Mechanical and substrate abnormalities of the left atrium assessed by 3-dimensional speckle-tracking echocardiography and electroanatomic mapping system in patients with paroxysmal atrial fibrillation

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BACKGROUND Left atrial (LA) remodeling progresses to electrical remodeling, contractile remodeling, and subsequently structural remodeling. Little is known about the relationship between LA electrical and anatomical remodeling and LA mechanical function.

OBJECTIVES We aimed to clarify the relationship between LA mechanical function using 3-dimensional speckle-tracking echocardiography (3D-STE) and LA electrical remodeling using an electroanatomic mapping system (CARTO 3) and to estimate atrial fibrillation (AF) substrate in patients with paroxysmal AF (PAF).

METHODS A total of 52 patients with PAF (41 (79%) men; mean age 61 ± 11 years) undergoing their initial pulmonary vein isolation (PVI) were examined. The standard deviation of the time to peak strain in each LA segment (%SD-TPS) was analyzed as an index of LA dyssynchrony using 3D-STE before PVI. Contact LA bipolar voltage and activation maps were constructed during sinus rhythm before PVI using CARTO 3. The LA total activation time was measured and low-voltage zones (LVZs) were determined with a local bipolar electrogram amplitude of <0.5 mV. The patients were divided into those with an LVZ (LVZ group; n = 23) and those without an LVZ (non-LVZ group; n = 29).

RESULTS The %SD-TPS was significantly higher (14.1 ± 5.7 vs 8.0 ± 5.1; $P = .0002$) in the LVZ group than in the non-LVZ group and was an independent determinant of the LVZ (odds ratio 1.21; 95% confidence interval 1.04–1.49; $P = .01$). In addition, the LA total activation time was weakly correlated with the %SD-TPS.

CONCLUSION LA dyssynchrony and conduction delay exist in patients with PAF. The 3D-STE enabled noninvasive estimation of LA electrical remodeling and AF substrate.

KEYWORDS Left atrium; Atrial fibrillation; Electroanatomic mapping; Speckle-tracking echocardiography; Conduction delay; Dyssynchrony; Remodeling

ABBREVIATIONS **2D** = 2-dimensional; **3D** = 3-dimensional; **%SD-TPS** = %standard deviation of the time to peak strain in each left atrial segment; **AF** = atrial fibrillation; **CTI** = cavotricuspid isthmus; **ICC** = intraclass correlation coefficient; **LA** = left atrium/atrial; **LV** = left ventricular; **LVZ** = low-voltage zone; **PAF** = paroxysmal atrial fibrillation; **PV** = pulmonary vein; **PVI** = pulmonary vein isolation; **RA** = right atrium/atrial; **RFCA** = radiofrequency catheter ablation; **SR** = sinus rhythm; **STE** = speckle-tracking echocardiography; **SVC** = superior vena cava; **TAT** = total activation time

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Introduction

The pathogenesis of atrial fibrillation (AF) is multifactorial. Sometimes, AF is accompanied by various pathological conditions; however, it may also occur in normal hearts as lone AF.[1] Reportedly, most of the paroxysmal AF (PAF) is triggered by ectopy from pulmonary veins (PVs) and partially from non-PV sites.[2] Presently, AF usually requires a trigger for initiation and a vulnerable electrophysiological and/or anatomical substrate for maintenance.[3] Allessie et al[4] reported that left atrial (LA) remodeling progressed in a series of electrical remodeling, subsequent contractile remodeling, and finally structural remodeling. Previous research indicated that LA electrophysiological disorders in AF were represented by low-voltage zones (LVZs) obtained by contact bipolar voltage mapping during sinus rhythm (SR)[5] and that LVZs correlated with local conduction delays.[6]

The recent 3-dimensional speckle-tracking echocardiography (3D-STE) technology allows us to obtain reliable data

This work was not supported by any external funding. **Address reprint requests and correspondence:** Dr Yukiko Nakano, Department of Cardiovascular Medicine, Hiroshima University Graduate School of Biomedical and Health Sciences, 1-2-3 Kasumi, Minami-ku, Hiroshima 734-8551, Japan. E-mail address: nakanoy@hiroshima-u.ac.jp.

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to calculate global LA mechanical function and synchrony.[7,8] Little is known about the relationship between LA electrical and anatomical remodeling and LA mechanical function. We aimed to investigate the relationship between LA electrical remodeling and LA mechanical function and to estimate AF substrate in patients with PAF.

Methods

Study population

We retrospectively enrolled 52 consecutive patients with PAF (41 men; mean age 61 ± 11 years) who had drug-refractory symptomatic AF, underwent the LA area strain analysis, and were scheduled for the initial radiofrequency catheter ablation (RFCA) of AF at Hiroshima University Graduate School of Biomedical and Health Sciences. Those with concomitant coronary artery disease, cardiomyopathy, heart failure, pacemaker implantations, valvular disease, or inadequate acoustic windows were excluded. Their PAF duration was 49 ± 67 months, and the clinical characteristics are summarized in Table 1. Antiarrhythmic drugs including β-blockers were discontinued at least 5 half-lives before the procedure. No patients were taking amiodarone. This study was approved by the ethics committee of Hiroshima University Graduate School of Biomedical and Health Sciences.

Table 1 Clinical characteristics and echocardiographic parameters of patients (N = 52)

Clinical characteristics	
Age (y)	61 ± 11
Sex: male	41 (79)
Body mass index (kg/m^2)	23.7 ± 3.1
Hypertension	28 (54)
Diabetes mellitus	2 (4)
PAF duration (mo)	49 ± 67
Class I AADs	20 (38)
Class II AADs	14 (27)
Class III AADs	0 (0)
Class IV AADs	13 (25)
Conventional echocardiographic parameters	
LA dimension (mm)	35.5 ± 4.9
LV end-diastolic dimension (mm)	48.5 ± 4.5
LV end-systolic dimension (mm)	31.6 ± 3.2
Interventricular septum thickness (mm)	8.7 ± 1.2
Posterior wall thickness (mm)	8.7 ± 1.1
LV mass index (g/m^2)	84.8 ± 19.0
LV ejection fraction (%)	63.6 ± 3.2
E wave (m/s)	66.2 ± 16.1
A wave (m/s)	62.4 ± 20.2
E/A ratio	1.2 ± 0.5
LAVI (mL/m^2)	33.6 ± 8.1
Doppler tissue imaging parameters	
e′ wave (cm/s)	11.0 ± 3.0
a′ wave (cm/s)	10.7 ± 3.1
E/e′ ratio	6.5 ± 2.4
3D strain echocardiographic parameters	
GPS	48.5 ± 19.8
%SD-TPS	10.7 ± 6.1

Values are presented as mean ± SD or n (%).

3D = 3-dimensioanl; %SD-TPS = % standard deviation of time to peak strain; AAD = antiarrhythmic drug; GPS = global peak strain; LA = left atrial; LAVI = left atrial volume index;

LV = left ventricular; PAF = paroxysmal atrial fibrillation.

Echocardiographic measurements

The transthoracic echocardiographic examinations, including 2-dimensional (2D), M-mode, pulsed wave, continuous wave, color flow, and 3D-STE, were performed using an Aplio Artida ultrasound system (Toshiba, Tokyo, Japan). We used the PST-30SBT (1–5 MHz) transducer for 2D imaging and the PST-25SX matrix array transducer for 3-dimensional (3D) imaging.

All images were obtained in the parasternal short- and long-axis views or the apical 2- and 4-chamber views according to the American Society of Echocardiography guidelines.[9] Experienced echocardiographers conducted all echocardiographic examinations and analyzed echocardiographic parameters.

LA area strain analysis

For 3D-STE analysis, a volumetric image of LA from the apical view was obtained and stored on the 3D wall motion tracking software (Toshiba, Tokyo, Japan).

LA area strain analysis was performed according to the methods described by other researchers.[7,8] By manually tracing the LA endocardial border in both 4- and 2-chamber views during the left ventricular (LV) end-diastolic phase (minimum LA cavity area), the software automatically tracked endocardial contours and divided the LA image into 16 segments. Area strain was obtained as a ratio of endocardial area change during a cardiac cycle, and the area strain curves of each segment and global strain curve were obtained. We defined the first peak value of the LA global area strain curve as global peak strain, representing the total amount of LA reservoir function. To quantify the dispersed motion between each LA segment (dyssynchrony), we used the standard deviation of the time to peak of each area strain curve and expressed it as the percentage of the interbeat (R-R) interval (%SD-TPS) (Figure 1).

All measurements were taken during SR before RFCA. The independent investigators who analyzed clinical information performed echocardiographic examinations. We examined intraclass correlation coefficients (ICCs) for %SD-TPS measurements in the randomly selected 15 patients to check the reproducibility. ICC(2, 1) was .86, and ICC(1, 1) was .92.

Detection of the LVZ and measurement of LA total activation time

A 64-slice multidetector computed tomography (LightSpeed VCT, GE Healthcare, Little Chalfont, Buckinghamshire, UK) was performed before the procedure to evaluate LA anatomy. Briefly, a 6-F 16-pole catheter (Irvine Biomedical, Irvine, CA) was used, with the distal poles placed within the coronary sinus and proximal poles located from the superior vena cava (SVC) to the superior right atrium (RA) via the right internal jugular vein.

Before RFCA, a 3D anatomical contact voltage map and an activation map of LA were constructed during SR with a 3.5-mm irrigated-tip electrode catheter (ThermoCool, Biosense Webster Inc, Diamond Bar, CA) in a point-by-point

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