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Myocardial structure and function and predictors of recurrent atrial fibrillation in hypertensive patients after electrical cardioversion

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Abstract

Objective. To determine predictors of atrial fibrillation (AF) recurrence in patients with persistent AF and arterial hypertension (HTN) after sinus rhythm (SR) restoration by electrical cardioversion (ECV) using data on the dynamics of structural and functional changes in the heart during prolonged preventive antiarrhythmic therapy (AART). **Design and methods.** The study involved 127 patients with HTN and non-valvular form of persistent AF who underwent ECV leading to the restoration of sinus rhythm. All patients underwent Holter monitoring (HM) and echocardiography, and were randomized into groups for certain anti-arrhythmic drug (propafenone, sotalol, amiodarone). **Results and conclusions.** There is a strong negative correlation between the duration of sinus rhythm after ECV with the prolonged antiarrhythmic therapy and the value of anteroposterior size of the left atrium referred to the body surface area. There was no change in the structural parameters of the left heart one year after ECV when SR was maintained in patients receiving AART (propafenone, sotalol, amiodarone). Amiodarone is more effective for the prevention of AF recurrence after ECV than sotalol or propafenone; and is associated with a decrease in left ventricular isovolumic relaxation time and deceleration time of mitral flow.

Key words: atrial fibrillation, hypertension, electrical cardioversion, sotalol, amiodarone, propafenone, structure and function of the myocardium

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Структурно-функциональное состояние миокарда и предикторы рецидива фибрилляции предсердий у больных артериальной гипертензией после электрической кардиоверсии

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Резюме

Цель работы — идентифицировать предикторы рецидива фибрилляции предсердий (ФП) у лиц с персистирующей ФП и артериальной гипертензией (АГ) после проведенной электрокардиоверсии (ЭКВ), используя данные о динамике структурно-функциональных изменений сердца во время длительной профилактической антиаритмической терапии (ААТ). **Материалы и методы.** В исследовании приняло участие 127 пациентов с АГ и неклапанной формой персистирующей ФП, у которых проведение ЭКВ привело к восстановлению синусового ритма. Всем пациентам было проведено холтеровское мониторирование (ХМ) и эхокардиография (ЭхоКГ) с определением комплекса общепринятых морфофункциональных параметров. Все больные были разделены на группы для получения определенного антиаритмического препарата (ААП) (пропафенон, соталол, амиодарон). **Результаты и выводы.** Длительность сохранения синусового ритма (СР) после ЭКВ на фоне ААТ имеет наиболее тесную отрицательную связь с величиной переднезаднего размера левого предсердия, отнесенного к площади поверхности тела. Поддержание СР на фоне приема изучавшихся ААП (пропафенон, соталол, амиодарон) в течение 1 года после ЭКВ не сопровождалось изменением основных структурных показателей левых отделов сердца. Амиодарон является более эффективным ААП для профилактики рецидивов ФП после ЭКВ, чем соталол и пропафенон; его действие сопровождается уменьшением времени изоволюмического расслабления левого желудочка и времени замедления трансмитрального кровотока.

Ключевые слова: фибрилляция предсердий, артериальная гипертензия, электрокардиоверсия, соталол, амиодарон, пропафенон, структурно-функциональное состояние миокарда

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Introduction

With increase of life expectancy in the 1990s, atrial fibrillation (AF) became a major cause of hospitalization for heart rhythm disturbances [1] and a heavy burden for the healthcare systems of the developed countries [2, 3]. Most frequently, AF develops in patients with arterial hypertension (HTN), especially when left ventricular hypertrophy (LVH) is present [4].

According to current concepts, there are several goals of treatment in AF: sinus rhythm (SR) recovery and support, heart rate (HR) control in persistent AF, and prevention of tachycardia-induced cardiomyopathy [5] and thromboembolic complications [4].

SR recovery with various types of cardioversion in AF patients is one of the major goals determining the prognosis and quality of life. In the last decades, more and more attention has been paid to new methods for AF treatment, based on intracardiac ablation procedures in the areas responsible for the development and maintenance of this arrhythmia. However, to date, there is no consensus on the primary choice of invasive treatment methods in AF patients. This is partly due to many factors that affect safety and efficacy of the interventional treatment.

Strengths and weaknesses of various cardioversion types are widely discussed in the literature. In this regard, it remains only to point out that electrical cardioversion (ECV) is effective in 80–98 % of cases, cheaper than pharmacological cardioversion, and its safety is comparable with placebo [6–8]. This makes ECV a method of choice for the planned SR recovery.

As a results of cardioversion, SR recovery is successful in the majority of patients, however, high recurrence rate is an unsolved question requiring further research in this area in order to identify predictors of recurrence, as well as effective methods of SR support in this group of patients. In most cases, AF recurrence develops within the first month after ECV [9, 10]. Indicators of heart autonomic regulation violation [11] and myocardial electrical instability [12, 13] are considered the strongest predictors. Various indicators of heart structural remodeling in HTN are discussed as predictors for both AF onset and recurrence after the successful cardioversion [14]. Nevertheless, the studies aimed at finding risk-stratification models are still relevant for predicting cardioversion efficacy in patients with HTN that is considered the most common AF cause.

ECV efficacy is improved in combination with antiarrhythmic drugs (AAD); in particular, it was shown for amiodarone [15, 16], propafenone and sotalol [17]. After SR recovery, preventive therapy reduces the recurrence risk.

Objective of our study was the identification of AF recurrence predictors in patients with persistent AF and HTN after the ECV based on dynamics of heart structural and functional changes in patients receiving long-term preventive antiarrhythmic therapy.

Design and methods

The prospective part of the study included 127 patients with persistent AF and SR recovery after ECV.

Inclusion criteria were the following: age from 18 to 75 years old, presence of non-valvular persistent AF in hypertensive patients, indications and absence of contraindications to SR recovery, successful SR recovery by planned ECV, written informed patient's consent to participate in the study.

Exclusion criteria were the following: cancer, mental disease, alcoholism, uncontrolled HTN, thyrotoxicosis, obesity grade III–IV, sick sinus syndrome, functional class III and higher chronic heart failure, dilated cardiomyopathy, angina pectoris, heart disease, unorganized intracardiac thrombus, "normalizing" thromboembolism in past, left atrium size greater than 50 mm.

All patients were diagnosed with hypertension stage I or II, among them 37 patients (29 %) had HTN 1 degree, 62 patients (49 %) had HTN 2 degree and 28 patients (22 %) had HTN 3 degree. HTN degree was determined in accordance with the guidelines of the European Society of Cardiology [18]. Stage was determined according to the guidelines of the All-Russian Scientific Society of Cardiology. All patients were administered antihypertensive therapy, including angiotensin converting enzyme inhibitors in combination with hydrochlorothiazide, angiotensin 2 receptor blockers in combination with hydrochlorothiazide,

beta-adrenoceptor blockers in combination with either angiotensin converting enzyme inhibitors, or angiotensin 2 receptor blockers.

In order to prevent thromboembolism during cardioversion, all patients received an indirect anticoagulant warfarin controlled by international normalized ratio value ranging from 2 to 3.

Prior to ECV, three treatment groups each of 36 people were formed by simple randomization in order to receive treatment with one of three AAD. Patients of the first group (32 persons with recovered SR) received propafenone 150 mg 3 times a day, patients of the second group (32 persons with recovered SR) received sotalol 242.8 ± 26.4 mg daily, patients of the third group (33 persons with recovered SR) received amiodarone 200 mg once daily in the evening for five days a week and a two-day discontinuation [19]. A group of 30 patients with HTN stage I–II and persistent AF, admitted to the hospital for SR recovery, was also allocated. They received amiodarone for SR maintenance.

ECV was performed under short-term intravenous anesthesia. Special conductive gel was applied on the contact electrode surface. Simultaneous defibrillation mode was set, which did not expose electrical discharge in the vulnerable period of the cardiac cycle. One defibrillator electrode was placed in the second and third intercostal space to the right of the right edge of the sternum, and the second electrode was applied to the apex of the heart. The discharge was deposited in the expiratory phase. The ECV began with 200 J discharge. When inefficient, the discharge energy was increased sequentially by 100 J, then by another 60 J to achieve maximum of 360 J [20].

In order to evaluate the structural and functional myocardial characteristics, all patients underwent echocardiography (Sonos-5500, Hewlett Packard, USA) with 3.5 MHz sensor in V- and M-mode, and Doppler echocardiography in SR. The study was conducted in standard echocardiographic positions before the ECV, 1 month and 12 months after the ECV.

The interventricular septum (IVS) thickness was measured from IVS endocardium of the right ventricle to IVS endocardium of the left ventricle (LV), left ventricular posterior wall (LVPW) thickness was measured from LVPW endocardium to LVPW epicardium, end-diastolic diameter (EDD) from IVS endocardium to LVPW endocardium in diastole. All measurements were carried out for at least three cardiac cycles, and then the average of three values was calculated.

The cardiac chamber size, valves and aorta changes, global and local myocardial contractility were assessed.

LV myocardial mass (LVMM) was calculated by R. Devereux and N. Reicheck formula [21]:

$LVMM = 1.04 \times [(IVST + LVPWT + EDD)^3 — (EDD)^3] — 13.6$, where

1.04 — myocardium density ratio;

IVST — interventricular septum thickness (mm);

LVPWT — left ventricular posterior wall thickness (mm);

EDD — left ventricular end-diastolic diameter (mm).

Left ventricular muscle mass index (LVMMI) was calculated as the ratio between LVMM and body surface area.

LV myocardial hypertrophy was diagnosed in case of LVMMI increase more than 115 g/m^2 for men and more than 95 g/m^2 in women [22].

Index of anterior-posterior size of the left atrium (LAI) was calculated as the ratio between anterior-posterior size of the left atrium and body surface area. Normally, LAI is less than 2.0 cm/m^2 [22].

LV end-diastolic diameter index (EDDI) was calculated as the ratio between LV end-diastolic diameter and body surface area. Normally, EDDI is less than 2.8 cm/m^2 [22].

Left ventricular diastolic function (LVDF) was assessed based on the blood flow rate through the mitral valve in the period of LV filling. In this position, maximum velocity of early diastolic filling — E (cm/s) and the blood flow rate during atrial systole — A (cm/s) were measured. Their ratio E/A was calculated, which normally did not exceed 1.9 ± 0.6 for patients < 50 years old and 1.1 ± 0.3 for patients ≥ 50 years old [23].

The deceleration time through the mitral valve in phase of LV early diastolic filling (DT, ms) was also calculated.

During simultaneous recording of blood flow in the LV outflow tract and transmitral blood flow, LV isovolumic relaxation time was measured (ms).

For Doppler assessment of LV diastolic function the following conditions were essential: SR, absence of mitral stenosis or severe mitral or aortic insufficiency, and heart rate below 90 beats per minute [24].

Holter monitoring (HM) of 12-lead electrocardiography were used to assess the efficacy of cardioversion and the conducted antiarrhythmic treatment (INKART, St Petersburg, Russia). To assess late relapses, HM was conducted 5 days after the ECV, and subsequently 1, 6 and 12 months after the ECV. When AF paroxysms were not registered at HM or surface electrocardiographic study, the treatment was considered effective.

Statistical analysis was performed using STATISTICA 6.0 for Windows. The data were described as arithmetic mean M and standard deviation σ (M ± σ), and if the distribution did not meet the normality criteria median Me and interquartile range [Q1; Q3] were evaluated. Qualitative characteristics were presented as absolute frequencies and percentages. The differences between two groups were assessed by the Student's parametric t-test for independent samples in case of normal distribution, and by the Manna-Whitney in case of non-parametric test. When comparing more than two groups, analysis of variance was used, or its non-parametric

Table 1

CLINICAL CHARACTERISTICS OF PATIENTS

Parameter	Propafenone (n = 32)	Sotalol (n = 32)	Amiodarone (n = 33)	Amiodarone (inpatients) (n = 30)	p
Gender, Male:Female	26:6	22:10	24:9	22:8	> 0.05
Age, years	57.6 ± 8.3	59.9 ± 7.9	57.5 ± 6.9	57.7 ± 8.0	> 0.05
Smoking, n (%)	14 (43.8%)	11 (34.4%)	12 (36.4%)	15 (50.0%)	> 0.05
BMI, kg/m^2	28.6 ± 3.7	29.3 ± 3.8	29.6 ± 4.8	29.8 ± 4.2	> 0.05
DM, n (%)	6 (18.8%)	5 (15.6%)	5 (15.2%)	8 (26.7%)	> 0.05
CHF I-II FC, n (%)	24 (75.0%)	21 (65.6%)	22 (66.7%)	25 (83.3%)	> 0.05
Duration of AF history, months	24 [16; 48]	26 [23; 54]	36 [24; 52]	24 [9; 48]	> 0.05
Duration of persistent AF, months	2 [1; 4]	3 [1.25; 4]	2 [1; 4]	1.5 [1; 2.5]	> 0.05
LA, mm	45.5 ± 3.9	46.2 ± 4.4	45.6 ± 4.5	46.5 ± 4.6	> 0.05
LA/BSA, cm/m^2	2.46 ± 0.37	2.45 ± 0.28	2.40 ± 0.37	2.53 ± 0.35	> 0.05
EDD, mm	53.6 ± 4.3	51.7 ± 5.5	50.9 ± 5.1	53.1 ± 5.4	> 0.05
EDD/BSA, cm/m^2	2.92 ± 0.39	2.77 ± 0.35	2.68 ± 0.40	2.87 ± 0.35	> 0.05
EF LV, %	0.61 ± 0.08	0.61 ± 0.07	0.62 ± 0.10	0.61 ± 0.10	> 0.05

Note: BMI — body mass index; DM — diabetes mellitus; CHF — chronic heart failure; FC — functional class; AF — atrial fibrillation; LA — left atrium; BSA — body surface area; EDD — end-diastolic diameter left ventricular; EF — ejection fraction; LV — left ventricular.

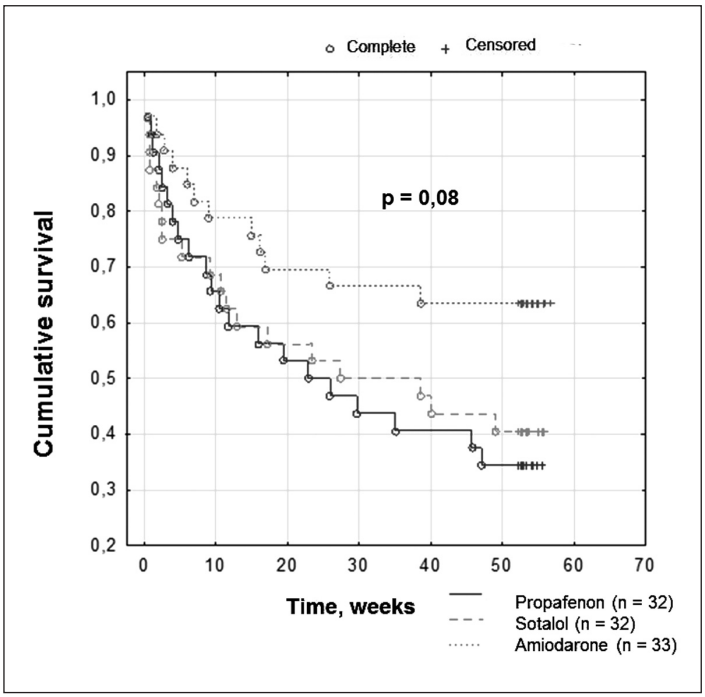
Table 2

THE EFFECTIVENESS OF LONG-TERM AMIODARONE, PROPAFENONE AND SOTALOL THERAPY IN PREVENTING ATRIAL FIBRILLATION RECURRENCE

Number of patients with SR after ECV	Propafenone (n = 32)	Sotalol (n = 32)	Amiodarone (n = 33)	Amiodarone (inpatients) (n = 30)
After 1 month	25 (78%)	24 (75%)	29 (87.9%)	24 (80%)
After 6 months	16 (50%)	17 (53%)	23 (69.7%)	20 (67%)
After 12 months	11 (34%)	13 (41%)	21 (63.6%)	16 (53%)

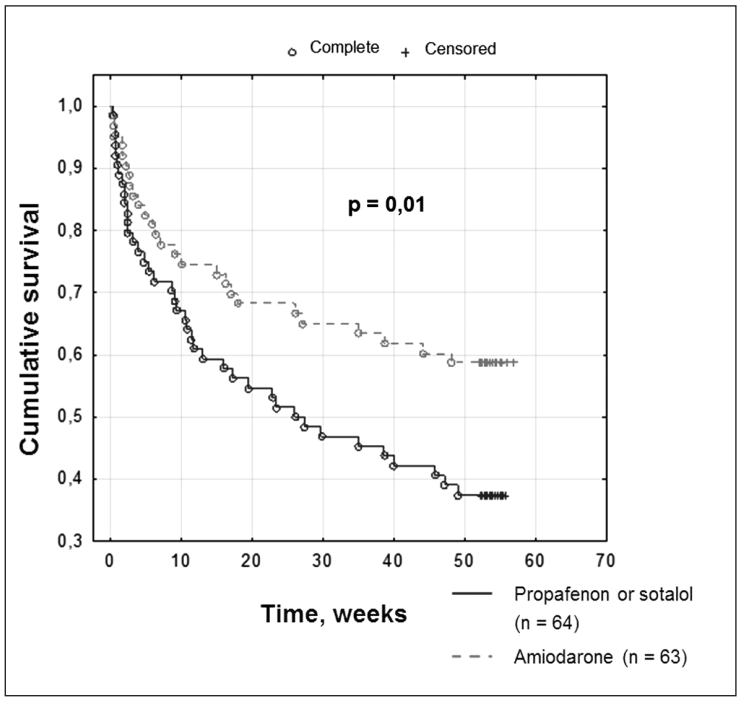
Note: SR — sinus rhythm; ECV — electrical cardioversion.

Figure 1. Time before the atrial fibrillation recurrence depending on the antiarrhythmic drugs after electrical cardioversion in outpatient setting



Note: AF — atrial fibrillation.

Figure 2. Time before the atrial fibrillation recurrence depending on the received antiarrhythmic therapy (pooled groups of patients receiving amiodarone or other antiarrhythmic therapy)



Note: AF — atrial fibrillation.

analogue — the Kruskal–Wallis test. When comparing paired samples, the Wilcoxon test was used. To compare percentage, the χ^2 test was used, and when the expected number of cases in at least one of the cells of 2 × 2 table was less than 5, the Fisher's exact test was used. Bilateral variants of statistical tests were used. The relationships were assessed by the Spearman rank correlation. The Kaplan–Meier curve analysis was used to evaluate the time before the outcome onset. The Cox test was used to compare survival. Differences were considered significant at p-level < 0.05.

Results and discussion

Clinical characteristics of patients are presented in Table 1, and the results of preventive AAD therapy are presented in Table 2.

Regarding the rate of SR maintenance, no significant differences were found between three groups after 1 or 6 months. After 12 months, there was a tendency to predominant SR in the group of amiodarone (p = 0.08). However, the analysis of the results of anti-relapse and antiarrhythmic therapy between the pooled group of patients treated with amiodarone (n = 63), and the pooled group treated with other antiarrhythmic drugs (propafenone or sotalol, n = 64), the difference were significant in favor of amiodarone, but only after 12 months (59% vs 38%, p = 0.02). Similar results were obtained when comparing the Kaplan–Meier curves: there was a trend (p = 0.08) to a longer time till AF recurrence in the amiodarone group compared to three groups of patients who underwent ECV (Fig. 1). The absence of significant results may be due to the small sample size, while the pooled twice greater groups (amiodarone against other AAD) differed regarding this indicator (Fig. 2). Almost half of recurrent AF cases occurred within the first 10 weeks after the ECV with a significant reduction afterwards.

LVH was diagnosed based on echocardiography in 75 patients included in the prospective part of the study (59%), and LVMMI did not exceed the normal range in 52 patients (41.0%) (Fig. 3).

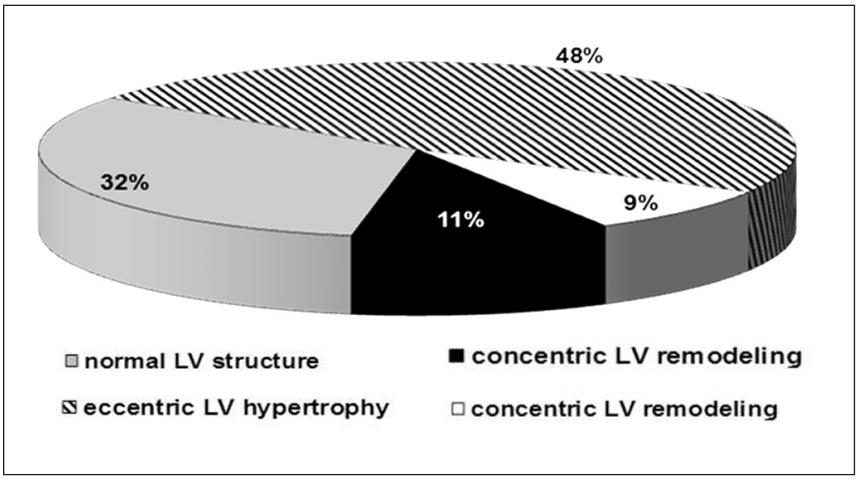
Twelve months after ECV, there were no changes in LVMM and LVMMI in patients with stable sinus rhythm. Eccentric LVH remained in 44% of patients, but there was an increase in the number of patients with normal geometry of the left ventricle (LV) (LVMMI was in the normal range, relative wall thickness was less than 0.42) up to 49% (Fig. 4).

The mean LV ejection fraction (EF) in HTN patients with AF was 0.61 ± 0.09 conventional units, there were no patients with EF less than 0.40 conventional units. Thus, LV systolic function in HTN patients was within the normal range.

After 12 months EF did not change and was 0.62 ± 0.10, 0.61 ± 0.07, and 0.62 ± 0.09 conventional units in patients receiving amiodarone, sotalol, and propafenone, respectively.

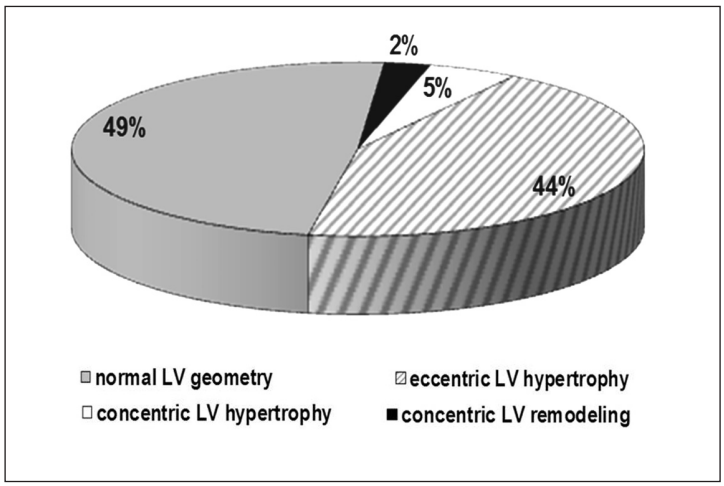
Assessment of the LV structure in patients with stable SR showed that the anterior-posterior size of the left atrium (LA), LV EDD, as well as

Figure 3. The left ventricular remodeling in patients with atrial fibrillation and arterial hypertension before electrical cardioversion



Note: LVH — left ventricle.

Figure 4. The left ventricular remodeling in patients with atrial fibrillation and arterial hypertension after electrical cardioversion



Note: LVH — left ventricle.

Table 3

DYNAMICS OF STRUCTURAL CHARACTERISTICS LEFT HEART IN PATIENTS WITH ARTERIAL HYPERTENSION AND ATRIAL FIBRILLATION AFTER ELECTRICAL CARDIOVERSION WITH STABLE SINUS RHYTHM WITHIN 12 MONTHS ON PROPAFENONE THERAPY (n = 11)

Parameter	Baseline	After 12 months	p
LA, mm	44.7 ± 3.7	45.3 ± 3.8	> 0.05
LA/BSA, cm/m^2	2.32 ± 0.32	2.35 ± 0.35	> 0.05
EDD, mm	53.0 ± 4.3	51.5 ± 4.4	> 0.05
EDD/BSA, cm/m^2	2.75 ± 0.39	2.67 ± 0.44	> 0.05

Note: LA — left atrium; BSA — body surface area; EDD — end-diastolic diameter left ventricular.

Table 4

CHANGES IN LEFT HEART STRUCTURE IN PATIENTS WITH ARTERIAL HYPERTENSION AND ATRIAL FIBRILLATION AFTER ELECTRICAL CARDIOVERSION WITH STABLE SINUS RHYTHM WITHIN 12 MONTHS OF SOTALOL THERAPY (n = 13)

Parameter	Baseline	After 12 months	p
LA, mm	44.3 ± 5.6	42.7 ± 5.8	> 0.05
LA/BSA, cm/m^2	2.29 ± 0.29	2.21 ± 0.30	> 0.05
EDD, mm	49.5 ± 6.2	48.9 ± 5.1	> 0.05
EDD/BSA, cm/m^2	2.56 ± 0.32	2.53 ± 0.29	> 0.05

Note: LA — left atrium; BSA — body surface area; EDD — end-diastolic diameter left ventricular.

Table 5

CHANGES IN LEFT HEART STRUCTURE IN PATIENTS WITH ARTERIAL HYPERTENSION AND ATRIAL FIBRILLATION AFTER ELECTRICAL CARDIOVERSION WITH STABLE SINUS RHYTHM WITHIN 12 MONTHS OF AMIODARONE THERAPY (n = 21)

Parameter	Baseline	After 12 months	p
LA, mm	44.1 ± 4.5	43.8 ± 4.3	> 0.05
LA/BSA, cm/m^2	2.30 ± 0.34	2.28 ± 0.31	> 0.05
EDD, mm	50.8 ± 5.5	51.3 ± 4.3	> 0.05
EDD /BSA, cm/m^2	2.63 ± 0.39	2.67 ± 0.35	> 0.05

Note: LA — left atrium; BSA — body surface area; EDD — end-diastolic diameter left ventricular.

Table 6

LEFT HEART STRUCTURE IN PATIENTS WITH ARTERIAL HYPERTENSION AND ATRIAL FIBRILLATION AFTER SUCCESSFUL ELECTRICAL CARDIOVERSION DEPENDING ON SINUS RHYTHM MAINTENANCE WITHIN 1 YEAR OF AMIODARONE, SOTALOL AND PROPAFENONE THERAPY

Parameter	SR, n = 45	AF, n = 52	p
LA, mm	44.3 ± 4.6	47.0 ± 3.5	0.002
LA/BSA, cm/m^2	2.30 ± 0.30	2.55 ± 0.33	< 0.001
EDD, mm	50.8 ± 5.5	53.1 ± 4.5	0.02
EDD /BSA, cm/m^2	2.63 ± 0.37	2.89 ± 0.38	0.003

Note: SR — sinus rhythm; AF — atrial fibrillation; LA — left atrium; BSA — body surface area; EDD — end-diastolic diameter left ventricular.

Table 7

THE DIASTOLIC LEFT VENTRICULAR FUNCTION IN PATIENTS WITH RECOVERED SINUS RHYTHM RECEIVING AMIODARONE, SOTALOL AND PROPAFENONE THERAPY

Parameter	Propafenone (n = 11)		Sotalol (n = 13)		Amiodarone (n = 21)	
	Baseline	After 1 year	Baseline	After 1 year	Baseline	After 1 year
DDT, msec	204.1 ± 18.1	200.3 ± 19.4	197.6 ± 22.3	198.6 ± 22.6	196.3 ± 20.2	184.2 ± 20.3*
IVRT, мсек	85.7 ± 6.6	83.4 ± 6.9	88.3 ± 7.2	83.1 ± 6.0*	88.9 ± 7.8	85.1 ± 7.1*
E, cm/s	68.9 ± 6.1	66.7 ± 5.1	69.3 ± 6.4	68.7 ± 6.7	61.0 ± 5.7	59.4 ± 6.2
A, cm/s	72.6 ± 6.2	72.3 ± 6.5	74.8 ± 5.8	75.0 ± 6.1	73.3 ± 6.0	71.1 ± 6.9
E/A	0.96 ± 0.11	0.93 ± 0.08	0.94 ± 0.10	0.93 ± 0.10	0.85 ± 0.09	0.83 ± 0.10

Note: DDT — diastolic deceleration time; E — maximum velocity of early diastolic filling; A — maximal velocity of the blood flow during atrial systole; IVRT — isovolumic relaxation time; * — $p < 0.05$ significant differences compared to baseline.

the ratio of these indices to the body surface area did not change significantly in any of the groups (Tables 3–5).

The baseline LV structural indicators were analyzed in a subgroup of patients with AF recurrence by the end of 1-year follow up after successful ECV. The dimensions of the LA, LV EDD, and the ratio of these indices to the body surface area were significantly greater than those in the subgroup of patients with stable SR (Table 6).

After SR recovery, almost all HTN patients showed the decreased ratio of peaks E and A in comparison with the reference values, indicating rigid type LV diastolic dysfunction.

The average values of transmitral flow deceleration time and LV isovolumic relaxation time were within the reference values, but the upper limit was outside that range.

After AAD therapy, the patients with stable SR demonstrated no significant changes in peaks E and A, as well as their ratio (Table 6). Amiodarone treatment was associated with the decrease in LV isovolumic relaxation time and transmitral flow deceleration time, and sotalol treatment was associated with LV isovolumic relaxation time decrease that is considered a positive prognostic sign. Propafenone therapy had no impact on the indicators of LVDF (Table 7).

A moderate negative correlation was found between the duration of SR persistence after the ECV and the ratio between the LV anterior-posterior size and the body surface area. At the same time, there was only a weak negative correlation between the LV absolute size and the duration of SR persistence after the ECV. So

Table 8

RELATIONSHIP BETWEEN THE DURATION OF SINUS RHYTHM MAINTENANCE AFTER ELECTRICAL CARDIOVERSION AND THE LEFT VENTRICULAR STRUCTURE

Parameter	Correlation coefficient
End-diastolic diameter LV	–0,18*
End-diastolic diameter LV / body surface area	–0,26**
Anterior-posterior diameter LA	–0,27**
Anterior-posterior diameter LA / body surface area	–0,37†
Left ventricular myocardial mass index	–0,26**

Note: LV — left ventricle; LA — left atrium; * — $p < 0.05$; ** — p — 0.01; † — $p < 0.001$.

the indicators normalized to the body surface area (BSA) are more significant.

A weak negative correlation ($r = –0.39$, $p < 0.05$) was found between the duration of SR persistence after the ECV and the patient age, indicating the increase in AF frequency in older populations. Also, a weak negative correlation was found between the duration of SR persistence after the ECV and the ratio between LV EDD with respect to BSA and LVMMI that, apparently, indicates the leading role of LV remodeling in AF pathogenesis (Table 8).

The proportion of patients with a successful ECV receiving any of selected drugs (amiodarone, sotalol, propafenone) was comparable and ranged from 88 to 92% which corresponds to published data [25].

Identifying predictors of effective SR recovery and maintenance in AF patients is a relevant issue in cardiology; the data are scarce and conflicting, possibly due to the etiological heterogeneity of AF in different groups of patients. I.A. Paraskevaidis et al. (2005) found out that, in patients with isolated AF, the ECV efficacy was the most strongly predicted by the degree of LV anterior-posterior shortening (> 30%) and flow velocity in the LA appendage (> 20 cm/s) assessed by the pulsed Doppler mode of transesophageal echocardiography [26].

Olshansky B. et all (2005) showed that an increase in LA absolute size (odds ratio of 1.32 for the size of more than 5.5 cm; 1.16 — for the size of 4.6–5.5 cm; 1.23 — for the size of 4.1–4.5 cm; $p = 0.008$) was an independent predictor of arrhythmia recurrence after the ECV [27].

Twelve months after the ECV when treated by any of selected drugs patients with stable SR demonstrated no changes of LVMM and LVMMI. Neither LVEF changes were registered.

Based on published data, hypertensive myocardial remodeling is a risk factor for AF development in HTN patients [28, 29]. In patients with long-term maintenance of stable SR, the proportion of patients with rigid type LV diastolic dysfunction did not change, however, amiodarone therapy was associated with the reduction in LV isovolumic relaxation time and transmitral blood flow deceleration time, which indicates a more favorable effect of amiodarone on diastolic dysfunction and its potential preventive effect compares with other studied antiarrhythmic drugs. We suggest that in addition to the antiarrhythmic effect, amidarone has a number of other properties, e. g. the block of the mechanisms promoting myocardial remodeling, such as autonomic imbalance and cardiac diastolic function [30]. LV isovolumic relaxation time decreased in patients treated with sotalol. Propafenone therapy did not lead to any changes in LV diastolic function.

Conclusions

1. The duration of SR maintenance after the ECV in patients receiving antiarrhythmic therapy negatively correlated with the value of LA anterior-posterior size related to the BSA.

2. SR maintenance in patients receiving the studied AAD (propafenone, sotalol, amiodarone) for 1 year after the ECV was not accompanied by changes in the structural parameters of the left heart.

3. Amiodarone is more effective for the prevention of AF recurrence after the ECV than sotalol and propafenone. Treatment with amiodarone is accompanied by a decrease in LV isovolumic relaxation time and transmitral flow deceleration time.

Conflict of interest

The authors declare no conflict of interest.

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