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Predictive value of atrial electromechanical delay for atrial fibrillation recurrence

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Abstract

Background: *We investigated the predictive value of atrial electromechanical delay (AEMD) for recurrence of atrial fibrillation (AF) at 1-month after cardioversion.*

Methods: *Seventy-seven patients with persistent AF were evaluated and finally 50 patients (12 men, 38 women) were included. All patients underwent transthoracic electrical DC cardioversion under amiodarone treatment. AEMD was measured as the time interval from the onset of the P wave on electrogram (ECG) to the beginning of late diastolic wave (Am) from the ventricular annulus and atrial walls on tissue Doppler imaging, in the apical 4-chamber view 24 h after cardiversion. P wave maximum-duration (P_{max}), P wave minimum-duration (P_{min}) and P wave dispersion-duration (P_{dis}) were calculated on the 12-lead ECG at 24-h postcardioversion. We followed the heart rate and rhythm by 12-lead ECG at 24-h, 1-week and 1-month.*

Results: *At 1-month follow-up after cardioversion, 28 (56%) patients were in sinus rhythm (SR), whereas 22 (44%) patients reverted to AF. The AEMD durations were longer in AF group than SR group ($p < 0.001$) and were significantly correlated with P_{max} and P_{dis} ($p < 0.001$ for both). For AF recurrence; duration of AF, left atrial (LA) diameter, maximum LA volume index, mitral A velocity and LA lateral AEMD were significant parameters in univariate-analysis, however LA lateral AEMD was the only significant parameter in multivariate-analysis (OR: 1.46; 95% CI 1.02–2.11; $p = 0.03$).*

Conclusions: *Our results suggest that AEMD is associated with an increased risk of recurrence of AF within 1-month. These data may have implications for the identification of patients who are most likely to experience substantial benefit from cardiversion therapy for AF.* (Cardiol J 2013; 20, 6: 639–647)

Key words: atrial fibrillation, cardioversion, electromechanical delay

Introduction

Atrial fibrillation (AF), is the most common sustained cardiac arrhythmia, affecting approximately 1–2% of the population and increasing their risk of stroke 5 times more than general population [1]. AF is also associated with increased mortality, heart failure, increased hospitalization, and decreased quality of life and exercise capacity [1].

In general, there are 2 treatment strategies for AF patients; rate control or rhythm control [1]. Even though rate control is acceptable for initial

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therapy, rhythm control is generally preferred over rate control due to offering better symptomatic reliefand improved quality of life [1]. Conversion of AF to sinus rhythm (SR) prevents electrical and structural remodeling in the left atrium (LA) [1]. Moreover, restored atrial contraction increases left ventricular (LV) filling and contributes to hemodynamic improvement [2]. However, despite the widespread use of antiarrhythmic drugs, AF recurs in substantial proportion of patients achieving SR after cardioversion. For this reason, prediction of the recurrence of AF after cardioversion would be helpful for tailoring the treatment strategies.

The atrial electromechanic delay (AEMD) is the time interval from the onset of P wave on surface electrocardiography (ECG) to the beginning of the late diastolic wave on tissue Doppler (Am wave) [3, 4]. The delay between the electrical stimulation and mechanic contraction results from structural changes in the atria. These structural changes also lead to a prolongation in P wave duration. P wave duration > 120 ms is considered abnormal [5, 6].

We aimed to investigate the predictive value of postcardioversion AEMD durations for the recurrence of AF at 1 month after electrical cardioversion in patients with persistent AF.

Methods

Patient selection

In this study, we enrolled 70 patients with persistent AF presented to cardiology clinic between September 2010 and June 2011. Patients with contraindication to anticoagulation, NYHA class III and IV heart failure, prosthetic heart valve or severe native valvular heart disease, thrombi in the LA, sick sinus syndrome and LA diameter > 50 mm were excluded from the study. Fifteen patients who met any of the exclusion criteria were excluded and 55 patients included to the study. The ethical committee of Sevket Yilmaz Training and Research Hospital approved the study protocol and informed consent was obtained from each patient.

All patients underwent electrical cardioversion. SR could not be restored in 5 cases so they were excluded. In the remaining 50 patients, cardioversion was successful and those patients were followed up.

Study patients

Patients were questioned about demographic features and physical examinations were performed. After admission to coronary care unit, peripheral venous blood samples were drawn for analyzing the complete blood count and main biochemical markers. A 12-lead surface ECG was obtained before and after cardioversion.

Body mass indices (BMI) were calculated according to the following formula: weight [kg]/square of the height [m^2] and body surface area was calculated as weight $^{(0.425)}$ [kg] × height $^{(0.725)}$ [cm] × × 0.007184.

LV mass was calculated using the formula; 0.8 × 1.04 × [(LV end diastolic diameter + posterior wall thickness + interventricular septum thickness)^3 – (LV end diastolic diameter)^3] + 0.6 [7].

Transthoracic echocardiograpy

All patients underwent transthoracic echocardiogram (TTE) before cardioversion according to the recommendations of the American Society of Echocardiography. TTE was performed using Vivid 7 Pro TTE echocardiography system with 3.5 MHz probe on the lateral decubitus position.

LV ejection fraction (LVEF) was calculated using the Teichholz formula from the parasternal long-axis view using M-mode and LA volume was measured using modified biplane area-length method [8, 9].

In the apical 4-chamber view, a pulsed wave Doppler (PWD) sample volume (3 mm) is placed between the mitral leaflet tips and peak E wave velocity and E wave deceleration time is measured. The interval from the beginning of the R wave on electrogram to the beginning of the E wave on PWD is measured and defined as T_E. Tissue Doppler imaging (TDI) was performed at the septal and lateral mitral annulus in apical 4-chamber view. Gain adjustments were minimized, TDI filter and Nyquist limits adjusted to (16–20 cm/s) for getting clear tissue signals. Peak systolic (Sm) and early diastolic (Em) annular velocities were measured. The time between the beginning of the R wave on electrogram and beginning of the Em velocity on TDI was measured and defined as T_{Em}. The difference between T_{Em} and T_E intervals was calculated by subtracting T_E from T_{Em} and defined as $T_{Em–E}$.

Transesophageal echocardiography

Transesophageal echocardiogram (TEE) was performed on all patients with 6 MHz TEE probe to exclude atrial thrombus prior to cardioversion. Left atrial appendage filling and emptying velocities were also recorded with PWD.

Electrical cardioversion

Anticoagulation with heparin was given by continuous intravenous infusion (17 U/kg) to all

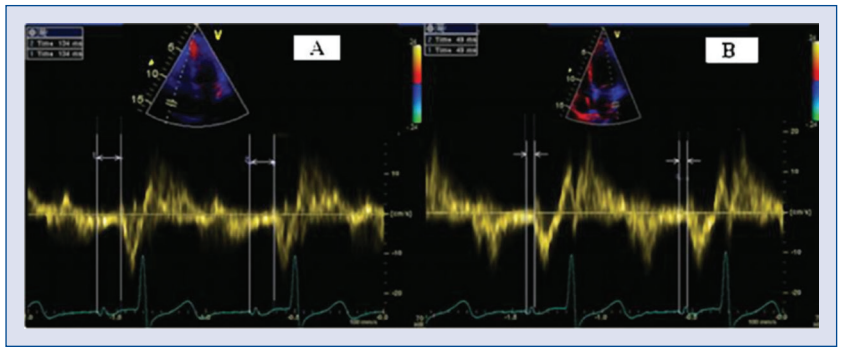


Figure 1. A. Left atrium lateral atrial electromechanical delay (AEMD) duration; **B.** Right atrium lateral AEMD duration.

patients before cardioversion and its dose adjusted to an activated partial thromboplastin time of 1.5–2 times normal. All antiarrhythmic drugs including digoxin were stopped before cardioversion. Amiodarone infusion started (5 mg/kg IV loading dose infused over 10 min, followed by 10–15 mg/kg/h infusion over 24 h) to patients who did not have intracardiac thrombus on TTE and TEE studies. Patients were sedated with intravenous midazolam during the procedure. Transthoracic electrical cardioversion was performed with delivery of synchronized biphasic DC shocks of 150, 200 and 270 J in the intensive care unit. Cardioversion was considered successful if atrial-P waves were unmistakably identified ≥ 1 min after the shock. Patients achieving SR received warfarin in a dose intended to achieve therapeutic international normalized ratio (INR) of 2.0 to 3.0 after the procedure. Oral amiodarone was continued 600 mg/kg for the first 2 weeks and then 200 mg/kg the following 2 weeks. Patients evaluated at 1, 2 and 4 weeks after the procedure by physical exam, ECG and INR measurements.

Electromechanical time interval measurements

The time interval from the onset of P wave on surface ECG to the beginning of late diastolic wave (Am wave) on TDI, is named as AEMD. AEMD interval was obtained on apical 4 chamber view from the lateral mitral annulus (mitral lateral AEMD), medial mitral annulus (mitral medial AEMD), lateral tricuspid annulus (tricuspid lateral AEMD), lateral LA wall (LA lateral AEMD), interatrial septum (LA medial AEMD) and lateral right atrium (RA) wall (RA lateral AEMD) (Fig. 1). The difference between mitral lateral AEMD and tricuspid lateral AEMD (mitral lateral AEMD – tricuspid lateral AEMD) and, LA lateral AEMD and RA lateral AEMD (LA lateral AEMD – RA lateral AEMD) was defined as inter-atrial electromechanical delay (inter-AEMD). The difference between mitral lateral AEMD and mitral medial AEMD (mitral lateral AEMD – mitral medial AEMD) and, LA lateral AEMD and LA medial AEMD (LA lateral AEMD – LA medial AEMD) was defined as left intra-electromechanical delay (intra-$AEMD_{LEFT}$). The difference between mitral medial AEMD and tricuspid lateral AEMD (mitral medial AEMD – tricuspid lateral AEMD) and, LA medial AEMD and RA lateral AEMD (LA medial AEMD – RA lateral AEMD) was defined as right intra-electromechanical delay (intra-$AEMD_{RIGHT}$).

P-wave duration and P-wave dispersion measurement

A 12 lead ECG (filter range, 0.15 to 100 Hz; AC filter, 100 Hz; low pass filter, 150 Hz; 25 mm/s; 10 mm/mV) was obtained from all patients. ECGs were transferred to computer and measurements were made using AutoCAD 2007 software program. The onset of P-wave was defined as the point of the first visible upward departure of the trace from the bottom of the baseline. The return to the baseline of the bottom of the trace in wave was considered to be the end of the P-wave. The difference between P-wave maximum (P_{max}) and P wave minimum (P_{min}) durations was defined as P-wave dispersion (P_{dis}).

Data analysis

The data were analyzed using the SPSS 10.0 statistics package (SPSS Inc, Chicago, Ill, USA). Continuous variables are reported as means ± standard deviation and categorical variables are reported as percentages. Student's t test was used for comparison of normal distributed variables and Mann-Whitney U test was used for non-normally distributed variables. Categorical variables were

Table 1. Baseline characteristics of patients in group 1 and group 2.

	Group 1: SR at follow-up (n = 28)	Group 2: AF at follow-up (n = 22)	P
Age [years]	61.67 ± 6.89	61.27 ± 7.88	0.84
Female gender	22 (78.6%)	16(72.7%)	0.63
AF duration [months]	6.50 ± 2.44	9.31 ± 1.98	< 0.001
Baseline heart rate [bpm]	112.89 ± 14.28	114.40 ± 20.87	0.76
Body mass index [kg/m^2]	32.77 ± 6.99	30.94 ± 5.46	0.31
Body surface area [m^2]	1.88 ± 0.17	1.85 ± 0.21	0.59
Blood pressure [mm Hg]			
Systolic	129.21 ± 9.11	128.77 ± 9.77	0.87
Diastolic	80.57 ± 7.32	79.22 ± 6.78	0.50
Hemoglobin [g/dL]	13.22 ± 1.28	13.31 ± 1.38	0.81
Creatinine [mg/mL]	0.77 ± 0.28	0.80 ± 0.25	0.72
Cardiversion energy [J]	235.0 ± 35.64	247.72 ± 33.37	0.20
Comorbidities:			
Diabetes mellitus	7 (25%)	1 (4.5%)	0.06
Hypertension	21 (75%)	15 (68.2%)	0.59
COPD	4 (14.3%)	3 (13.6%)	0.94
Coronary artery disease	4 (14.3%)	3 (13.6%)	0.94
Smoker	3 (10.7%)	2 (9.1%)	0.84
Medications:			
Beta-blocker	19 (67.9%)	17 (77.3%)	0.46
Calcium channel blocker	9 (32.1%)	5 (22.7%)	0.46
ACE inhibitor	12 (42.9%)	7 (31.8%)	0.42
ARB	6 (21.4%)	7 (31.8%)	0.40
Diuretics	3 (10.7%)	4 (18.2%)	0.45

AF — atrial fibrillation; SR — sinus rhythm; COPD — chronic obstructive pulmonary disease; ACE — angiotensin converting enzyme; ARB — angiotensin receptor blocker

compared by the χ^2 test or Fisher's exact test as appropriate. Univariate and multivariate logistic regression analyses were used to determine significant predictors of AF recurrence following cardioversion. Relationship between AEMD durations and P_{max}, P_{min} and P_{dis} were calculated using Pearson's correlation tests. The sensitivity and specificity of AEMD duration to predict AF recurrence was analyzed by receiver operating characteristic (ROC) analysis. P values less than 0.05 were considered significant.

Results

Fifty patients who were successfully cardioverted to SR were followed for 1 month. We divided study population according to their rhythm at the end of 1 month follow-up period. Group 1 consisted of 28 patients who were in SR and group 2 consisted of 22 patients who reverted to AF at the end of the follow-up period. Analysis of baseline characteristics revealed that patients in group 2 had longer duration of AF than group 1 (9.3 ± 2 vs. 6.5 ± 2 months; $p < 0.001$), the other parameters were similar (Table 1).

Precardioversion echocardiographic analysis of the study population showed significantly larger LA diameter ($p < 0.001$), LA maximum volume ($p < 0.001$), LA maximum volume index ($p < 0.001$), and LA minimum volume ($p = 0.001$) in group 2 than in group 1. Pulmonary artery pressure (PAP) was also found to be higher in patients in group 2 ($p = 0.01$) (Table 2). The tissue Doppler parameters and T_{Em-E} intervals measured from mitral and tricuspid annulus were not significant between the groups.

The analysis of echocardiographic data during 24 h follow-up after cardioversion revealed that LA diameter, LA volume index and LA minimum volume were larger in patients in group 2 ($p < 0.001$ for all). The follow-up PAP value was similar between two groups. Group 2 had lower mitral A velocities ($p = 0.005$) and higher mitral E/A ratios ($p = 0.01$) as compared to patients in group 1 (Table 3). The

Table 2. Comparison of precardioversion echocardiographic data in Group 1 and Group 2.

	Group 1: SR	Group 2: AF	P
LV end-systolic diameter [mm]	31.64 ± 4.27	31.72 ± 4.67	0.94
LV end-diastolic diameter [mm]	47.85 ± 3.70	47.72 ± 3.04	0.89
LV EF [%]	60.92 ± 5.94	58.31 ± 7.24	0.16
LV mass [g]	188.37 ± 28.49	184.38 ± 20.80	0.58
LA diameter [cm]	40.25 ± 2.84	44.68 ± 2.33	< 0.001
LA maximum volume [mL]	59.75 ± 15.14	78.69 ± 19.04	< 0.001
LA maximum volume index	31.76 ± 8.03	42.75 ± 11.14	< 0.001
LA minimum volume [mL]	29.59 ± 10.22	40.16 ± 11.84	0.001
Pulmonary artery pressure [mm Hg]	34.42 ± 9.06	41.86 ± 10.85	0.01
Mitral E velocity [m/s]	1.02 ± 0.24	1.09 ± 0.30	0.40
Mitral lateral Em velocity [m/s]	0.13 ± 0.05	0.13 ± 0.05	0.83
Mitral lateral E/Em ratio	9.54 ± 6.49	9.04 ± 3.83	0.75
Tricuspid lateral Em velocity [m/s]	0.13 ± 0.04	0.13 ± 0.04	0.75
Mitral lateral interval $_{E-Em}$	34.12 ± 24.07	35.30 ± 28.32	0.87
Tricuspid lateral interval $_{E-Em}$	38.55 ± 28.21	36.93 ± 30.99	0.84
LA lateral Sm velocity [m/s]	0.07 ± 0.06	0.06 ± 0.01	0.20
LA lateral Em velocity [m/s]	0.11 ± 0.03	0.11 ± 0.03	0.80
LA medial Sm velocity [m/s]	0.07 ± 0.01	0.06 ± 0.02	0.13
LA medial Em velocity [m/s]	0.10 ± 0.02	0.09 ± 0.03	0.61
RA lateral Sm velocity [m/s]	0.12 ± 0.03	0.11 ± 0.03	0.43
RA lateral Em velocity [m/s]	0.16 ± 0.05	0.17 ± 0.05	0.56

LV — left ventricle; LA — left atrium; RA — right atrium; E — early diastolic wave, A — late diastolic wave; Sm — tissue Doppler systolic wave; Em — tissue Doppler early diastolic wave; Am — tissue Doppler late diastolic wave

Table 3. Comparison of echocardiographic findings at 24 hour follow-up after cardioversion between group 1 and group 2.

	Group 1: SR	Group 2: AF	P
Pulmonary artery pressure [mm Hg]	37.10 ± 8.68	39.72 ± 10.37	0.33
Mitral E velocity [m/s]	0.97 ± 0.20	0.97 ± 0.21	0.98
Mitral A velocity [m/s]	0.62 ± 0.20	0.48 ± 0.13	0.005
Mitral E/A ratio	1.70 ± 0.60	2.12 ± 0.57	0.01
Mitral lateral Em velocity [m/s]	0.10 ±0.02	0.10 ± 0.02	0.78
Mitral lateral Am velocity [m/s]	0.07 ± 0.03	0.06 ± 0.03	0.22
Mitral lateral E/Em ratio	10.15 ± 5.27	9.52 ± 3.79	0.64
Tricuspid lateral Em velocity [m/s]	0.11 ± 0.03	0.12 ± 0.04	0.85
Tricuspid lateral Am velocity [m/s]	0.08 ± 0.03	0.08 ± 0.03	0.76
Mitral lateral interval $_{E-Em}$	35.94 ± 27.18	36.47 ± 27.68	0.94
Tricuspid lateral interval $_{E-Em}$	46.30 ± 29.90	50.56 ± 36.69	0.65
LA lateral Sm velocity [m/s]	0.07 ± 0.01	0.07 ± 0.02	0.49
LA lateral Em velocity [m/s]	0.10 ± 0.02	0.10 ± 0.02	0.97
LA lateral Am velocity [m/s]	0.07 ± 0.03	0.05 ± 0.02	0.004
LA medial Sm velocity [m/s]	0.07 ± 0.01	0.06 ± 0.02	0.21
LA medial Em velocity [m/s]	0.08 ± 0.02	0.08 ± 0.01	0.43
LA medial Am velocity [m/s]	0.07 ± 0.02	0.05 ± 0.02	0.053
RA lateral Sm velocity [m/s]	0.13 ± 0.03	0.12 ± 0.03	0.49
RA lateral Em velocity [m/s]	0.15 ± 0.04	0.14 ± 0.04	0.53
RA lateral Am velocity [m/s]	0.10 ± 0.04	0.10 ± 0.03	0.93

SR — sinus rhythm; AF — atrial fibrillation; LA — left atrium; RA — right atrium; E — early diastolic wave, A — late diastolic wave; Sm — tissue Doppler systolic wave; Em — tissue Doppler early diastolic wave; Am — tissue Doppler late diastolic wave

Table 4. Comparison of atrial electromechanical parameters.

	Group 1: SR	Group 2: AF	P
Ventricular annular measurements:			
Mitral lateral AEMD	72.71 ± 5.62	82.00 ± 5.61	< 0.001
Mitral medial AEMD	63.36 ± 4.84	74.34 ± 7.02	< 0.001
Tricuspid lateral AEMD	58.20 ± 4.41	68.06 ± 7.76	< 0.001
Inter-AEMD	14.51 ± 6.50	13.93 ± 8.76	0.79
Intra-$AEMD_{LEFT}$	9.34 ± 4.92	7.66 ± 6.31	0.29
Intra-$AEMD_{RIGHT}$	5.16 ± 5.90	6.27 ± 7.95	0.57
Atrial walls measurements:			
LA lateral AEMD	70.23 ± 6.66	83.43 ± 8.82	< 0.001
LA medial AEMD	61.38 ± 4.04	70.45 ± 10.13	< 0.001
RA lateral AEMD	55.27 ± 4.31	65.54 ± 9.27	< 0.001
Inter-AEMD	14.96 ± 6.47	17.89 ± 11.33	0.25
Intra-$AEMD_{LEFT}$	8.85 ± 5.94	12.98 ± 8.72	0.053
Intra-$AEMD_{RIGHT}$	6.10 ± 3.11	4.91 ± 6.73	0.41

SR — sinus rhythm; AF — atrial fibrillation; AEMD — atrial electromechanical delay; LA — left atrium; RA — right atrium

Table 5. Correlation analysis between P-wave measurements and atrial electromechanical delay parameters.

Parameter	Duration	P maximum		P minimum		P-wave dispersion	
		R	P	R	P	R	P
Mitral lateral AEMD	76.80 ± 7.25	0.54	< 0.001	0.01	0.89	0.55	< 0.001
Mitral medial AEMD	68.19 ± 8.02	0.59	< 0.001	–0.18	0.19	0.67	< 0.001
Tricuspid lateral AEMD	62.54 ± 7.81	0.61	< 0.001	–0.04	0.78	0.64	< 0.001
Inter-AEMD	14.26 ± 7.50	–0.11	0.43	0.06	0.67	–0.13	0.34
Intra-$AEMD_{LEFT}$	8.60 ± 5.58	–0.14	0.32	0.21	0.09	–0.25	0.07
Intra-$AEMD_{RIGHT}$	5.65 ± 6.83	–0.08	0.95	–0.17	0.22	0.06	0.68
LA lateral AEMD	76.04 ± 10.08	0.62	< 0.001	0.08	0.55	0.60	< 0.001
LA medial AEMD	65.37 ± 8.58	0.46	0.001	–0.09	0.51	0.51	< 0.001
RA lateral AEMD	59.79 ± 8.58	0.59	< 0.001	0.03	0.79	0.59	< 0.001
Inter-AEMD	16.25 ± 8.95	0.12	0.37	0.06	0.67	0.10	0.45
Intra-$AEMD_{LEFT}$	10.67 ± 7.50	0.30	0.03	0.22	0.11	0.21	0.12
Intra-$AEMD_{RIGHT}$	5.58 ± 5.01	–0.21	0.12	–0.22	0.11	–0.13	0.34

AEMD — atrial electromechanical delay; LA — left atrium; RA — right atrium

$T_{Em\text{-}E}$ intervals measured at mitral and tricuspid annulus were not significant between the groups. LA lateral Sm and Em velocities, LA medial Sm and Em velocities and, RA lateral Sm and Em velocities were similar between groups, while LA lateral Am velocities were lower in group 2 ($p = 0.004$). LA medial and RA lateral velocities were also similar between two groups (Table 3).

Mitral lateral AEMD, mitral medial AEMD, tricuspid lateral AEMD, LA lateral AEMD, LA medial AEMD and RA lateral AEMD durations were significantly higher in group 2 compared with group 1 ($p < 0.001$ for all) (Table 4). However, inter-AEMD, intra-$AEMD_{LEFT}$ and intra-$AEMD_{RIGHT}$ durations were similar in both groups.

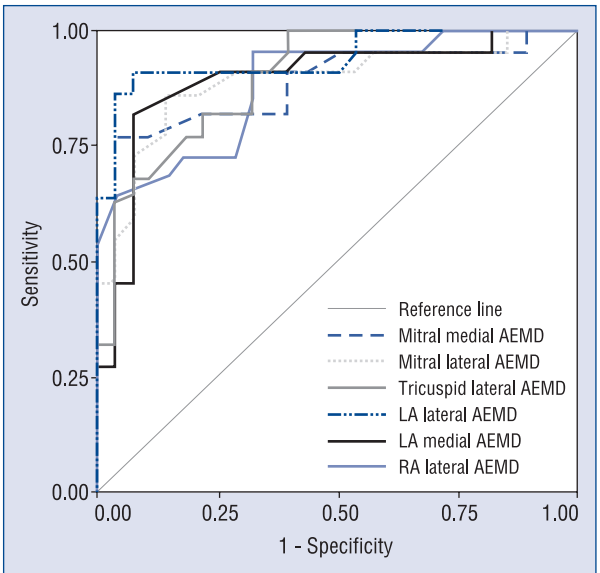
Bivariate correlation analysis between LA diameter, LA maximum volume and LA lateral AEMD duration showed that both LA diameter and LA maximum volume are significantly correlated with LA lateral AEMD 24 h after cardioversion (LA diameter: $r = 0.54$, $p < 0.001$; LA maximum volume: $r = 0.51$, $p < 0.001$).

Analysis of P-wave durations showed prolonged P_{max} (85.55 ± 5.43 vs. 110.78 ± 8.38) and P_{dis} (28.79 ± 5.55 vs. 53.36 ± 8.31) in group 2 ($p < 0.001$ for both). Bivariate correlation analysis

between P-wave duration and AEMD durations showed that both P_{max} and P_{dis} are significantly correlated with mitral lateral AEMD, mitral medial AEMD, tricuspid lateral AEMD, LA lateral AEMD, LA medial AEMD and RA lateral AEMD durations (Table 5).

A ROC analysis was then performed to assess the predictive power of the AEMD durations for AF recurrence at 1 month postcardioversion. The area under the curve was calculated to be 0.89, 0.89, 0.89, 0.94, 0.88 and 0.89 for mitral lateral AEMD, mitral medial AEMD, tricuspid lateral AEMD, LA lateral AEMD, LA medial AEMD and RA lateral AEMD, respectively, indicating that all these parameters are associated with AF recurrence following electrical cardioversion (Fig. 2).

Using a univariate and multivariate regression models, the patients's age, AF duration, energy delivered during cardioversion, LA diameter, mitral A velocity, PAP, LA maximum volume index, LA lateral Am velocity and LA lateral AEMD duration, which was found to have highest sensitivity and specificity to predict AF recurrence in ROC analysis, were included as predictor variables for arrhythmia recurrence at 1 month follow-up after successful cardioversion. In univariate analysis, AF duration, LA diameter, LA maximum volume index, mitral A velocity, LA lateral Am velocity and LA lateral AEMD duration were found to be significant predictor of AF recurrence. In multivariate analysis, only LA lateral AEMD duration was a significant predictor of AF recurrence with an odds ratio of 1.46 (p = 0.03) (Table 6).



Parameter	AUC	P	95% CI	
			Lower bound	Upper bound
Mitral lateral AEMD	0.89	< 0.001	0.79	0.98
Tricuspid lateral AEMD	0.89	< 0.001	0.81	0.98
Mitral medial AEMD	0.89	< 0.001	0.79	0.99
LA lateral AEMD	0.94	< 0.001	0.87	1.00
RA lateral AEMD	0.88	< 0.001	0.79	0.97
LA medial AEMD	0.89	< 0.001	0.80	0.99

Figure 2. Atrial electromechanical delay (AEMD): atrial electromechanical delay; CI — confidence interval; LA — left atrium; RA — right atrium; AUC — area under the curve.

Discussion

In our study, we found that AEMD durations predict recurrence of AF at 1 month following electrical cardioversion in patients with persistent AF. In addition, we demonstrated that P-wave dispersion and P-wave maximum duration are prolonged in recurrent patients and there is a significant

Table 6. Univariate and multivariate logistic regression analysis of predictors of recurrence.

	Univariate analysis		Multivariate analysis	
	Odds ratio (95% CI)	P	Odds ratio (95% CI)	P
Age	0.99 (0.91–1.07)	0.84		
AF duration	1.88 (1.28–2.76)	0.001	1.49 (0.80–2.77)	0.20
Cardioversion energy	1.01 (0.99–1.02)	0.19		
LA diameter	1.96 (1.35–2.86)	0.004	1.60 (0.90–2.82)	0.10
LA maximum volume index	1.13 (1.04–1.23)	0.003	1.02 (0.81–1.28)	0.85
Mitral A velocity	0.03 (0.005–2.65)	0.01	0.01 (0.002–5.62)	0.50
Pulmonary artery pressure	1.03 (0.97–1.09)	0.33		
LA lateral Am velocity	0.02 (0.005–10.08)	0.009	0.01 (0.008–12.66)	0.39
LA lateral AEMD duration	1.58 (1.18–2.12)	0.002	1.46 (1.02–2.11)	0.03

CI — confidence interval; AF — atrial fibrillation; AEMD — atrial electromechanical delay; LA — left atrium

correlation between those two parameters and AEMD durations.

Identification of predictors for AF recurrence might play a key role in selection of treatment strategies. Several studies have been conducted to determine the predictors of AF recurrence, however none of the parameters have achieved to predict with 100%. That is why new parameters are still studied for predicting the recurrence of AF.

Long durations of AF result in electrical and structural changes in the LA, the so-called atrial remodeling. This atrial remodeling is characterized by atrial dilatation and deterioration of atrial conduction. Tieleman et al. [10] have demonstrated that persistent AF changes atrial refractory period and leads recurrence of AF by this mechanism. Total atrial conduction time (TACT) has been proposed as a marker of atrial remodeling and represents the time between the onset of atrial depolarization from the sinus node to the farthest point of atria [3, 4, 10]. This interval increases in patients with persistent AF due to increased atrial diameter and intra-atrial conduction delay [10, 11]. The TACT resulting from beat-to-beat variability is also associated with increased AF recurrence [10, 11]. The delay in TACT can be measured on 12-lead surface ECG by maximum P-wave duration, whereas the beat-to-beat varying atrial conduction time is measured by P-wave dispersion.

The delays in inter-atrial conduction time and intra-atrial conduction time and, prolonged P_{dis} have been shown to be related to AF recurrence [12, 13]. Ermis et al. [14] reported increased risk of persitent AF development in pre-hypertensive patients who had prolonged P-wave duration. In the present study, patients who reverted back to AF after cardioversion had longer P_{max} and P_{dis} than patients who stayed in SR. However, increased LA diameters and the diseases in the atrial conduction pathways make the evaluation of these parameters difficult and can cause errors in the measured patterns. High-resolution ECG records and electrophysiologic studies could get a more accurate P wave duration measurements, however this not practical.

In our study, the AEMD durations obtained from the atrial walls and ventricular annulus predicted the recurrence of AF at 1 month. AEMD duration is the sum of impulse propagation from sinus node to the atria and atrial electromechanical coupling duration. The histopathological changes in the atria are the most significant determinants of AEMD duration. Atrial fibrosis, myocyte atrophy and scattered fibrotic foci in the normal atrial tissue lead a non-homogenious conduction of impulses in the atriums. The prolonged P_{max} and P_{dis} durations resulting from non-homogenious atrial conduction are significantly correlated with AEMD durations. AEMD duration is also well correlated with the degree of histopathological changes [11]. LA lateral AEMD duration significantly correlated with LA diameter and LA maximum volume. These data explain the importance of AEMD durations, which is an indicator of pathological changes in the atrium, for predicting AF recurrence.

The time of AEMD measurement could also be important to predict AF recurrence. Atrial stunning is prominent in the first hours following cardioversion and measurements within this period can have a significant impact on AEMD durations. To exclude this limitation we performed the measurements at 24 h postcardioversion.

We did not find a relation between inter-AEMD duration and AF recurrence. This might be attributed to the fact that our patients were relatievely young (mean age; 61 years) and had mildly dilated LA (mean diameter; 42.5 mm). Age is an important factor affecting atrial histopathological changes and conduction times. Atrial conduction times are prolonged in patients with older age and increased LA enlargement.

AF duration before cardioversion is an important clinical parameter for prediction of AF recurrence [10, 13]. In our study, patients with AF recurrence had longer duration of AF than patients who did not recur. However, duration of AF can not be accurately assessed in some patients and this limits the implication of this parameter.

LA diamater and volume are among the predictors for recurrence of AF [14–16]. Both precardioversion and postcardioversion LA dimaters and LA volumes were found to be significantly increased in patients with AF recurrence. These results support that LA dimater and LA volume are useful parameters for prediction of AF. However, beat-to-beat variations in atrial contractile function during AF can cause errors in the measurements of these parameters. Particularly, the beat-to-beat variability will be very marked in in patients with significant mitral regurgitation. These factors limit the use of LA diameter and volume as predictor of recurrence.

Our study also demonstrated increased LA lateral Sm, Am and mitral A velocities in patients staying in SR at 1 month as compared to patients with AF recurrence. As the duration of AF and stress in the LA increases, the degree of atrial remodeling enhances and this result in reduce in

atrial contraction velocities [17]. Several previous studies are consistent with our results [13, 15]. However, measurements of LA systolic and late diastolic velocities in the early periods following cardioversion may show lower values than the real values because of atrial stunning. Therefore, these parameters have limited value in the prediction of AF recurrence.

Duration of AF, LA diameter, LA maximum volume index, mitral A velocity, LA lateral Am velocity and LA lateral AEMD duration were found to be significant predictors of AF recurrence at 1 month follow-up in univariate analysis, whereas LA lateral AEMD duration was the only significant parameter in multivariate analysis. These data show that AEMD durations are independent predictors of AF recurrence. The results of study conducted by Park et al. [18] support our findings. Park et al. [18] showed that AEMD duration significantly longer in patients with AF recurrence than in patients with SR 6 months after cardioversion and normal control population. Also they showed that AEMD duration correlated with LA volumes as our study results.

Clinical implication

The atrial mechanical delay duration is prolonged in patients with AF recurrence. Prolonged AEMD durations predict short-term AF recurrence (at 1 month), hence they could be used for patient follow-up after cardioversion. These early results also suggest that more aggressive treatment of patients with prolonged AEMD durations might prevent recurrence of AF after cardioversion.

Limitations of the study

The first limitation of our study was the relatively small number of patients. The limited patients number in this study may not represent the whole population. Second limitation was that patients were not randomized according to the antiarrhythmic treatment. However, the number and group of antiarrhythmic drugs were not different between two groups and all patients received amiodarone after cardioversion. Third limitation was the short duration of follow-up. One month follow-up results might not be an indicator for AEMD duration to predict AF recurrence for long--term. However, the results of this pilot study can be a guide to further studies.

Conclusions

Postcardioversion AEMD durations predict recurrence of AF at 1 month follow-up in patients with persistent AF. If rhythm control is desired, more aggressive treatment approaches might be needed in patients with marked AEMD prolongation. The results of this pilot study showing AEMD durations as predictor of AF recurrence need to be confirmed with larger group of patients and long--term follow-up studies.

Conflict of interest: none declared

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