

# The effects of radiofrequency ablation on left atrial systolic function in patients with atrioventricular nodal reentrant and atrioventricular reentrant tachycardias

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## Abstract

**Background:** Recurrent supraventricular arrhythmias may cause remodelling of the atria. The effects of radiofrequency (RF) ablation of these arrhythmias on left atrial function have not been well established.

**Aim:** To evaluate the effects of RF ablation on left atrial systolic function in patients with atrioventricular nodal reentrant tachycardia (AVNRT) and atrioventricular tachycardia (AVRT).

**Methods:** The study group consisted of 70 patients (22 men), in whom successful RF ablation of slow pathway (35 patients) or accessory pathway (35 patients) was performed. Patients with atrial fibrillation, structural heart disease, ventricular arrhythmias (> class 3 Lown), impaired left ventricular systolic function or on antiarrhythmics were excluded. All the patients had echocardiographic study before and 6 months after ablation. Left atrial systolic function was assessed using atrial ejection force (AEF) according to Manning's formula ( $AEF = 0.5 \times \rho \times MA \times A^2$ ,  $\rho$ : blood density = 1.06 g/cm<sup>3</sup>, MA: mitral orifice area [cm<sup>2</sup>], A: A wave velocity). The following left atrial dimensions were assessed: antero-posterior (LA-AP), infero-superior (LA-IS, long axis), medio-lateral (LA-ML, short axis). The correlations between AEF and electrophysiological parameters were analysed (VA — ventriculo-atrial conduction, VA/CL — tachycardia cycle length).

**Results:** The AEF increased significantly in the AVNRT group (7.78 vs 10.75 kdynes;  $p < 0.001$ ) whereas it did not change in the AVRT group (8.96 vs 9.50, NS). Left atrial dimensions decreased significantly in both groups (AVNRT group: LA-AP: 38 vs 34 mm; LA-ML: 37 vs 33 mm; LA-IS: 51 vs 45 mm;  $p < 0.001$ ; AVRT group: LA-AP: 38 vs 36 mm;  $p < 0.01$ ; LA-ML: 37 vs 35 mm,  $p < 0.001$ ; LA-IS: 50 vs 46 mm;  $p < 0.001$ ). There was a significant correlation between the increment of AEF and electrophysiological parameters of the tachycardia (VA,  $r = -0.51$  and VA/CL,  $r = -0.53$ ).

**Conclusions:** 1. RF ablation of AVNRT is associated with the improvement of left atrial systolic function. 2. Left atrial size decreases following RF ablation of both AVNRT and AVRT. 3. The effects of RF ablation on the left atrial systolic function depends on electrophysiological parameters of the tachycardia (VA and VA/CL).

**Key words:** radiofrequency ablation, AVNRT, AVRT, left atrium

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## INTRODUCTION

Radiofrequency ablation (RF) is an established method of treatment of atrioventricular nodal reentrant tachycardia (AVNRT) and atrioventricular reentrant tachycardia (AVRT) [1–3].

During AVNRT or AVRT, adverse haemodynamic changes occur, such as pulmonary artery wedge pressure elevation and cardiac index drop [4]. Recurrent attacks lead to deterioration of left ventricular (LV) systolic function and the development of arrhythmic cardiomyopathy [5]. It has been demonstrated that elimination of arrhythmia by ablation can restore normal LV function [6]. A tachycardia attack also causes an increase of atrial pressures, which are higher in case of AVNRT than AVRT [7]. This results from differences in anatomical substrate and electrophysiological characteristics of the two types of arrhythmia. During AVNRT, atrial contraction occurs almost simultaneously with ventricular contraction, when atrioventricular (AV) valves are closed. During AVRT, the reentry loop is greater and consequently atrial contraction is delayed in relation to ventricular contraction, when AV valves are partially open [8, 9].

In patients with paroxysmal atrial fibrillation (AF) it has been demonstrated that recurrent arrhythmia causes remodelling of the atrial wall and can change its electrophysiological parameters such as refractoriness and dispersion of the refractoriness [10, 11]. Prevention of AF recurrence can inhibit the remodelling process. However, current knowledge concerning the remodelling of the atria in patients with AVNRT and AVRT and the impact of therapy on this process is incomplete, which prompted our research.

## METHODS

### Patients

Patients were selected from 196 consecutive patients (72 males) treated by RF ablation in our institution. The following inclusion criteria were adopted: 1. good immediate effect and no complications of slow pathway RF ablation (AVNRT subgroup) or accessory pathway (WPW subgroup); 2. no arrhythmia recurrence at 6-month follow-up; 3. normal LV systolic function (ejection fraction [EF] > 50%, absence of regional wall motion abnormalities), end-diastolic LV dimension within the normal range on pre-ablation echocardiography study.

Exclusion criteria were as follows: 1. more than 1 tachycardia type as confirmed by electrophysiology study (EPS); 2. structural heart disease such as ischaemic, congenital or valvular heart disease or cardiomyopathy diagnosed according to standard criteria; 3. co-morbid AF as confirmed by ECG, Holter study or induced during EPS; 4. ventricular arrhythmia of intensity exceeding Lown class 3 on 24-hour Holter ECG; 5. antiarrhythmic drug therapy or other drug therapy that could potentially alter the left atrial (LA) function after ablation (rennin–angiotensin–aldosterone system blocking agents, statins).

Finally, 70 patients were included (22 men) aged  $43.9 \pm 11.1$  years (23–68); 35 patients in whom RF ablation was performed for AVNRT and 35 patients in whom ablation was carried out for AVRT. The subgroups did not differ significantly in terms of either mean age (45.6 years vs 42.2 years) or sex (8 males vs 13 males, respectively).

### Electrophysiology study and RF ablation

The procedure was performed after obtaining written, informed consent from all participants. Quadripolar diagnostic electrodes were introduced via the right femoral vein and positioned in the right ventricular apex, high right atrium and in para-hisian area. Through left subclavian vein, decapolar electrode was introduced into coronary sinus.

Full programmed pacing protocol of the ventricles and the atria was performed, aiming at induction of the arrhythmia. The type of arrhythmia was diagnosed based on the commonly adopted criteria [1]. After study completion, RF ablation of the arrhythmia substrate was performed. In all 35 of the AVNRT patients, slow pathway ablation was performed using an electro-anatomical method [12, 13]. The procedure was successful if no AVNRT or no more than one reciprocating impulse (“echo”) were induced during pacing in basic conditions and after intravenous orciprenaline administration.

In the WPW subgroup, ablation of the overt accessory pathway (AP) (i.e. with antegrade conduction) was performed during sinus rhythm. Concealed pathways (conducting only retrogradely) were mapped during ventricular pacing or during AVRT, in search for a site of the earliest retrograde atrial activation [14]. Ablation of the right-sided AP was done via the right atrium, after introducing the electrode via the right femoral vein. Left-sided pathways were eliminated via the LA, by trans-septal puncture. The procedure was successful when AP conduction was fully blocked and when no tachycardia could be induced. The AP localisation in the study group is presented in Table 1. Five of the left-sided lateral pathways were concealed. In all patients, only orthodromic AVRTs were induced.

The following parameters were analysed: 1. ventriculo-atrial coupling (VA) during arrhythmia — measured as the interval between local right ventricular activation to local high right atrial activation; 2. tachycardia cycle length (CL) measu-

**Table 1.** Localisation of accessory pathways in WPW patients

|                            |          |
|----------------------------|----------|
| Left-sided lateral         | 17 (48%) |
| Left-sided postero-septal  | 5 (14%)  |
| Right-sided postero-septal | 8 (23%)  |
| Right-sided antero-septal  | 2 (6%)   |
| Right-sided mid-septal     | 1 (3%)   |
| Right-sided lateral        | 2 (6%)   |

**Table 2.** Left atrial dimensions and atrial ejection force (AEF) before ablation and 6 weeks post-procedurally

| Parameter    | Before ablation | After ablation | P     |
|--------------|-----------------|----------------|-------|
| AVNRT group: |                 |                |       |
| LA-AP [mm]   | 38 ± 3          | 34 ± 2.9       | 0.001 |
| LA-ML [mm]   | 37 ± 3.6        | 33 ± 3.4       | 0.001 |
| LA-IS [mm]   | 51 ± 4.1        | 45 ± 5.2       | 0.001 |
| AEF [kdyn]   | 7.78 ± 2.87     | 10.75 ± 3.76   | 0.001 |
| WPW group:   |                 |                |       |
| LA-AP [mm]   | 38 ± 3          | 36.5 ± 3.1     | 0.01  |
| LA-ML [mm]   | 37 ± 2.8        | 35 ± 3.4       | 0.001 |
| LA-IS [mm]   | 50 ± 3          | 46 ± 3.9       | 0.001 |
| AEF [kdyn]   | 8.96 ± 2.65     | 9.50 ± 3.58    | NS    |

AVNRT — atrioventricular nodal reentrant tachycardias; WPW — accessory pathway; rest abbreviations: see “Methods”

red as RR interval on surface ECG; 3. the VA/CL ratio; 4. the number of RF applications.

In all patients before ablation and at 6 months post-procedurally, history was taken and physical examination performed, along with standard surface ECG recording, 24-hour Holter ECG and echocardiographic study.

### Echocardiography

The study was performed using Sonos 1000 Hewlett-Packard machine with a 2.5/3.5 MHz transducer and included M-mode, two-dimensional (2D) and Doppler examinations, with patient in supine left-lateral position. Simultaneous lead II surface ECG recording was obtained. Cardiac chamber sizes were measured by M-mode, in parasternal long axis view, under control of 2D imaging. Left atrial size was also assessed in the apical 4-chamber (4CH) view in end-systole. All measurements were performed during sinus rhythm of less than 80 bpm.

Mitral valve area was assessed based on mitral annulus measurement in apical 4CH view. The following parameters were assessed; 1. antero-posterior LA dimension (LA-AP) in parasternal long axis view; 2. inferior-superior LA dimension (LA-IS, long axis) in the apical 4CH view; 3. medio-lateral (LA-ML, short axis) in the apical 4CH view; 4. mitral valve area (MA); 5. peak atrial wave velocity of the LV inflow (A); 6. LA ejection force (AEF), calculated by Manning formula [15]:  $AEF = 0.5 \times \rho \times MA \times A^2$ , where  $\rho$  — blood density index =  $1.06 \text{ g/cm}^3$ , MA — mitral valve area [ $\text{cm}^2$ ], A — peak velocity of the mitral inflow A wave.

### Statistical analysis

Measurements are presented as mean values ± SD and ranges or numbers and percentages. Numerical data were compared by Student t test and within groups, the formula for paired data was used. Non-parametric data were compared with use of  $\chi^2$  test. Correlations were tested by Pear-

son correlation coefficient. Significance level was set at  $p < 0.05$ .

## RESULTS

### Electrophysiological parameters

The VA time in the entire group was  $107.1 \pm 57.6$  ms (25–225 ms). It was significantly shorter in the AVNRT subgroup ( $53.7 \pm 17.5$  ms; 25–90 ms) in comparison with the WPW subgroup ( $160.6 \pm 23.6$  ms; 125–225 ms;  $p < 0.001$ ). Tachycardia CL did not differ significantly between subgroups ( $340.3 \pm 52.4$  ms vs  $350.1 \pm 46$  ms, respectively). The VA/CL ratio was significantly lower in the AVNRT subgroup ( $0.2 \pm 0.1$ ) in comparison with the WPW subgroup ( $0.5 \pm 0.1$ ;  $p < 0.001$ ). The number of RF applications was similar in both groups ( $8.6 \pm 7.6$  vs  $9.2 \pm 7.6$ ; NS).

### Echocardiographic parameters

In the AVNRT subgroup, a significant reduction of the LA size and significant AEF increase were noted (Table 2). In the WPW subgroup, a significant reduction of LA size was also noted but AEF was not significantly altered after ablation. The reduction of LA size and AEF change were significantly greater in the AVNRT subgroup (Table 3). In the entire study group, a negative correlation was found between AEF increment ( $\Delta AEF$ ) and VA time, ( $r = -0.51$ ) and VA/CL ratio (Figs. 1, 2). The number of RF applications had no impact on the studied parameters.

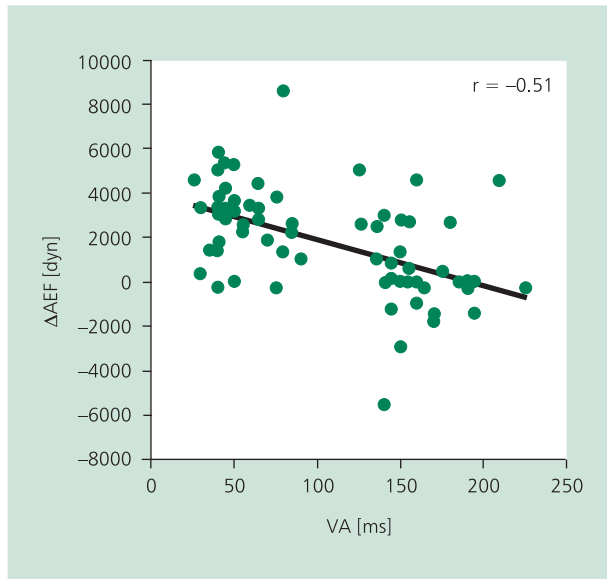
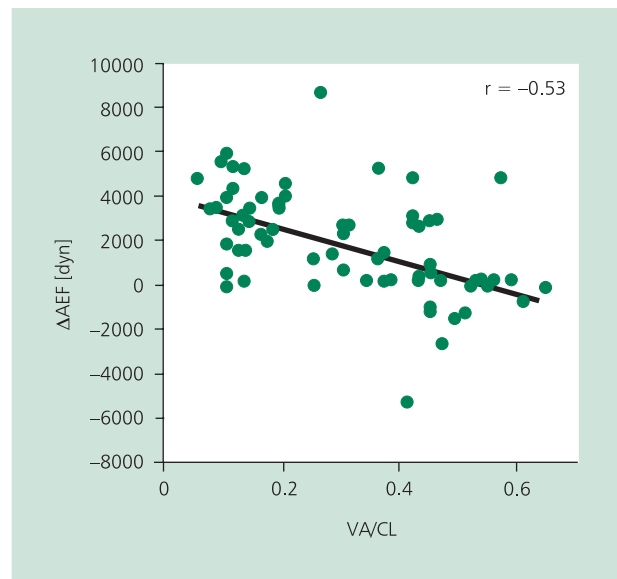
## DISCUSSION

The improvement of the LA systolic function and LA size reduction after RF ablation have been well demonstrated in patients with AF or atrial flutter [16, 17]. However, current knowledge concerning treatment of reentrant tachycardias of AVNRT or AVRT type on LA systolic function and remodelling is incomplete. This issue was a matter of only a few studies. Left atrial size reduction was reported after AVNRT

**Table 3.** Comparison of changes ( $\Delta$ ) of left atrial size and atrial ejection force (AEF) in both groups after ablation

| Parameter           | AVNRT group | WPW group | P      |
|---------------------|-------------|-----------|--------|
| $\Delta$ LA-AP [mm] | -3.7        | -1.5      | < 0.05 |
| $\Delta$ LA-ML [mm] | -3.6        | -2.4      | < 0.05 |
| $\Delta$ LA-IS [mm] | -6.0        | -4.2      | < 0.05 |
| $\Delta$ AEF [kdyn] | 2.97        | 0.54      | 0.001  |

Abbreviation as in Table 2.

**Figure 1.** Correlation between atrial ejection force increment ( $\Delta$ AEF) and ventriculo-atrial conduction (VA) time during arrhythmia in the study group**Figure 2.** Correlation between atrial ejection force increment ( $\Delta$ AEF) and tachycardia cycle length (VA/CL) ratio in the study group

and AVRT ablation [18], however, in the literature to date, there is a lack of data concerning the impact of ablation on the LA systolic function.

In the present study LA systolic function was assessed by AEF, which was introduced by Manning et al. [15]. The AEF value is calculated based on mitral inflow data acquired during echocardiographic study, included in a formula based on Newton's second law of dynamics. It is believed that AEF is a more sound index of LA systolic function compared to peak A wave velocity of the mitral inflow.

So far the AEF measurements were used chiefly for the assessment of atrial mechanics following AF cardioversion. Mattioli et al. [19] carried out a prospective observation in which AEF increased within 3 months of sinus rhythm restoration. Manning et al. [15] demonstrated that after successful AF cardioversion, AEF increased gradually only in those patients in whom no AF recurrence was observed. It has been assumed that AEF increase results from reversal of adverse effects of electromechanical remodelling of the atria.

Clinical utility of AEF is not limited to atrial systolic function assessment. In patients with sick sinus syndrome (SSS) it was demonstrated that lower AEF values are an independent risk factor of stroke [20]. Tokushima et al. [21] observed more frequent AF occurrence in patients with SSS and decreased AEF. Increased AEF was in turn observed in patients with hypertrophic cardiomyopathy with the LV outflow tract obstruction [22], and in patients with chronic heart failure [23].

In the present study, a significant increase of AEF function was noted after AVNRT ablation, accompanied by LA size reduction. The elimination of AVRT, however, caused LA size reduction without impact on LA systolic function. The LA size reduction was significantly greater in the AVNRT subgroup. Currently there are no commonly adopted normal values of the AEF, against which our results could be evaluated. It is known that age is one of the factors potentially influencing the AEF values. The AVNRT and AVRT subgroups were comparable in terms of patients' age, hence post-procedural changes of this parameter could not have been in-

fluenced by this factor. In studies by Manning et al. [15], the mean value of AEF in healthy volunteers was 16.3 kdynes.

The present study lacks a control group, to which AEF values measured in the study group could have been compared. However, AEF assessment was carried out in the same patient before and after ablation, which allowed for assessment of changes of the parameter resulting from the procedure. In our study group, low AEF values were noted pre-procedurally. These values were significantly improved only in the AVNRT subgroup, reaching values comparable to the results obtained by Mattioli et al. [19] in the group of patients after electrical cardioversion.

Our results demonstrate that AVNRT exerts a more unfavourable effect on the LA function in comparison with AVRT. This can be explained by different electrophysiological characteristics and different anatomical structure of the reentry loop, as reflected by shorter VA and lower VA/CL value in the AVNRT subgroup. Recurring AVNRT attacks cause a greater pressure increase in the atria accompanied by stronger atrial wall distension. This leads to more advanced remodelling and greater systolic function impairment when compared to AVRT patients. Hence, elimination of AVNRT brings about greater improvement in terms of LA systolic function and leads to greater LA size reduction, what was confirmed by the negative correlation between post-procedural AEF increase and VA time during tachycardia as well as VA/CL values.

In a proportion of AVNRT and AVRT patients, paroxysmal AF can occur [24, 25]. In our study, AF occurrence was one of the exclusion criteria. The relationship between AF, haemodynamic remodelling and electrophysiological parameters in AVNRT and AVRT patients was studied by other authors. Kalarus et al. [26] evaluated LA pressures during AVRT in patients with WPW. They showed that repeated atrial wall distension, secondary to pressure overload during AVRT, plays a significant role in the development of atrial cardiomyopathy, which promotes AF occurrence. These authors also demonstrated, that electrophysiological parameters of the tachycardia, with special emphasis on atrial and ventricular activation temporal sequence, are important factors of AF occurrence in WPW patients. During AVRT, in patients with AF, ventricular activation occurs markedly earlier and atrial activation markedly later than in patients without AF. It was also demonstrated that in AVNRT patients, inter-atrial conduction abnormalities and alterations of refraction promote AF [27].

### Limitations of the study

The number of patients in our study is limited. Due to the lack of more detailed data, the duration and frequency of arrhythmia recurrences and duration of palpitations were not analysed in either group. Those factors could have been of importance for the study results, as it can be assumed that longer and more frequent arrhythmias could have impaired LA function to a greater extent.

## CONCLUSIONS

1. Ablation of AVNRT improves LA function, whereas AVRT ablation does not exert such an effect.
2. After AVNRT ablation, LA size reduction was demonstrated and was significantly greater than after AVRT ablation.
3. The impact of ablation on the LA systolic function depends on electrophysiological parameters of the tachycardia (VA and VA/CL).

**Conflict of interest:** none declared

## References

1. Blomstrom-Lundqvist C, Scheinman MM, Aliot EM et al. ACC/AHA/ESC guidelines. Task force management of patients with supraventricular arrhythmias: executive summary. A report of the American College of Cardiology/American Heart Association task force on practice guidelines and the European Society of Cardiology committee for practice guidelines (writing committee to develop guidelines for the management of patients with supraventricular arrhythmias) developed in collaboration with NASPE-Heart Rhythm Society. *J Am Coll Cardiol*, 2003; 42: 1493–1531.
2. Scheinman MM, Huang S. The 1998 NASPE prospective catheter ablation registry. *PACE*, 2000; 23: 1020–1028.
3. Majewski J, Lelakowski J, Bednarek J et al. Odległa skuteczność zabiegów przezskórnej ablacji RF w leczeniu zaburzeń rytmu serca: doświadczenia własne. *Kardiologia Pol*, 2004; 61: II-70–II-75.
4. Alboni P, Fuca G, Paparella N et al. Hemodynamics of supraventricular reentrant tachycardia. *Eur J Cardiac Pacing Electrophysiol*, 1997; 7: 115–119.
5. Brugada P, Andries E. "Tachycardiomyopathy". The most frequently unrecognized cause of heart failure? *Acta Cardiol*, 1993; 48: 165–169.
6. Corey WA, Markel ML, Hoit BD et al. Regression of dilated cardiomyopathy after radiofrequency ablation of incessant supraventricular tachycardia. *Am Heart J*, 1993; 126: 1469–1473.
7. Gursoy S, Steurer G, Brugada J et al. Brief report: the hemodynamic mechanism of pounding in the neck in atrioventricular nodal reentrant tachycardia. *N Engl J Med*, 1992; 327: 772–774.
8. Kaye GC, Astridge P, Perrins J. Tachycardia recognition and diagnosis from changes in right atrial pressure waveform: a feasibility study. *PACE*, 1991; 14: 1384–1392.
9. Mele D, Alboni P, Fuca G et al. Atrioventricular nodal versus atrioventricular supraventricular reentrant tachycardias: characterization by an integrated Doppler electrophysiological hemodynamic study. *PACE*, 2000; 23: 2078–2085.
10. Goette A, Honeycutt C, Langberg JJ. Electrical remodeling in atrial fibrillation: time course and mechanisms. *Circulation*, 1996; 94: 2968–2974.
11. Nattel S, Li D. Ionic remodeling in the heart: Pathophysiological significance and new therapeutic opportunities for atrial fibrillation. *Circ Res*, 2000; 87: 440–447.
12. Kalbfleisch SJ, Strickberger SA, Williamson B et al. Randomized comparison of anatomic and electrogram mapping approaches to ablation of the slow pathway of atrioventricular node reentrant tachycardia. *J Am Coll Cardiol*, 1994; 23: 716–723.
13. Koźluk E, Walczak F, Szufladowicz E et al. Sygnały drogi wolnej i ich znaczenie jako wskaźnika właściwego położenia elektrody ablacynowej. *Folia Cardiol*, 2001; 8: 171–179.



14. Chen SA, Tai CT. Ablation of atrioventricular accessory pathways: current technique — state of the art. *PACE*, 2001; 24: 1795–1809.
15. Manning WJ, Silverman DI, Katz SE et al. Atrial ejection force: a noninvasive assessment of atrial systolic function. *J Am Coll Cardiol*, 1993; 22: 221–225.
16. Rodrigues AC, Scannavacca MI, Caldas MA et al. Left atrial function after ablation for paroxysmal atrial fibrillation. *Am J Cardiol*, 2009; 103: 395–398.
17. Rhee KS, Kang DH, Song JK et al. Restoration of atrial mechanical function after successful radiofrequency catheter ablation of atrial flutter. *Korean J Intern Med*, 2001; 16: 69–74.
18. Lelakowski J, Dreher A, Majewski J et al. Comparison of the selected electrophysiological and echocardiographical parameters in patients with atrio-ventricular reentrant tachycardia and atrioventricular nodal reentrant tachycardia treated with RF ablation. *Pol Przegl Kardiol*, 2009; 11: 85–91.
19. Mattioli AV, Castelli A, Bastia E et al. Atrial ejection force in patients with atrial fibrillation: comparison between DC shock and pharmacological cardioversion. *PACE*, 1999; 22: 33–38.
20. Mattioli AV, Tarabini Castellani E, Mattioli G et al. Stroke in paced patients with sick sinus syndrome: influence of left atrial function and size. *Cardiology*, 1999; 91: 150–155.
21. Tokushima T, Utsunomiya T, Yoshida K et al. Left atrial systolic function assessed by left atrial ejection force in patients with sick sinus syndrome and paroxysmal atrial fibrillation. *Jpn Heart J*, 2000; 41: 723–731.
22. Anwar AM, Soliman OI, Geleijnse ML et al. Assessment of left atrial ejection force in hypertrophic cardiomyopathy using real-time three-dimensional echocardiography. *J Am Soc Echocardiogr*, 2007; 20: 744–748.
23. Triposkiadis F, Harbas C, Sitafidis G et al. Echocardiographic assessment of left atrial ejection force and kinetic energy in chronic heart failure. *Int J Cardiovasc Imag*, 2008; 24: 15–22.
24. Brugada J, Mont L, Matas M et al. Atrial fibrillation induced by atrioventricular nodal reentrant tachycardia. *Am J Cardiol*, 1997; 79: 681–682.
25. Kalbfleisch SJ, El-Atassi R, Calkins H et al. Inducibility of atrial fibrillation before and after radiofrequency catheter ablation of accessory atrioventricular connections. *J Cardiovasc Electrophysiol*, 1993; 4: 499–503.
26. Kalarus Z, Lenarczyk R, Kowalski O et al. Influence of reciprocating tachycardia on the development of atrial fibrillation in patients with preexcitation syndrome. *Pacing Clin Electrophysiol*, 2007; 30: 85–92.
27. Lenarczyk R, Kowalski O, Pruszkowska-Skrzep P et al. Sequence of electrical activation, atrial remodeling and atrial fibrillation in patients with nodal reentrant tachycardia. *Acta Cardiol*, 2007; 62: 599–606.

# Wpływ ablacji prądem o wysokiej częstotliwości na czynność skurczową lewego przedsionka u chorych z nawrotnymi częstoskurczami węzłowymi i przedsionkowo-komorowymi

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## Streszczenie

**Wstęp:** Odmienne podłoże anatomiczne i charakterystyka elektrofizjologiczna częstoskurczu węzłowego (AVNRT) i przedsionkowo-komorowego (AVRT) są odpowiedzialne za różnice hemodynamiczne między tymi dwoma typami częstoskurczów nawrotnych. Podczas AVNRT ciśnienie w przedsionkach wzrasta w większym stopniu niż w trakcie AVRT. Wpływ obu typów częstoskurczu na remodeling i funkcję lewego przedsionka (LA) nie jest dobrze poznany. Brakuje danych na temat wpływu ablacji AVNRT i AVRT na czynność skurczową LA.

**Cel:** Celem pracy była ocena wpływu leczenia AVNRT i AVRT na czynność skurczową LA.

**Metody:** Badana grupa obejmowała 70 pacjentów (22 mężczyzn, 48 kobiet) w wieku śr. 43,9 roku, u których wykonano skuteczną ablację AVNRT (35 osób, podgrupa I) lub AVRT (35 osób, podgrupa II). Kryteriami wyłączenia z badań były: współistniejące choroby układu sercowo-naczyniowego, schorzenia przewlekłe, upośledzona funkcja skurczowa lewej komory, migotanie przedsionków, arytmie komorowe (> klasa III wg Lowna), stosowanie leków antyarytmicznych po ablacji. Analizie poddano następujące parametry z inwazyjnego badania elektrofizjologicznego i ablacji RF: czas sprężenia komorowo-predsionkowego (VA) podczas częstoskurczu, długość cyklu częstoskurczu (CL), stosunek VA/CL, liczbę wykonanych aplikacji RF. U wszystkich osób przed ablacją, a następnie po 6 miesiącach od zabiegu wykonano następujące badania: wywiad i badanie przedmiotowe, standardowe powierzchniowe EKG, 24-godzinne EKG metodą Holtera, przezklatkowe badanie echokardiograficzne. Ocenił: wymiar przednio-tylny LA (LA-AP) w projekcji przymostkowej w osi długiej, wymiar dolno-górny (oś długa) LA (LA-IS) oraz wymiar boczno-przyśrodkowy (oś krótka) LA (LA-ML) w projekcji koniuszkowej 4-jamowej. Funkcję skurczową LA oceniano na podstawie siły wyrzutowej LA, obliczanej wg wzoru Manninga:  $AEF = 0,5 \times \rho \times MA \times A^2$ , gdzie:  $\rho$ : współczynnik gęstości krwi = 1,06 g/cm<sup>3</sup>, MA: pole powierzchni ujścia mitralnego [cm<sup>2</sup>], A: maksymalna prędkość fali A napływu mitralnego.

**Wyniki:** Czas VA w całej badanej grupie wynosił śr. 107,1 ± 57,6 ms (25–225 ms). Był on znamienne krótszy w podgrupie I (śr. 53,7 ± 17,5 ms; 25–90 ms) w porównaniu z podgrupą II (160,6 ± 23,6 ms; 125–225 ms),  $p < 0,001$ ; CL wynosiła śr. 345,2 ± 49,2 ms (255–500 ms) i nie różniła się znamienne między podgrupami I (śr. 340,3 ± 52,4 ms; 275–435 ms) i II (350,1 ± 46 ms; 255–500 ms). Stosunek VA/CL wynosił śr. 0,3 ± 0,2 (0,06–0,66). Wartość VA/CL była znamienne niższa w podgrupie I (śr. 0,2 ± 0,1; 0,06–0,31) w porównaniu z podgrupą II (śr. 0,5 ± 0,1; 0,31–0,66);  $p < 0,001$ . Liczba aplikacji RF w całej badanej grupie wynosiła śr. 8,9 ± 7,6 (1–35). W podgrupie I wykonano śr. 8,6 ± 7,6 (1–33) aplikacji RF, a w podgrupie II śr. 9,2 ± 7,6 (1–35) aplikacji,  $p = NS$ . W podgrupie I stwierdzono istotne zmniejszenie wymiarów LA (LA-AP: 38 ± 3 v. 34 ± 2,9 mm, LA-ML: 37 ± 3,6 v. 33 ± 3,4 mm, LA-IS: 51 ± 4,1 v. 45 ± 5,2 mm;  $p < 0,001$ ) oraz przyrost AEF (7,78 ± 2,87 v. 10,75 ± 3,76 kdyn;  $p < 0,001$ ). W podgrupie II również obserwowano redukcję wielkości LA (LA-AP: 38 ± 3 v. 36,5 ± 3,1 mm;  $p < 0,01$ , LA-ML: 37 ± 2,8 v. 35 ± 3,4 mm,  $p < 0,001$ , LA-IS: 50 ± 3 v. 46 ± 3,9 mm;  $p < 0,001$ ), jednak AEF nie uległa istotnej zmianie po ablacji (8,96 ± 2,65 v. 9,50 ± 3,58 kdyn;  $p = NS$ ). Wymiary LA znamienne się zmniejszyły w podgrupie I ( $p < 0,05$ ). Różnica między podgrupami w zakresie zmiany AEF po ablacji była istotna ( $p < 0,001$ ). W całej badanej grupie wykazano ujemną korelację między przyrostem siły wyrzutowej LA ( $\Delta AEF$ ) a czasem VA ( $r = -0,51$ ). Stwierdzono również ujemną korelację między  $\Delta AEF$  a stosunkiem VA/CL ( $r = -0,53$ ). Liczba aplikacji RF nie wpływała na badane parametry.

**Wnioski:** 1. Ablacja AVNRT powoduje poprawę czynności skurczowej LA. 2. Ablacja AVRT nie wpływa istotnie na funkcję skurczową LA. 3. Po ablacji AVNRT wykazano zmniejszenie wymiarów LA istotnie większe niż po ablacji AVRT. 4. Wpływ ablacji na funkcję skurczową LA zależy od parametrów elektrofizjologicznych częstoskurczu (VA i VA/CL).

**Słowa kluczowe:** ablacja RF, AVNRT, AVRT, lewy przedsionek

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