Stunning of the left atrium after pharmacological cardioversion of atrial fibrillation

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Abstract

Introduction: Stunning of the left atrium and atrial appendage is a well known but not fully clarified phenomenon observed during the cardioversion of atrial fibrillation regardless of the cardioversion method attempted.

Aim: To assess the effects of propafenone and amiodarone on left atrium and left atrial appendage contractility.

Methods: Forty patients with paroxysmal atrial fibrillation (20 females, 20 males), aged 60-83 (mean 72.0±10.1) years, were enrolled into the study. Half of these patients had sinus rhythm restored by the administration of oral propafenone (150-300 mg) and the remaining 20 patients were treated with intravenous amiodarone (150-450 mg). The control group consisted of 20 patients (10 females, 10 males) aged 52-78 (mean 61.2±9.3) years with sinus rhythm and no history of atrial fibrillation. All the patients had a transthoracic (TTE) and transesophageal (TEE) echocardiography performed while still in the AF, before drug administration and 1 hour after sinus rhythm restoration.

Results: All haemodynamic parameters of the left atrium measured after the sinus rhythm restoration were significantly worse when compared with the control group. Left atrial fractional shortening and total atrial fraction were significantly lower after propafenone than amiodarone (8.6±3.6% *vs* 11.7±5.5%, $p<0.05$; and LA FC 16.2±5.3% *vs* 23.3 (±6.3)% respectively, $p<0.05$).

Doppler echocardiographic parameters included in the analysis such as mitral flow and superior left pulmonary vein flow were significantly lower in the sinus rhythm restoration group than in the control group. Among them the end-diastolic mitral flow velocity amplitude and flow velocity integral as well as the maximum pulmonary retrograde velocity were significantly worse in the group treated with propafenone than in patients receiving amiodarone. All the atrial appendage Doppler velocity parameters were significantly reduced after the sinus rhythm restoration in both groups. In the patients treated with propafenone, values of these parameters were significantly decreased compared with the patients receiving amiodarone.

Conclusions: Successful pharmacological cardioversion of atrial fibrillation causes the left atrium and left atrial appendage contractility impairment similar to that observed with other methods of the sinus rhythm restoration. Following the AF cardioversion the level of left atrial stunning is higher in the patients treated with propafenone than in subjects receiving amiodarone.

Key words: atrial fibrillation, echocardiography, stunning, left atrial appendage, propafenone, amiodarone

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Introduction

Atrial fibrillation (AF) is the form of cardiac arrhythmia that is associated with a higher risk of systemic thromboembolic events, especially of stroke [1]. The risk of such events is associated with the presence of arrhythmia itself as well as the attempts of pharmacological and electrical sinus rhythm restoration. Decreased contractility of the left atrium (LA) and left atrial appendage (LAA), known as stunning, is probably responsible for the thromboembolic complications.

Left atrial stunning is a well known but not entirely understood phenomenon observed in AF patients during the process of sinus rhythm restoration. The LLA contractility impairment may be caused by the arrhythmia itself [2]. Impairment of LA and LAA muscle fibre contractility is mainly observed after sinus rhythm

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restoration. The LAA stunning phenomenon has been most frequently described after electrical cardioversion [3, 4]. Other studies have shown that regardless of the method of electrical cardioversion, the LA and LAA contractility impairment should be expected [5].

LA and LAA contractility reduction has also been observed after pharmacological cardioversion [6, 7]. Pharmacotherapy diminishes the contractile function of LA and LAA also in patients with AF who do not respond to attempted cardioversion [8]. Single case reports of spontaneous sinus rhythm return during transesophageal echocardiography also point to the possibility of the stunning phenomenon [9].

The aim of the study was to compare the effects of propafenone and amiodarone on the contractile function of LA and LAA.

Methods

Patients

Forty patients with AF (20 females, 20 males), aged 60-83 years (mean 72.0±10.1) were involved in the study. Of these 20 had sinus rhythm restored by the use of oral propafenone (150-300 mg) and the remaining 20 patients had intravenous amiodarone (150-450 mg) administrated. All patients had idiopathic AF.

The control group consisted of 20 patients with sinus rhythm and no evidence of AF in their history.

The Regional Ethics Committee's permission was granted to conduct the study.

Echocardiographic examinations

Transthoracic (TTE) and transesophageal (TEE) echocardiographic examinations were performed in all patients using the commercially available Sonos 5500 imaging system with a 5 MHz transesophageal multiplane annular phased array probe. TEE examination was performed according to the standard protocol. TEE was performed prior to the introduction of pharmacological treatment. Control TEE was performed one hour after successful sinus rhythm restoration.

In TTE the following parameters were analysed:

LVEDd [cm] – left ventricular end diastolic diameter in M – mode presentation
EFLV [%] – ejection fraction calculated according to the Teicholz formula
FSLV [%] – left ventricular fractional shortening
SVLV [ml] – left ventricular stroke volume
CO [l/min] – left ventricular cardiac output
E dcct [ms] – deceleration time of early diastolic left ventricular filling
LA max [cm] – maximum size of left atrium in M-mode presentation
LA min [cm] – minimum size of left atrium in M-mode presentation
LA P wave – size of left atrium in M-mode presentation on peak of P wave of synchronised ecg recording
LA short [cm] – left atrial cross sectional dimension in 4CH projection
LA long [cm] – left atrial longitudinal dimension in 4CH projection
LA circ [cm] – left atrial circumference in 4CH projection
LA area [cm^2] – left atrial surface area in 4CH projection

FS LA [%] – left atrial fractional shortening:

$$\text{FS LA [\%]} = \frac{\text{LA wave P} - \text{LAmin}}{\text{LA max}} \times 100\%$$

FBO LA [%] – left atrial passive ejection fraction:

$$\text{FBO LA \%} = \frac{\text{LAmax} - \text{LA P wave}}{\text{LA max}} \times 100\%$$

FC LA [%] = left atrial total atrial fraction:

$$\text{FC LA [\%]} = \frac{\text{LA max} - \text{LA min}}{\text{LA max}} \times 100\%$$

IE LA [%] – left atrial expansion index:

$$\text{IE LA [\%]} = \frac{\text{LA max- LA min}}{\text{LAmin}} \times 100\%$$

ET LA [ms] – left atrial ejection time

A ampl LV [cm/s] – end diastolic mitral velocity flow amplitude
A intg LV [cm] – end diastolic mitral flow integral

In TEE the following parameters were analysed:

LAAF [cm/s] – maximum LAA outflow velocity
LAAB [cm/s] – maximum LAA inflow velocity
LAAF intg [cm] – maximum LAA outflow velocity integral
LAAB intg [cm] – maximum LAA inflow velocity integral
PVS [cm/s] – maximum left superior pulmonary vein (LSPV) systolic flow velocity
PVD [cm/s] – maximum diastolic LSPV flow velocity
PVA [cm/s] – maximum retrograde LSPV flow velocity.
PVA intg [cm] – maximum retrograde LSPV flow velocity integral
PVA time [msec] – flow time in LSPV in active LA systole

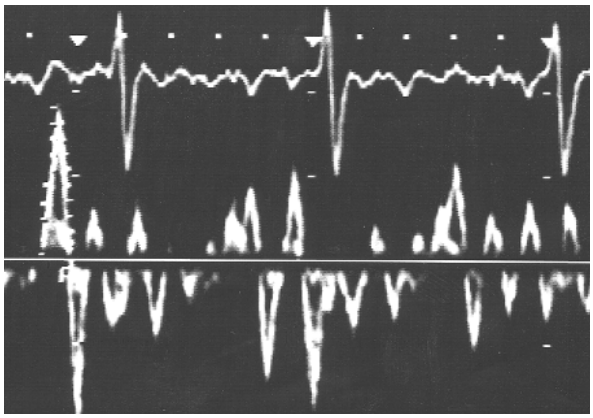

Figure 1. Doppler velocity flow in LAA during the atrial fibrillation

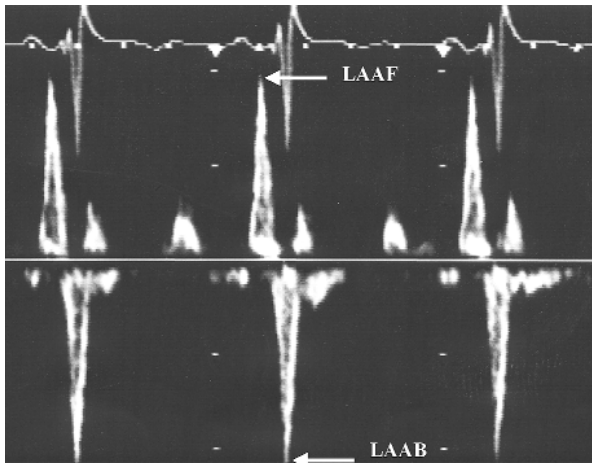

Figure 2. Doppler velocity flow in LAA during the sinus rhythm. The method of parameters calculation

SEC – spontaneous echocardiographic contrast
THR – thrombus

In the AF patients the measurements were performed during 5 consecutive heart beats and the mean value of all measurements of LAA flow velocity was used for further analysis. Once sinus rhythm was restored the arithmetical mean of 3 heart beats measurements was calculated and used for the analysis.

The flow velocities were measured using the pulse-wave Doppler with the lowest possible filter of 5-10 cm/s. During measurement the angle between the probe axis and the flow direction had to be less than 30°. The Doppler gate window was placed in LAA slightly below the LAA orifice. During AF the flow was chaotic, resembling saw teeth (Figure 1). Once sinus rhythm was restored, the flow became regurarly biphasic, typical characteristics (Figure 2) and occurred just after the P wave of synchronised ECG recording.

Each echocardiographic examination was verified by two experienced echocardiographists.

Functional LA parameters that could be assessed only after the sinus rhythm restoration, were compared with the control group. In the case of LAA parameters that are valid also in the presence of AF, measurements of LAAF, LAAB, LAAFintg , and LAABintg, obtained before and after the sinus rhythm restoration, were compared.

Statistical analysis

Measured values were expressed as mean values ± standard deviations or as numbers and percentages. Continuous variables were compared with using the t-Student's test, and discrete variables with the Chi^2 test, with or without Yates' correction. Differences were considered significant if the p value was lower than 0.05. The Statistica 5.0 PL software package was used for the statistical analysis.

Results

The studied groups treated with propafenone and amiodarone were clinically homogeneous and the analysed parameters before sinus rhythm restoration did not differ significantly (Table I).

The comparative analysis of selected haemodynamic parameters of LA demonstrated that all indexes in the patients with AF immediately after successful cardioversion were significantly worse than in the control patients with a stable sinus rhythm. Patients treated with propafenone had a significantly lower value of FSLA than patients treated with amiodarone, but FBOLA and IELA did not show any significant differences between the two groups (Table II).

Left atrial contractility reduction after sinus rhythm restoration was reflected by the indices calculated from the mitral inflow Doppler measurements. All these parameters were significantly lower in both propafenone and amiodarone groups as compared with the control group. AamplLV and AintgLV differentiated significantly the two treatment groups, being higher in patients receiving amiodarone than in those treated with propafenone. The duration of this flow (ETLP) was similar regardless of the type of pharmacotherapy used (Table III).

The functional indexes of LA derived from TEE measurements of LSPV flow characteristics were

Table I. Patient characteristics

parameter	propafenone	p	amiodarone	p	control group	p
	1	1 *vs* 2	2	1 *vs* 3	3	2 *vs* 3
Age [years]	71.3 (±8.5)	NS	72.8 (±11.2)	<0.05	61.2(±9.3)	<0.05
Male gender [%]	10 (50%)	NS	10 (50%)	<0.05	10 (50%)	<0.05
Arrythmia duration [h]	29.1 (±18.2)	NS	31 (±17.5)	–	–	–
LVEDd [cm]	5.3 (±0.61)	NS	5.4 (±0.55)	<0.05	4.7 (±0.42)	<0.05
EF [%]	53.1 (±7.9)	NS	51.3 (±8.1)	<0.05	58.3 (±8.5)	<0.05
FS [%]	27.0 (±4.1)	NS	25.8 (±3.9)	<0.05	29.3 (±5.3)	<0.05
SV [ml]	61.8 (±12.1)	NS	62.3 (±12.8)	<0.05	68.1 (±10.1)	<0.05
CO [l/min]	6.3 (±1.9)	NS	6.0 (±1.7)	NS	5.8 (±1.2)	NS
E dcct [ms]	138.0 (±21.8)	NS	143.1 (±28.3)	<0.05	180.3 (±28.3)	<0.05
LA max [cm]	4.2 (±0.48)	NS	4.3 (±0.54)	<0.05	3.8 (±0.51)	<0.05
LA short [cm]	4.1 (±0.41)	NS	4.2 (±0.46)	<0.05	3.6 (±0.33)	<0.05
LA long [cm]	7.1 (±0.59)	NS	7.4 (±0.61)	<0.05	6.3 (±0.71)	<0.05
LA circ [cm]	20.1 (±1.7)	NS	21.2 (±1.6)	<0.05	17.1 (±1.8)	<0.05
LA area [cm^2]	27.3 (±4.3)	NS	28.1 (±3.8)	<0.05	20.4 (±2.0)	<0.05
PVS [cm/s]	38.3 (±12.3)	NS	35.1 (±11.0)	<0.05	58.3 (±7.8)	<0.05
PVD [cm/s]	51.3 (±18.1)	NS	53.8 (±17.3)	<0.05	41.2 (±9.1)	<0.05
PVD dcct [ms]	102.1 (±21.8)	NS	99.8 (±28.3)	<0.05	121.3 (±30.1)	<0.05

Table II. The comparison of the selected left atrial haemodynamic parameters between the patients treated with propafenone, amiodarone and the control group

parameter	propafenone n=20	p	amiodarone n=20	p	control group n=20	p
	1	1 *vs* 2	2	1 *vs* 3	3	2 *vs* 3
FS LA [%]	8.6 (±3.6)	<0.05	11.7 (±5.5)	<0.05	15.1 (±6.2)	<0.05
FBO LA [%]	6.7 (±2.9)	NS	7.4 (±3.2)	<0.05	8.1 (±3.0)	<0.05
FC LA [%]	16.2 (±5.3)	<0.05	23.3 (±6.3)	<0.05	28.1 (±7.8)	<0.05
IE LA [%]	17.1 (±4.9)	NS	19.3 (±6.2)	<0.05	25.6 (±6.1)	<0.05

Table III. The comparison of the selected mitral Doppler velocity flow parameters reflecting LA function: patients treated with propafenone, amiodarone and the control group

parameter	propafenone n=20	p	amiodarone n=20	p	control group n=20	p
	1	1 *vs* 2	2	1 *vs* 3	3	2 *vs* 3
A ampl LV [cm/s]	34.6 (±8.3)	<0.05	46.5 (±13.7)	<0.05	66.1 (±15.4)	<0.05
A intg LV [cm]	3.2 (±0.9)	<0.05	4.7 (±1.6)	<0.05	7.6 (±1.7)	<0.05
ET LA [msec]	161.1 (±39.0)	NS	169.0 (±32.1)	<0.05	213.2 (±41.3)	<0.05

significantly lower in the propafenone and amiodarone group than in the control group. Comparison of subgroups receiving medical therapy showed significantly lower values of the amplitude and LSPV flow integral recorded during left atrial systole in the patients treated with propafenone as compared with those receiving amiodarone. The duration of such flow was similar in both medical treatment groups, very much alike the previously reported mitral inflow duration (Table IV).

The analysis of LAA flow parameters after sinus

Table IV. The comparison of the selected left superior pulmonary vein velocity flow parameters of LA function between the patients treated with propafenone, amiodarone and the control group

parameter	propafenone n=20	p	amiodarone n=20	p	control group n=20	p
	1	1 *vs* 2	2	1 *vs* 3	3	2 *vs* 3
PVA [cm/s]	20.1 (±5.1)	<0.05	25.5 (±6.1)	<0.05	32.2 (±7.9)	<0.05
PVA intg [cm]	2.2 (±0.5)	<0.05	2.9 (±0.7)	<0.05	4.1 (±1.1)	<0.05
PVA time [msec]	81.4 (±22.8)	NS	87.13 (±29.6)	<0.05	117.5 (±37.9)	<0.05

rhythm restoration showed a significant decrease of LAAF (Figure 3), LAAB (Figure 4), LAAFintg (Figure 5) and LAABintg (Figure 6) as compared with the values recorded during AF.

The comparison of these parameters before and after sinus rhythm restoration showed significant differences

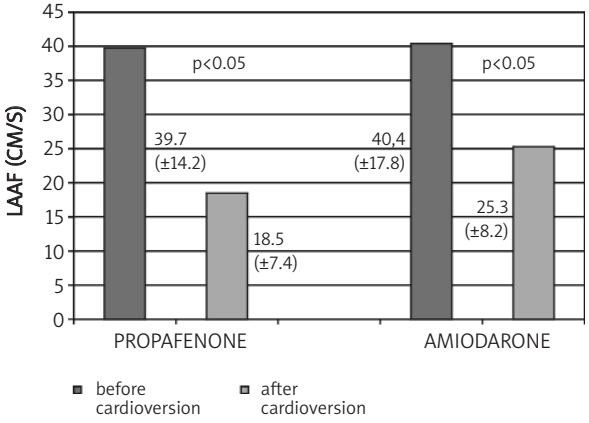

Figure 3. Velocity LAA outflow before and after AF cardioversion in the propafenone and amiodarone – treated patients

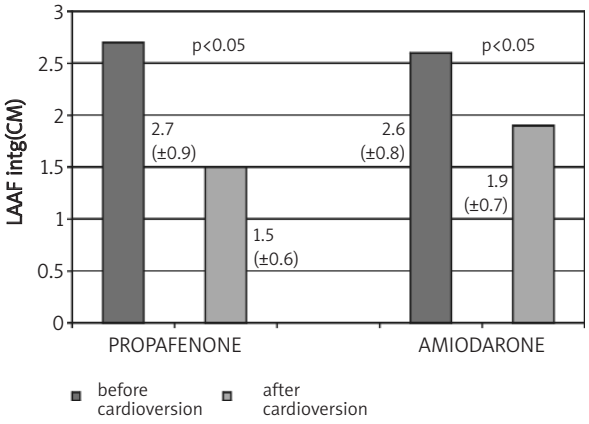

Figure 5. Velocity LAA outflow integral before and after AF cardioversion in the propafenone and amiodarone – treated patients

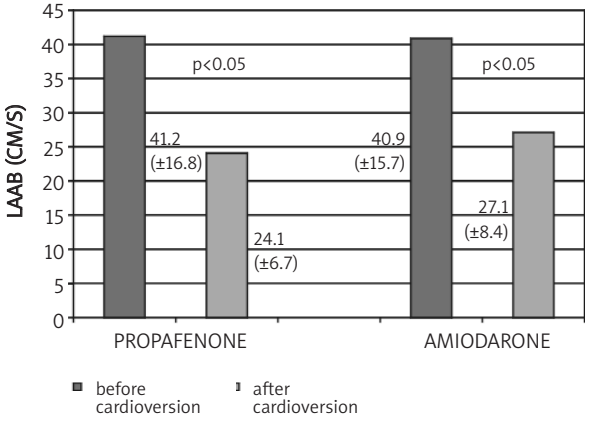

Figure 4. Velocity LAA inflow before and after AF cardioversion in the propafenone and amiodarone – treated patients

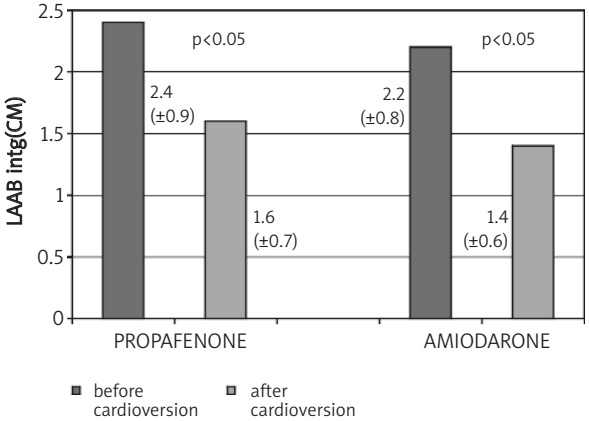

Figure 6. Velocity LAA inflow integral before and after AF cardioversion in the propafenone and amiodarone – treated patients

between the subgroups treated with propafenone or amiodarone, except of LAABintg, which was reduced to a similar extent in both studied subgroups. The prevalence of spontaneous echocardiographic contrast was similar in both subgroups, and the only thrombus was found in a patient treated with propafenone (Table V).

Table V. The Comparison of LAA stunning magnitude during AF cardioversion with propafenone and amiodarone (before *vs* after sinus rhythm restoration)

parameter	propafenone n=20	amiodarone n=20	p
diff LAAF [cm/s]	21.2 (±11.3)	15.2 (±9.2)	p<0.05
diff LAAB [cm/s]	18.4 (±10.7)	13.3 (±8.8)	p<0.05
diff LAAF intg [cm]	1.1 (±0.7)	0.7 (±0.5)	p<0.05
diff LAAB intg [cm]	0.9 (±0.4)	0.8 (±0.4)	NS
SEC	5 (25%)	4 (20%)	NS

Discussion

Some believe that pharmacological cardioversion of AF does not lead to significant impairment of LA contractile function, thus allowing less or shorter anticoagulation before and after the sinus rhythm restoration. Our study showed that such a concept is only partially true. Left atrial haemodynamic indexes after sinus rhythm restoration were significantly lower than in the control patients. The previous studies (10) revealed that only after a long period of sustained sinus rhythm did these parameters approached values typical for patients without a history of AF.

We have also confirmed that amiodarone reduces LA contractility less extensively than propafenone, showing that shortening fraction and total atrial fraction of LA are significantly higher after amiodarone administration. After pharmacological cardioversion the A wave becomes evident, however, is of a small amplitude for the first hours as compared with controls. In the propafenone group, the A wave had a significantly lower amplitude than in the group treated with amiodarone, indicating the more pronounced LA contractility impairment after propafenone.

Restoration of LA systolic function was investigated by Jović et al. [11], who demonstrated gradual recovery after pharmacological restoration of sinus rhythm after quinidine administration, which was evidenced by an increase of A wave amplitude of the mitral inflow with a concomitant reduction of flow velocity in early diastole. Additional confirmation of less depressive effects of amiodarone on LA systolic function was provided by the values of PVA and PVAintg calculated from the LSPV flow velocity. These parameters, as well as A wave values, were higher in patients treated with amiodarone. Nevertheless, regardless of the medication used, the parameters we studied had rather low values, confirming the findings of the previous studies on the electrical cardioversion [10]. So far these parameters have not been assessed in the setting of medical cardioversion.

LAA stunning after pharmacological restoration to sinus rhythm is a less known phenomenon. While the use of medications is an effective alternative to the electrical cardioversion for sinus rhythm restoration, this is especially holds true for short arrhythmic episodes. Studies investigating the LA and LAA function after medical cardioversion of AF are sparse and their results do not allow any definite conclusions to be made.

Our study indicates that LAA systolic impairment occurs regardless of the drug used. Patients treated with propafenone presented a significantly higher value of LAA stunning. Clinical studies investigating the effects of amiodarone on LA stunning were conducted by Amuchastegui et al. [12], who claimed that electrical cardioversion of AF caused mechanical dysfunction of LAA. Such dysfunction was not observed after medical sinus rhythm restoration with the use of amiodarone.

The results of Falcone et al. [6] were completely different. They have shown that LAA stunning occurs also after pharmacological AF cardioversion. A significant LAA inflow and outflow velocity reduction was accompanied by the new spontaneous contrast that was absent during AF. In contrast to the Amuchastegui et al. findings [12] they administrated procainamide, quinidine and flecainide, class I antiarrhythmic drugs according to the Vaughan – Williams classification.

In our own study we have noted a significant LAA inflow and outflow velocity as well as flow velocity integrals decrease. New spontaneous contrasting was present or increased its intensity in 20% of all patients. In one case TEE performed immediately after a successful sinus rhythm restoration provided evidence of a thrombus in LAA which was not present before the cardioversion.

The observation that both electrical and pharmacological cardioversion have a similar effect on LA and LAA, causing stunning, challenges the hypothesis that it is the electrical power that damages the atrial myocardium, suggesting other reasons for temporary dysfunction. Falcone et al. [6] proposed a very interesting interpretation that a significant increase of cell membrane depolarisation frequency in AF causes an excessive increase in calcium ions concentration, which disturbs atrial calcium receptors sensitivity. Sinus rhythm restoration eliminates the state of calcium overload,

resulting in a transitional deficit of Ca^{2+}. This, in turn, is associated with myocardial contractility reduction lasting until the calcium receptors reach their basic state.

Left atrial and LAA contractility reduction after sinus rhythm restoration was confirmed regardless of the cardioversion method used. Such deterioration is also observed after spontaneous sinus rhythm return [9].

The mechanism of LA and LAA stunning is not completely clarified. Several experimental and clinical studies demonstrated that parameters of the LA and LAA contraction are reduced immediately after sinus rhythm restoration. The presence of arrhythmia itself causes atrial contractility reduction [13]. One of the most important observations made in the previously mentioned study was that right atrial fibres, harvested from an appendage at the time of the mitral valve surgery of patients with AF, contracted less vigorously than those of patients with a sinus rhythm before the surgery. Myolysis and sarcomer replacement with glycogen accompanied the process. Earlier observations from the animal studies were similar suggesting that the reduced sarcomer number can lead to atrial mechanical dysfunction after successful AF cardioversion [14]. Taking into account the relatively small decrease of in the number of sarcomers (14%) observed in humans with AF, Schotten et al. [13] suggested that atrial dysfunction after sinus rhythm restoration is more likely associated with myofilament activation impairment than with a decreased number of sarcomers. Borgers et al. [15] proved that 80% of myocytes became non-differentiated, which was manifested as the loss of myofibres, glycogen accumulation, changes in the shape and size of mitochondria changes, endoplasmic reticulum fragmentation and nucleic chromatin dispersion. All these changes are potentially reversible, but such a process takes time. Transient atrial dysfunction may reflect this process.

One of the important signals causing myocardial systole is β-adrenergic receptor activation, which initiates a cascade of mediators and eventually induces myocardial systole. Schotten et al. [13] noted that the atrial myocardial response to isoproterenol stimulation in patients with AF was reduced. Acknowledging the fact that β receptor density and protein G level did not differ between the AF and sinus rhythm patients, they suggested that the mechanism of a weak reaction to β-adrenergic stimulation was different to that of "down regulation" observed in a tachycardiomyopathy.

The role of calcium in atrial stunning has been postulated for a long time now [16]. Leisted et al. [17] in a porcine model proved that both the degree of LA dysfunction and its duration after AF conversion into the sinus rhythm were less pronounced if verapamil had been given to the animals before the treatment initiation. The concomitant administration of calcium mimic agent BAY K8644 enhanced the postarrhythmic LA contractility impairment. The authors suggested that transmembrane calcium inflow was responsible for transitional LA dysfunction. Schotten et al. [13] in the study of calcium mimic agent acting on L channels Bay K8644 did not observe any improvements of atrial myofibres contractility after this agent. They claimed that the possible explanations were either a functional change or decreased number of L-type Ca^{2+} channels. Such observations also provide an explanation of the finding of the reduced response to isoproterenol despite unchanged β-adrenergic receptor density and normal protein G expression on atrial myocytes. Isoproterenol improves contractility in two mechanisms: the agent increases calcium inflow via L channels and enhances calcium reabsorption mediated by endoplasmic reticulum ATP-ase. The first mechanism is impaired in the AF patients while the other is intact, providing indirect evidence confirming that the endoplasmic reticulum in such cases is well preserved. This however contradicts the observations by Bogers et al. [15], who described endoplasmic reticulum fragmentation.

The differences between the effects of propafenone and amiodarone on LA and, especially, on the LAA function we have observed, could be explained by differences in drug-related mechanism of action interfering with previously described mechanisms of stunning. More pronounced impairment of LAA contractility in the case of propafenone may be related to β-blocking properties of the agent. Intravenous amiodarone administration facilitates its calcium channel blocking properties and decreases calcium inflow in the AF patients that have L calcium channels impaired. This mechanism, similar to the one observed after verapamil administration, seems to protect functional myofibres within LA and LAA in patients with AF.

Conclusions

1. Pharmacological cardioversion of atrial fibrillation into sinus rhythm results in the left atrial and left atrial appendage contractility impairment.
2. Following sinus rhythm restoration, the level of the left atrial stunning is more pronounced in patients treated with propafenone than in patients, who received amiodarone.

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Ogłuszenie lewego przedsionka po farmakologicznym umiarowieniu migotania przedsionków

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Streszczenie

Wstęp: Ogłuszenie lewego przedsionka i jego uszka jest znanym, nie do końca wyjaśnionym zjawiskiem obserwowanym w czasie umiarawiania migotania przedsionków (AF), niezależnie od zastosowanej metody kardiowersji.

Cel: Ocena wpływu propafenonu i amiodaronu na kurczliwość lewego przedsionka i jego uszka.

Metodyka: Badaniami objęto 40 chorych z napadowym AF (20 kobiet i 20 mężczyzn) w wieku 60–83 lat (śr. 2,0±10,1 lat). 20 chorych umiarowiono propafenonem (150–300 mg *p.o.*) a kolejnych 20 amiodaronem (150–450 mg *i.v.*). Do grupy kontrolnej zakwalifikowano 20 chorych (10 kobiet 10 mężczyzn) w wieku 52–78 (śr. 61,2±9,3) lat, z rytmem zatokowym i brakiem AF w wywiadzie. U wszystkich chorych wykonano badanie echokardiograficzne przezklatkowe i przezprzełykowe w czasie AF, przed podaniem leków oraz w czasie 1. godziny po powrocie rytmu zatokowego.

Wyniki: Wszystkie parametry dotyczące czynności hemodynamicznej lewego przedsionka były wyraźnie gorsze u chorych po umiarowieniu w porównaniu do grupy kontrolnej. Frakcja skracania lewego przedsionka była istotnie niższa po zastosowaniu propafenonu w porównaniu do podgrupy leczonej amiodaronem (FS LP: 8,6±3,6% *vs* 11,7±5,5%, p<0.05 oraz FC LP: 16,2±5,3% *vs* 23,3±6,3%, p<0,05). Podobnie wszystkie parametry dopplerowskie rejestrowane u chorych z AF z przepływu przez zastawkę mitralną oraz w żyle płucnej górnej lewej były mniejsze niż w grupie kontrolnej. Wśród nich zarówno amplituda i całka przepływu późnorozkurczowego przez zastawkę mitralną, jak i maksymalna prędkość przepływu zwrotnego przez żyłę płucną były wyraźnie gorsze w przypadku leczenia propafenonem niż amiodaronem. Wszystkie parametry rejestrowane z przepływu dopplerowskiego przez uszko lewego przedsionka uległy istotnemu zmniejszeniu po przywróceniu rytmu zatokowego, niezależnie od zastosowanego leku. Jednak w grupie chorych leczonych propafenonem zaobserwowano istotnie większy spadek wartości tych parametrów w porównaniu do wartości zarejestrowanych w podgrupie chorych leczonych amiodaronem.

Wnioski: Farmakologiczna rewersja migotania przedsionków do rytmu zatokowego powoduje upośledzenie kurczliwości lewego przedsionka i jego uszka podobnie do innych znanych sposobów umiarawiania. Po przywróceniu rytmu zatokowego stopień ogłuszenia uszka lewego przedsionka jest większy w przypadku zastosowania propafenonu niż amiodaronu.

Słowa kluczowe: migotanie przedsionków, echokardiografia, ogłuszenie, uszko lewego przedsionka, propafenon, amiodaron

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