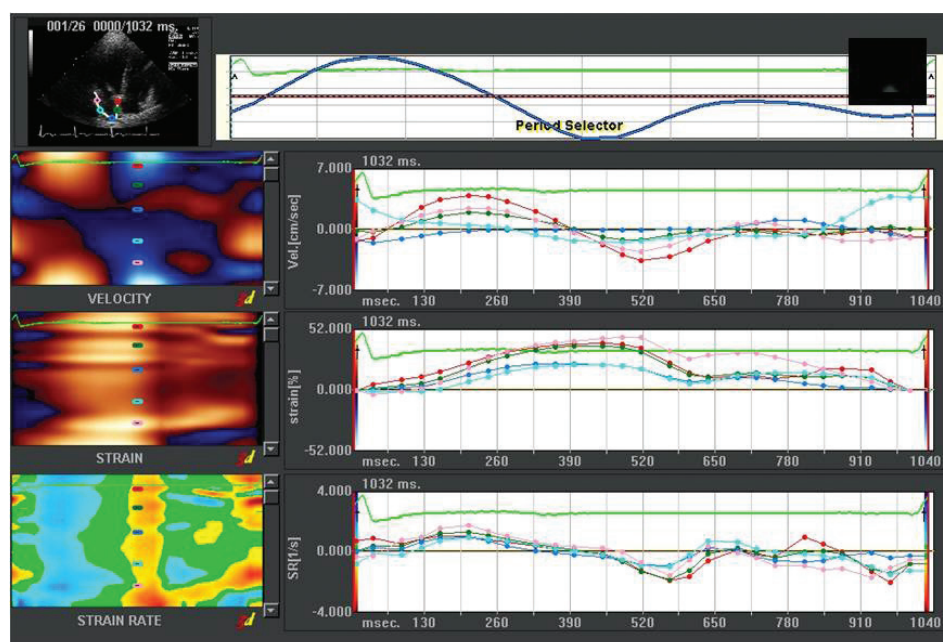
Guided by the apical four-chamber view, a 5 mm sample volume was placed just apical to the medial and lateral mitral annulus, identified using pulsed-wave TVI. Settings were adjusted for a frame rate of between 120 and 180 frame/s and a cine-loop of 3–5 consecutive heart beats was recorded. TVI-derived indices, namely peak early (e'), late diastolic (a'), and mitral annular velocities and e'/a' ratio were measured. Mitral E/e' ratio was calculated as previously described. All the measurements were calculated from three consecutive cycles, and the average of three measurements was recorded.

Aiming to evaluate atrial segmental function, we also recorded apical four- and two-chamber views of both the atria and determined sampling points for each basal, mid and superior atrial segment from septal, lateral, anterior and inferior walls of the LA. The peak atrial systolic ( $V_A$ ) velocity was measured from each segment, and an average value was calculated.

### *Velocity vector imaging*

Grey-scaled, 2D apical four- and two-chamber views of the LA were recorded. The frame rate was kept between 70 Hz and 100 Hz and a 1 beat acoustic capture function was used. We analysed LA deformation offline by using VVI software (Syngo VVI, Siemens Medical Solutions, Germany). After the endocardial borders had been defined manually by the user for LA, VVI software automatically tracked LA endocardial borders throughout the cardiac cycles and the velocity vectors were established (Fig. 1). Then we manually determined the sampling points for the basal, mid and superior atrial segments from septal, lateral, anterior and inferior walls for LA, during ventricular systole and at early and late diastole. Identifying the cardiac phases of systole and diastole, we measured the aortic valve closure time and the time interval from the beginning of QRS wave on ECG to peak E wave and to peak A wave, respectively, on Doppler images. Consequently, strain (S) and strain rate (SR) curves were automatically developed by the VVI software (Fig. 1).

'Strain' and 'strain rate' were defined as the change in the relative distance between localised tracked trace points, combined with the difference in the relative displacement of tissue motion behind the tracked points. Strain was defined as the instantaneous local trace lengthening or shortening, and systolic strain rate (SRs) as the rate of lengthening or shorten-



**Figure 1.** Colour M-mode echocardiography map and time curves showing left atrial velocity, strain and strain rate curves in an apical four-chamber view

**Table 1.** Clinical and demographic characteristics of the patient and control groups

	Patients with anterior myocardial infarction (n = 24)	Control group (n = 30)	P
Age [years]	63.8 ± 4.2	60.7 ± 5.3	0.48
Gender (% male)	56%	60%	0.56
Body surface area [m <sup>2</sup> ]	2.1 ± 0.2	2.0 ± 0.1	0.60
Heart rate [bpm]	74.2 ± 1.8	73.5 ± 1.4	0.82
Systolic blood pressure [mm Hg]	110.4 ± 19.6	105 ± 16.4	0.44
Diastolic blood pressure [mm Hg]	69.5 ± 12.2	65.2 ± 13.4	0.15

ing. Global S, SRs, early diastolic (ESRd) and late diastolic strain rate (LSRd) were calculated by averaging the data revealed from all segments analysed in LA. Peak systolic strain and SRs were used to evaluate atrial reservoir function, ESRd was used to determine atrial conduit function, and LSRd was used to determine atrial contractile function [7].

### Reproducibility

Intraobserver and interobserver variability for LA volumes and VVI measurements were assessed. For intraobserver variability, a sample of ten VVI measurements was randomly selected and examined by the same observer on two different days. For interobserver variability, a second observer blinded to the clinical information, and to the results of the first observer's results, examined the same ten measurements. Intraclass correlation coefficients for the same observer and different observers were calculated [8].

### Statistical analysis

Statistical data was performed with Statistical Package for the Social Sciences 16.0 (SPSS, Chicago, IL, USA) program. Results were expressed as means and standard deviations. Independent samples t-test was used for the comparison of the groups. Correlation analyses were derived by using Pearson analysis. The results were considered significant when the p value was less than 0.05.

## RESULTS

### Clinical characteristics and conventional echocardiographic data

Table 1 shows the clinical and demographic characteristics of the patients and the control group. Following presentation with acute anterior MI, 62.5% of patients (15/24) underwent primary revascularisation. None of the patients was readmitted for acute coronary syndrome or heart failure, and there were no deaths.

**Table 2.** Conventional echocardiographic parameters of the patient and control groups

	Patients with anterior MI (n = 24)	Control group (n = 30)	P
LVEDD [cm]	5.1 ± 0.3	4.8 ± 0.3	0.10
LVESD [cm]	3.6 ± 0.4	3.4 ± 0.4	0.15
IVS [cm]	1.1 ± 0.1	0.9 ± 0.1	0.001
Posterior wall [cm]	1.0 ± 0.2	1.0 ± 0.1	0.96
LVEF [%]	48.2 ± 3.2	66.4 ± 4.1	0.0001
Mitral E velocity [m/s]	0.64 ± 0.2	0.86 ± 0.1	0.0001
Mitral A velocity [m/s]	0.76 ± 0.2	0.72 ± 0.1	0.42
Deceleration time [ms]	203.1 ± 54	178.4 ± 15.2	0.05
IVRT [ms]	97.5 ± 35.8	85.7 ± 12.4	0.05
Mitral E/A ratio	0.8 ± 0.3	1.19 ± 0.1	0.0001

MI — myocardial infarction; LVEDD — left ventricular end diastolic diameter; LVESD — left ventricular end systolic diameter; IVS — interventricular septum; LVEF — left ventricular ejection fraction; IVRT — isovolumic relaxation time

**Table 3.** Left atrial dynamic and phasic volume measurements in the patient and control groups

	Patients with anterior MI (n = 24)	Control group (n = 30)	P
LA horizontal diameter [cm]	3.8 ± 0.3	3.6 ± 0.3	0.33
LA diameter index [cm/m <sup>2</sup> ]	1.91 ± 0.14	1.93 ± 0.18	0.78
LA maximum volume index [mL/m <sup>2</sup> ]	28.83 ± 7.20	19.72 ± 6.27	0.0001
LA preatrial volume index [mL/m <sup>2</sup> ]	15.02 ± 1.60	13.79 ± 2.54	0.06
LA minimum volume index [mL/m <sup>2</sup> ]	10.21 ± 1.07	8.33 ± 1.80	0.0001
LA conduit volume index [mL/m <sup>2</sup> ]	10.74 ± 1.91	8.75 ± 1.19	0.0001
LA AEVI [mL/m <sup>2</sup> ]	6.16 ± 0.70	5.46 ± 0.99	0.009
LA PEVI [mL/m <sup>2</sup> ]	6.09 ± 0.57	7.57 ± 0.61	0.0001
LA TEVI [mL/m <sup>2</sup> ]	12 ± 1.04	11.40 ± 1.52	0.13
LA PEF [%]	45.76 ± 6.86	56.45 ± 5.36	0.0001
LA AEF [%]	22.16 ± 3.07	16.78 ± 2.93	0.0001

MI — myocardial infarction; LA — left atrium; AEVI — active emptying volume index; PEVI — passive emptying volume index; TEVI — total emptying volume index; PEF — passive emptying fraction; AEF — active emptying fraction

LVEF was significantly impaired in patients with anterior MI compared to controls ( $p = 0.0001$ ). Mitral E wave velocity was decreased, E/A ratio was increased, while DT and IVRT were increased in the patient group, indicating LV diastolic dysfunction (Table 2).

#### Left atrium volumetric and functional analysis

LA max, LA min and LA pre volume index values were all increased in patients with prior anterior MI. LA PEV index and LA PEF were significantly decreased in the patient group, indicating impairment in the LA reservoir function. LA CV index was decreased, indicating impaired LA conduit function. In contrast, LA AEV index and LA AEF were increased in patients with anterior MI (Table 3).

#### Tissue velocity imaging

Left ventricular TVI-derived  $e'$ ,  $a'$ ,  $e'/a'$  ratio were significantly reduced, whereas  $E/e'$  ratio was increased in patients with

**Table 4.** Tissue velocity imaging derived diastolic measurements of the patient and control groups

	Patients with anterior MI (n = 24)	Control group (n = 30)	P
LV TVI- $e'$ [m/s]	0.09 ± 0.1	0.15 ± 0.1	0.02
LV TVI- $a'$ [m/s]	0.10 ± 0.001	0.13 ± 0.001	0.0001
LV TVI- $e'/a'$	0.85 ± 0.1	1.13 ± 0.01	0.0001
LV TVI-E/ $e'$	7.6 ± 1.5	4.4 ± 0.7	0.0001
LA $V_A$ [m/s]	0.12 ± 0.1	0.12 ± 0.1	0.88

MI — myocardial infarction; LV — left ventricle; TVI — tissue velocity imaging;  $e'$  — TVI-derived peak early diastolic velocity;  $a'$  — TVI-derived peak atrial velocity; E — mitral peak early velocity; LA  $V_A$  — left atrial late velocity

anterior MI, indicating LV diastolic dysfunction. Left atrial late velocity was similar in two groups (Table 4).

### Velocity vector imaging

A total of 1,152 segments were used to identify regional longitudinal function of the LA from apical four- and two-chamber views. Because the superior segments' velocity is lower and more difficult to identify compared to mid and basal segments, we were able to analyse 982 segments over 1,152 segments.

LA peak systolic strain and SRs were decreased in anterior MI patients ( $42.74 \pm 5.87$  to  $49.64 \pm 5.58$ ,  $p = 0.0001$  for S;  $1.63 \pm 0.24$  to  $2.07 \pm 0.22$ ,  $p = 0.0001$  for SR). This result supports impairment in LA reservoir functions in anterior MI. LA ESRd, which is known as a marker of LA conduit functions, was also decreased in the patient group ( $1.94 \pm 0.24$  to  $2.28 \pm 0.17$ ,  $p = 0.0001$ ). LA LSRd was slightly increased in patients with prior anterior MI, although the difference did not reach statistical significance ( $2.7 \pm 0.25$  to  $2.67 \pm 0.22$ ,  $p = 0.65$ ).

### Correlation analysis

Considering correlation analyses of LA deformation parameters with LV systolic and diastolic function, we demonstrated a significant positive correlation between LV E/e' and LA AEF ( $r = 0.52$ ,  $p = 0.0001$ ), and a significant negative correlation between LVEF and LA AEF ( $r = -0.469$ ,  $p = 0.001$ ). These results denominate an enhancement in LA booster function by compensatory mechanisms in anterior MI patients.

Additionally, we performed a correlation analysis of LA volume measurements with LA deformation parameters. Considering reservoir function, we observed a significant positive correlation between LA peak systolic S/SRs and LA PEV index/PEF ( $r = 0.51$ ,  $p = 0.0001$  for LA peak systolic S and LA PEV index;  $r = 0.57$ ,  $p = 0.0001$  for LA peak systolic S and LA PEF;  $r = 0.57$ ,  $p = 0.0001$  for LA peak systolic SRs and LA PEV index;  $r = 0.53$ ,  $p = 0.0001$  for LA peak systolic SRs and LA PEF). Regarding LA conduit function, we demonstrated a significant positive correlation between LA CV index and LA ESRd ( $r = 0.43$ ,  $p = 0.003$ ). For LA contractile function, we revealed a significant positive correlation between LA AEF and LA LSRd ( $r = 0.32$ ,  $p = 0.03$ ).

### Reproducibility

Intraclass correlations for intraobserver variability were good for LA volume measurements (LA max volume: 0.89, 95% confidence interval [CI] 0.75–0.96; LA pre volume: 0.88, 95% CI 0.70–0.95; LA minimal volume: 0.90, 95% CI 0.73–0.96. VVI-derived parameters (S: 0.85, 95% CI 0.65–0.90; SR: 0.80, 95% CI 0.60–0.95). The intraclass correlations for interobserver variability were also good for VVI-derived measurements (longitudinal strain: 0.88, 95% CI 0.78–0.94; SR: 0.85, 95% CI 0.76–0.98).

## DISCUSSION

In the present study, we evaluated the effect of anterior MI on LA regional wall motion by using a novel strain imaging method,

VVI. We observed that LA static volumes are enhanced in patients with prior anterior MI. In terms of LA dynamic volume measurements, LA CV and LA PEV were decreased, while LA AEF was increased. We also demonstrated that LA regional wall deformation is impaired in reservoir and conduit phases, whereas it is increased in the active contractile phase.

Although conventional echocardiography is widely used to evaluate LA functions, some limitations such as single plane assessment, dependence on LA haemodynamic and image quality deficits exist. A quantitative assessment of LA functions using invasive methods is clinically difficult because of simultaneous LA volume and pressure measuring requirements. TVI was introduced as an important step towards more reliable and accurate measures of cardiac regional functions [9]. Nevertheless, this method is still problematic due to cardiac rotational motion and tethering effects.

Doppler-based strain imaging, which has emerged as adjunctive to conventional TVI, is a quantitative technique that estimates myocardial contractility relatively independent of changes in load, cardiac rotational motion and tethering effects [10]. However, being 1D and angle dependent, Doppler-based strain imaging has limitations which have paved the way for 2D strain imaging techniques such as speckle tracking and VVI [11].

Increased LA static volumes may be the manifestation of LA remodelling which is the result of LV diastolic dysfunction due to anterior MI. LA size predicts prognosis in patients with heart disease [12], and LA volume is an important marker of diastolic dysfunction and LV filling pressure [13]. Myocardial ischaemia induces inflammatory and neurohormonal changes leading to diastolic dysfunction [14]. LV filling pressure increases following acute MI, with increases in LA wall tension and LA pressure. This is a compensatory mechanism of the LA to overcome reduced compliance of the LV.

Passive stretching of the LA wall during systole represents LA reservoir function, which is influenced by LA relaxation [15], LV contraction through the descent of the base during systole [16], and LA chamber stiffness [17]. In the present study, we demonstrated significant reductions in LA systolic strain and strain rate measurements, consistent with impairment in LA reservoir function. LA enlargement in patients with MI increases passive emptying, although acute ischaemia reduces LV longitudinal fibre shortening and base descent which results in a reduction in LA reservoir filling. Additionally, due to the increase in LA pressure and decrease in LV compliance, LA reservoir function deteriorates. Boyd et al. [18] studied changes in LA volumes, following non-ST elevation myocardial infarction (NSTEMI). They revealed that, within 48 h of presentation, LA total emptying volume decreased, indicating a reduction in LA reservoir function.

The conduit phase reflects passive blood flow from the pulmonary veins down a pressure gradient initiated by LV relaxation. Consequent to a decrease in compliance, LA CV,

which represents early diastolic filling, reduces in acute MI. In addition, we found that LA ESRd, which is known as an important determinant of LV early diastolic filling, was significantly decreased. Jarnert et al. [19] studied LA deformation by using VVI in order to detect LV diastolic dysfunction in patients with type 2 diabetes mellitus. They demonstrated that LA ESRd was significantly impaired in patients with LV diastolic dysfunction compared to the control group. Similar to the findings of our study, Boyd et al. [18] demonstrated a significant impairment in LA CV in patients who presented with NSTEMI. In our study, impairment of LA early diastolic phase deformation is supported by a decrease in mitral  $e'$  in TVI measurements.

Atrial contractile function is determined by preload, afterload and LA contractility [17]. In the active contractile phase, the LA contracts and projects blood into the LV in late diastole. When the LV is affected by ischaemia, LV end diastolic pressure, transmitral pressure gradient and LA afterload increase. Enhanced LA wall stretch results in an increase of the contractile force with the effect of the Frank-Starling law on the LA myocardium. A marked increase in LA AEF and AEF in the patient group supports this hypothesis. Moreover, we demonstrated a significant positive correlation between LV  $E/e'$  and LA AEF, expressing the relationship between enhanced contractility of the LA as a compensatory mechanism and LV diastolic function. Regarding VVI analysis, we revealed that LA late diastolic strain rate was slightly enhanced, but this increase was not significant. We assume that this result may be attributed to the limited number of patients in the study group. Our results are supported by some previous studies [20, 21]. In an experimental study, Mu et al. [22] addressed this issue by studying peak SR values of the LA walls during all phases of the LA cycle, in 11 healthy dogs, during acute LV ischaemia. Similar to our study, they observed an enhanced wall motion of the LA, after regional acute ischaemia.

VVI-based strain imaging provides measurements which describe subtle changes in myocardial tissue motion. These parameters are complementary to conventional echocardiographic indices and correlated with LV systolic and diastolic function. Based on results from current data, we suggest that assessment of LA volumes and deformation provides prognostic information about LV diastolic function. In the present study, we demonstrated that VVI is a novel strain imaging modality, which is highly available for the evaluation of changes in LA myocardial contractility, in clinical use.

### Limitations and strengths

There have been a limited number of studies analysing deformation in LA mechanical functions in myocardial ischaemia or MI. The major limitation of our study is the limited number of patients in our study group. Another limitation is that we were not able to compare our results to a gold standard method for the evaluation of LA function. The chief strength of this

manuscript is detailed LA function evaluations by conventional indices accompanied by a novel imaging technique, namely VVI-derived strain imaging, which may be used in the assessment of subclinical cardiac dysfunction. Further studies are warranted to determine the efficiency of VVI imaging modality on atrial functions.

### CONCLUSIONS

In the present study, we demonstrated an increase in LA booster function and decreases in conduit and reservoir functions in patients with previous anterior MI. Improvement in LA booster function is correlated with the degree of LV diastolic and systolic dysfunction, suggesting a compensatory enhanced contractility of the LA myocardium. We suggest that LA deformation indices may be used as adjunctive parameters in evaluating LA functions.

**Conflict of interest:** none declared

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## Czynność mechaniczna lewego przedsionka u chorych, którzy przebyli zawał ściany przedniej serca: analiza na podstawie obrazowania wektorów prędkości

Selen Yurdakul, Saide Aytekin

Florence Nightingale Hospital, Cardiology Division, Istanbul, Turcja

### Streszczenie

**Wstęp:** Wpływ czynności lewego przedsionka (LA) na czynność lewej komory (LV) zwiększa się w przypadku wystąpienia zawału serca (MI).

**Cel:** Celem badania była ocena czynności LA na podstawie pomiarów parametrów objętościowych i nowych metod obrazowania odkształceń mięśnia sercowego, obrazowania wektorów prędkości (VVI) u chorych z ostrym MI ściany przedniej.

**Metody:** Do badania włączono 24 chorych, którzy przebyli MI ściany przedniej (wiek:  $63,8 \pm 4,2$  roku, 56% mężczyzn) i 30 zdrowych osób stanowiących grupę kontrolną (wiek:  $60,7 \pm 5,3$  roku, 60% mężczyzn). Zmierzono parametry objętościowe LA i wykorzystano metodę VVI do oceny maksymalnego odkształcenia (*S*, *strain*) LA w czasie skurczu, tempa odkształcenia w czasie skurczu (SRs, *strain rate*), a także wczesnorozkurczowe (ESRd) i późnorozkurczowe (LSRd) tempo odkształcenia. Czynność rozkurczową LV oceniano na podstawie obrazowania metodą tkankowej echokardiografii dopplerowskiej.

**Wyniki:** U pacjentów po MI wskaźnik maksymalnej objętości LA był większy niż w grupie kontrolnej ( $28,83 \pm 7,2$  vs.  $19,72 \pm 6,27$  ml/m<sup>2</sup>;  $p = 0,0001$ ). Ponieważ objętość i frakcja aktywnego opróżniania LA były zwiększone (odpowiednio  $6,16 \pm 0,7$  vs.  $5,46 \pm 0,99$  ml/m<sup>2</sup>;  $p = 0,009$  i  $22,16 \pm 3,07$  vs.  $16,78 \pm 2,93\%$ ;  $p = 0,0001$ ), to objętość i frakcja biernego opróżniania LA były zmniejszone (odpowiednio  $6,09 \pm 0,57$  vs.  $7,57 \pm 0,61$  ml/m<sup>2</sup>;  $p = 0,0001$  i  $45,76 \pm 6,86$  vs.  $56,45 \pm 5,36\%$ ;  $p = 0,0001$ ). Jednak całkowity wskaźnik opróżniania LA był podobny w obu grupach. Maksymalne odkształcenie w czasie skurczu, SRs i ESRd były mniejsze u chorych, którzy przebyli MI w porównaniu z grupą kontrolną. Wartości LSRd LA były podobne w obu grupach. Stwierdzono dodatnią korelację między frakcją aktywnego opróżniania LA a dysfunkcją rozkurczową LV oraz ujemną korelację między tą frakcją a dysfunkcją skurczową LV.

**Wnioski:** Wykazano, że u pacjentów, którzy przebyli MI, nastąpiło zwiększenie wspomagającej czynności LA i ograniczenie roli LA jako drogi przepływu oraz zbiornika. Poprawa wspomagającej czynności LA korelowała ze stopniem dysfunkcji skurczowej i rozkurczowej LV, co wskazuje na kompensacyjną odpowiedź LA.

**Słowa kluczowe:** zawał ściany przedniej serca, lewy przedsionek, obrazowanie wektorów prędkości

Kardiologia 2013; 71, 12: 1266–1272

### Adres do korespondencji:

Saide Aytekin, MD, Florence Nightingale Hospital, Abide-i Hurriyet street. No: 290, Postal code: 34381, Caglayan, Istanbul, Turkey, tel: +0090 212 224 49 50, faks: +0090212-2244982, e-mail: saideaytekin@gmail.com

Praca wpłynęła: 17.09.2012 r. Zaakceptowana do druku: 20.03.2013 r.