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ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

Does a high percentage of right ventricular pacing influence the incidence of paroxysmal atrial fibrillation in myotonic dystrophy type 1 patients?

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Abstract

Background: Paroxysmal atrial tachyarrhythmias occur frequently in myotonic dystrophy type 1 (MD1) patients. Pacemakers, implanted for the treatment of bradyarrhythmias and including detailed diagnostic functions, may facilitate the diagnosis and management of frequent paroxysmal atrial fibrillation (AF) that may remain undetected during a conventional clinical follow-up. The effect of right ventricular pacing on AF incidence is still controversial.

Aim: To evaluate the influence of a high percentage of right ventricular pacing on AF in MD1 patients during a 12-month follow-up period.

Methods: We enrolled in the present study 70 MD1 patients (age 51.3 ± 5 years; 32 females) who underwent dual chamber pacemaker implantation. At 12 months of follow-up, the study population was divided into three groups according to the percentage of atrial and ventricular stimulation: Group 1, the atrial sensing ventricular sensing group (ASVS; n = 22; age 52 ± 7.7; eight female) with a percentage of atrial and ventricular stimulation lower than 50%; Group 2, the atrial sensing ventricular pacing group (ASVP; n = 24; age 50.5 ± 7.6; 13 female) with a percentage of atrial stimulation lower than 50% and percentage of ventricular stimulation higher than 80%; and Group 3, the atrial pacing ventricular pacing group (APVP; n = 24; age 56 ± 4.3; 11 female) with a percentage of atrial and ventricular stimulation higher than 80%. We counted the number of episodes of atrial arrhythmia that occurred during the observation period and the duration of each episode.

Results: We found a statistically significant difference in the number and duration of AF episodes between the three groups at the 12-month follow-up. In particular, there were more episodes (253 ± 30 vs. 80 ± 27 vs. 53 ± 32; $p < 0.03$) and longer durations of AF (8,700 ± 630 vs. 4,480 ± 975 vs. 3,853 ± 870 min; $p < 0.03$) in the ASVP group than in the ASVS group and the APVP group. Lead parameters remained stable over time and there were no displacements of the electrodes after implantation.

Conclusions: In a 12-month follow-up comparison, we showed a statistically significant increase in paroxysmal AF episodes in MD1 patients with a high percentage of right ventricular pacing and a lower percentage of atrial stimulation.

Key words: ventricular pacing, myotonic dystrophy, pacemaker, arrhythmias, atrial fibrillation

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INTRODUCTION

Myotonic dystrophy type 1 (MD1), or Steinert's disease, is the most common muscular dystrophy in adult life with an incidence of 1 in 8,000 births [1, 2]. It is an autosomal dominant disorder caused by an abnormal expansion of an unstable trinucleotide repeat in the 3-prime untranslated region of the

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DMPK gene on chromosome 19 [3, 4]. The phenotype is characterised by myotonia and muscle weakness, but multisystem involvement is frequent. Cardiac involvement is noticed in about 80% of cases, and it often precedes the skeletal muscle one. Heart block is the first and most clinically significant cardiac disease in this group of patients [5]. To prevent cardiac sudden death, implantation of a pacemaker (PM) is required in 3–22% of cases [6, 7]. Paroxysmal atrial arrhythmias (atrial fibrillation [AF], atrial flutter, atrial tachycardia) frequently occur in MD1 patients [8, 9]. In our previous study, we showed the protective effect of atrial preference pacing on paroxysmal AF incidence in MD1 patients [10]. PM may facilitate the diagnosis and management of frequent paroxysmal atrial tachyarrhythmias that may remain undetected during conventional clinical follow-up [11]. There is much clinical evidence [12–14] that right ventricular (RV) pacing increases the risk of AF in patients with sinus node disease during a long-term follow up. The aim of our study was to evaluate the influence of a high percentage of RV pacing on paroxysmal AF in MD1 patients during a 12-month follow-up period.

METHODS

Study population

From a large cohort of 200 patients referred to the Cardiomyology and Genetic Section of the Second University of Naples, we studied 70 MD1 patients (age 51.3 ± 5; 32 females) with conduction disorders who underwent dual chamber PM implantation in our division. A molecular genetic test was systematically performed in all patients to evaluate the CTG triplet expansion and to confirm the diagnosis of MD1. DNA was extracted from peripheral blood samples; the diagnosis was made in the presence of an expansion of the CTG triplet over 50. Before implantation of PM, physical examination, 12-lead electrocardiogram (ECG), 24-hour ECG Holter monitoring, echocardiogram and cardiac invasive electrophysiological study (EPS) were performed.

We excluded from the study all MD1 patients with patent foramen ovale, atrial septal aneurysm, severe mitral stenosis or regurgitation, left atrial enlargement, paroxysmal AF or who had undergone prior surgery involving the right atrium, coronary bypass or valvular heart surgery, sick sinus syndrome, or inducible ventricular tachycardia. Subjects with a history of hypertension (systolic and diastolic blood pressure > 140/90 mm Hg), diabetes mellitus or impaired glucose tolerance, obesity, electrolyte imbalance, systolic and diastolic dysfunction, connective tissue disorders, hepatic, renal, thyroid diseases, and sleep disorders were excluded from the study. All patients were in sinus rhythm, and none of them was taking statins, anticoagulants, antiaggregate medications or medications known to affect electrocardiographic intervals (antiarrhythmic agents, tricyclic antidepressants, antihistamines or antipsychotics). All patients were taking angiotensin-converting enzyme-inhibitors or sartans medications, because it has been shown that these drugs reduce myocardial damage and have a protective effect on cardiovascular remodelling. The indication for PM implantation was first degree atrioventricular (AV) block with a pathological infra-Hissian conduction in 20 patients — six patients of the atrial sensing ventricular sensing (ASVS) group; seven patients of the atrial sensing ventricular pacing (ASVP) group; seven patients of the atrial pacing ventricular pacing (APVP) group; symptomatic type 1 or 2 second degree AV block respectively in 23 (seven patients of the ASVS group; eight patients of the ASVP group; eight patients of the APVP group and 25 patients (seven patients of the ASVS group; nine patients of the ASVP group; and nine patients of the APVP group); paroxysmal third degree AV block in two patients (both of the ASVP group). To minimise confounding variables, with different electrode materials and interelectrode spacing, the identical model lead was used in all the patients. Similarly, PMs with identical behaviour and telemetric capabilities were used to assure accuracy in comparing measurements between patients.

The study was conducted according to the Declaration of Helsinki. Written informed consent was obtained from the patients before an implant, as approved by the institution's medical ethical committee.

Pacemaker system and programmed parameters

We used the standard technique for the implantation of a dual-chamber PM system (Medtronic Kappa D901, or Adapta ADDR01, Medtronic Inc., Minneapolis, MN, USA), performing percutaneous subclavian vein cannulation in all cases. First, the RV lead (Medtronic 4074 CapSure Sense) was positioned in the apex under fluoroscopic guidance. All MD1 patients received the bipolar atrial screw-in pacing lead CapSureFix® 5076 (Medtronic Inc., Minneapolis, MN, USA). The atrial pacing lead was positioned in the right atrial appendage or on the right side of the interatrial septum in the region of Bachmann's bundle, according to the optimal site defined as the location with lowest pacing and highest sensing thresholds. To minimise atrial lead oversensing, the sensitivity configuration was bipolar. The devices were programmed in DDD or AAI-DDD mode with an AV delay of 120/150 ms. The lower rate was set to 50 bpm, the upper rate to 120 bpm. Mode switches were programmed to occur for atrial rates > 200 bpm persisting for more than 12 ventricular beats. All the special algorithms to prevent AF were disabled. The devices used in this study were programmed to detect episodes of atrial tachycardia, and to record summary and detailed data, including atrial and ventricular electrograms.

The endpoints

The endpoints assessed in our study were the number and the total duration of AF episodes between the high synchronous ventricular pacing percentage group (ASVP), the sinus rhythm group (ASVS) and the dual chamber stimulation group (APVP) in MD1 patients during a 12-month follow-up.

Follow-up

Patients were discharged two days post implantation after confirming the electrical lead parameters. They were re-examined for the status of the wound at the site of PM implantation after 10 ± 2 days and were followed up at one, six and 12 months thereafter. They underwent clinical assessment, standard 12-lead ECG, 24 h-Holter monitoring, echocardiogram and assessment of device performance at every visit. Measurements of intrinsic P and R wave voltage, atrial and ventricular pacing threshold at a pulse duration of 0.4 ms, bipolar pacing impedance at 5 V and 0.4 ms were recorded at follow-up intervals. All PM models provided the same features for the measurement of pacing and sensing thresholds, and pacing impedance. The first two parameters were measured using semi-automatic function tests and the PM calculated the pacing impedance itself on interrogation. We counted the percentage of atrial and ventricular pacing in synchronous rhythm, the number and the total duration of AF episodes that occurred during the observation period.

For each AF episode, the device stored simultaneous atrial and ventricular electrograms. The number of episodes of AF was counted by two independent observers, both blinded to the patients' group location (i.e. ASVS or ASVP or APVP). Atrial tachycardia episodes, identified by regular atrial activity, were excluded from the analysis. At 12-month follow-up, we subdivided the synchronous ventricular beats into those preceded by an atrial sensed or paced event giving counts for the following: atrial sense ventricular pace (ASVP) and atrial pace ventricular pace (APVP).

Statistical analysis

Continuous variables are expressed as mean ± standard deviation. Statistical analysis was performed using Student's t-test for unpaired data, Fisher's exact test for comparing proportions and one-way analysis of variance (ANOVA) for multiple comparisons. P values < 0.05 were considered to be statistically significant. To identify possible significant independent determinants of AF incidence in MD1 patients with a high percentage of ventricular pacing, the individual association with clinical, ECG and EPS variables was assessed by multivariable logistic regression analysis. The following variables were included in the analysis: age, sex, PR interval, QRS duration and HV interval. Variable selection was performed in the multivariable logistic regression as an interactive stepwise backward elimination method, each time excluding the one variable with the highest p-value according to Wald statistics. Analyses were performed using the statistical package SPSS 11.0 software for Windows SPSS Inc. (Chicago, IL, USA).

RESULTS

All patients completed the 12-month follow-up, at which time the study population was retrospectively subdivided into three groups: ASVS (n = 18; age 52 ± 7.7; nine females) with a percentage of atrial and ventricular stimulation lower than 50%; ASVP (n = 16; age 50.5 ± 7.6; ten females) with a percentage of atrial stimulation lower than 50% and a percentage of ventricular stimulation higher than 80%; and APVP (n =16; age 56 ± 4.3; six females) with a percentage of atrial and ventricular stimulation higher than 80%. There were no statistically significant differences between the age and sex compositions of the groups and the medication intakes (Table 1). We found a statistically significant difference in the number and duration of AF episodes between the three groups at 12-month follow-up (Fig. 1A, B). In particular, there were more episodes (253 ± 30 vs. 80 ± 27 vs. 53 ± 32; $p < 0.03$) and longer durations of AF (8,700 ± 630 vs. 4,480 ± 975 vs. 3,853 ± 870 min; $p < 0.03$) in the ASVP group than in the ASVS group and the APVP group (Fig. 1A, B). No AF sustained episode that required clinical intervention prior to scheduled follow-up was reported. No statistically significant difference in ejection fraction was found at 12-month follow-up (55 ± 4 vs. 54 ± 3 vs. 53 ± 2%; $p = 0.4$). There was no significant difference in the lead pacing capture, sensing threshold or lead impedances at implant and at 12-month follow-up. Lead parameters remained stable over time and there were no lead-related complications. Multivariate logistic regression analysis did not show any significant effect of age, sex, PR interval, QRS duration or HV interval on the occurrence of AF in MD1 patients with a high percentage of ventricular pacing (Table 2).

DISCUSSION

In our clinical experience, MD1 patients who underwent PM implantation manifested during the follow-up period a high number of paroxysmal atrial arrhythmias. According to the data in the literature, our observations confirm their frequent occurrence in patients with myotonic dystrophy [15]. Cardiac involvement in MD1 patients is not limited to the specialised conduction system, as initially proposed, and cardiac myopathy, characterised by progressive selective fibrosis and scar replacement of initially unaffected areas, may be part of this disease. This anatomopathological substrate may facilitate the onset and perpetuation of AF in MD1 patients. RV apical pacing may facilitate the onset of AF through the following mechanisms, which indirectly influence cardiac performance: 1) ventricular pacing disruptions to left-sided AV timing due to interventricular conduction delays; 2) functional mitral regurgitation due to AV timing disruptions or ventricular conduction delay; and 3) reduction in left ventricular pump function due to ventricular conduction delay (desynchronisation).

According to our previous studies, Bachmann's bundle stimulation is a safe and feasible procedure in MD1 patients [16], but it seems not to provide a significant benefit for the prevention of paroxysmal AF [17]; therefore no evaluation of ventricular pacing effect on AF incidence was performed.

Table 1. Patient characteristics at 12-month follow-up

	ASVP group	ASVS group	APVP group	P
Patients	24	22	24	
Age [years]	50.5 ± 7.6	52 ± 7.7	56 ± 4.3	0.3
Sex (male/female)	11/13	14/8	13/11	
Ejection fraction [%]	55 ± 4	54 ± 3	53 ± 2	0.4
PA interval [ms]	24 ± 7	21 ± 12	21±10	0.2
AH interval [ms]	140 ± 30	135 ± 50	130 ± 50	0.3
HV interval [ms]	78 ± 12	55 ± 25	85 ± 20	0.001
Atrial diameter [mm]	37 ± 4	35 ± 6	36 ± 3	0.5
Atrial volume [mL]	43 ± 10	39 ± 6	41 ± 7	0.4
Regurgitant mitral orifice area [cm^2]	0.18 ± 0.01	0.15 ± 0.05	0.16 ± 0.03	0.3
Atrial pacing [%]	23 ± 2	14 ± 3	93 ± 10	0.03
Ventricular pacing [%]	95 ± 3	10 ± 4	85 ± 5	0.05
Ventricular pacing rate [bpm]	74 ± 12	69 ± 5	67 ± 5	0.06
Atrial fibrillation episodes	253 ± 30	80 ± 27	53 ± 32	0.03
Total duration atrial fibrillation [min]	8,700 ± 630	4,480 ± 975	3,853 ± 870	0.03
ACEI therapy [%]	11	13	15	0.4
Sartan therapy [%]	7	6	8	0.3
Magnesium pidolate [%]	8	7	8	0.5
Coenzyme Q10 [%]	5	4	6	0.3

ASVS — atrial sensing ventricular sensing; ASVP — atrial sensing ventricular pacing; APVP — atrial pacing ventricular pacing; ACEI — angiotensin converting enzyme inhibitor

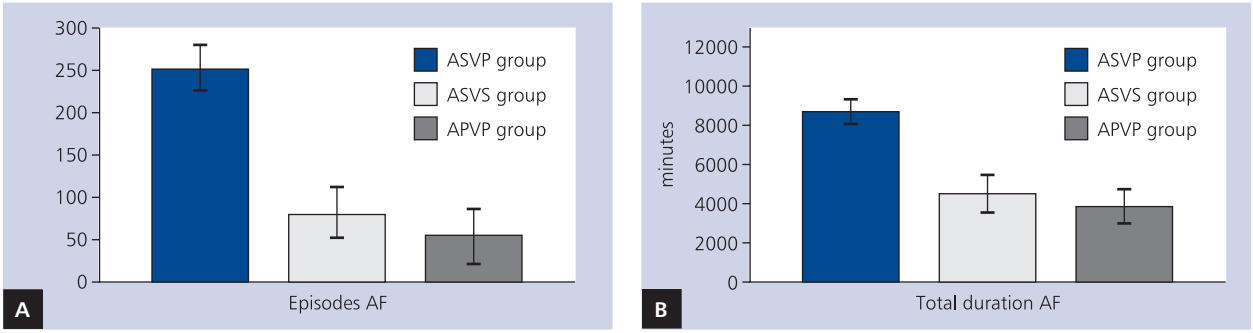

Figure 1. Number (**A**) and duration (**B**) of atrial fibrillation (AF) episodes in each group of the study population; abbreviations as in Table 1

Table 2. Multivariate analysis for atrial fibrillation incidence in myotonic dystrophy type 1 patients with a high percentage of ventricular pacing

Variables	Odds ratio	95% confidence interval	P
Age	1.2	0.6–1.7	0.5
Male sex	1.02	0.5–1.8	0.9
PR interval	0.66	0.5–1.1	0.08
QRS duration	1.27	0.8–2.4	0.7
HV duration	0.75	0.6–1.4	0.9

The aim of our study was to evaluate the long term effect of a high percentage of RV stimulation on AF incidence in MD1 patients who underwent dual-chamber PM during the 12-month follow-up period.

Ventricular pacing and atrial fibrillation

There is growing evidence that RV pacing increases the risk of AF in the long term. An analysis of the Mode Selection Trial (MOST) demonstrated a linearly increasing risk of AF with cumulative percentages of ventricular pacing (Cum%VP) in DDDR and VVIR modes up to ≈ 80% to 85%. The mag-

nitude of increased risk was ≈1% for each 1% increase in Cum%VP and was similar between pacing modes. This increased risk of AF, associated with increased Cum%VP in both modes, persisted when the models were adjusted for all other known baseline predictors of AF in the study population [12]. Sweeney et al. [13] concluded that the ventricular desynchronisation imposed by RV apical pacing, even when AV synchrony is preserved, increases the risk of heart failure and AF in patients with sinus node dysfunction and normal baseline QRS duration.

It is now almost universally accepted that the adverse effects of RV pacing are due to ventricular desynchronisation, similar to left bundle branch block. The hypothesis that these effects are due to a physiological consequence of RV pacing itself would seem to be supported by subsequent clinical investigation, demonstrating that severe minimisation of Cum%VP, using newer dual chamber pacing platforms, reduces the risk of persistent AF compared to high Cum%VP with conventional dual chamber pacing.

Nevertheless, Silberbauer et al. [14] demonstrated no linkage between atrial-synchronous %VP and AF burden among 554 patients from two clinical trials of PM suppression therapy: the Atrial Fibrillation Therapy (AFT) study and the Pacemaker Atrial Fibrillation Suppression (PAFS) study [18, 19]. The authors concluded that Cum%VP does not influence the risk of paroxysmal AF burden. Several comments regarding this study are necessary. Unlike previous studies [12, 13], which enrolled a large cohort of typical sinus node disease patients, Silberbauer et al. [14] targeted a small sample size patients with drug-resistant symptomatic AF, who were likely to have high AF incidence regardless of how they were managed. Veasey et al. [20] showed no significant reduction in AF burden with any minimal ventricular pacing algorithms compared to conventional DDD pacing, although the follow-up period of this study was relatively short and patients had high levels of ventricular pacing for only a two-month phase.

Main findings

The main finding of our study is that there was a significant increase in the number and the duration of AF episodes in MD1 patients with an RV stimulation percentage higher than 80% and an atrial stimulation percentage lower than 50% during the 12-month follow-up.

The potential reasons for these findings in MD1 patients are that the specific histopathological pattern, characterised by diffuse fibrosis, myocyte hypertrophy and fatty acid infiltration, should increase the AV timing disruptions or ventricular conduction delay. The lack of correlation on multivariate analysis between baseline ECG and EPS conduction severity parameters and AF incidence suggested that in MD1 patients with a high percentage of ventricular pacing, the increased AF burden was not related to worse severity of conduction disease and cardiac fibrosis, but to ventricular pacing *per se*.

It's interesting to note that a bicameral stimulation percentage higher than 80% decreased the occurrence and duration of AF episodes in respect of a high monocameral stimulation percentage. Probably the high percentage of atrial pacing had a protective effect against AF development and it successfully counteracted the propensity for AF expected with a high percentage of ventricular pacing. Atrial pacing may prevent the onset of AF through the following mechanisms: prevention of the relative bradycardia that triggers paroxysmal AF; prevention of the bradycardia-induced dispersion of refractoriness; suppression or reduction of premature atrial contractions which initiate re-entry and predispose to AF; and preservation of AV synchrony, which may prevent switch-induced changes in atrial repolarisation, predisposing to AF [21, 22]. Our results have potentially important clinical implications. The majority of MD1 patients who underwent PM implantation have a normal ventricular activation sequence reflected in QRS duration < 120 ms. Ventricular desynchronisation caused by a high percentage of RV apical pacing in the VDD mode (ASVP group) increases the incidence of AF. When sinus function is preserved in MD1 patients, the AF risk may be reduced by minimal ventricular pacing strategies that preserve the normal ventricular activation sequence as much as possible.

Limitations of the study

A more extensive study, including a greater number of patients, is necessary to confirm these preliminary findings. The episode detection criteria used in this study (atrial rate > 200 min – 1 for 12 or more ventricular beats) includes short arrhythmias with limited clinical relevance. A very small number of regular tachycardias were excluded from the analysis. The pacing percentage cut-offs used to subdivide the population study were arbitrary; our study excluded patients with less than 50% atrial pacing and between 50% and 80% ventricular pacing, because no patients were observed in this window. During the follow-up period, we performed several ECG recordings and 24 h-Holter monitoring to verify the true RV capture; however, we cannot exclude the possibility of RV pseudofusion. The present study evaluated the influence of a high percentage of RV apical pacing on AF in MD1 patients during a 12-month follow-up period; further studies are necessary to assess the possible relationship between alternative ventricular pacing sites or biventricular pacing and AF incidence.

This study does not demonstrate a definitive protective effect of atrial pacing because of the lack of patients with a high percentage of atrial pacing and a low percentage of ventricular pacing (the APVS group). Further data is necessary to confirm our preliminary observations and to determine the effectiveness of atrial pacing to prevent AF.

CONCLUSIONS

In a 12-month follow-up comparison, we showed a statistically significant increase in paroxysmal AF episodes in MD1 patients

with a high percentage of RV pacing and a lower percentage of atrial stimulation.

Conflict of interest: *none declared*

References

1. Phillips MF, Harper PS. Cardiac disease in myotonic dystrophy. Cardiovasc Res, 1997; 33: 13–22.
2. Harley HG, Brook JD, Rundle SA et al. Families with myotonic dystrophy with and without cardiac involvement. Arch Intern Med, 1983; 143: 2134–2136.
3. Rotundo IL, Faraso S, De Leonibus E et al. Worsening of cardiomyopathy using deflazacort in an animal model rescued by gene therapy. PLoS One, 2011; 6: 9.
4. Lancioni A, Rotundo IL, Kobayashi YM et al. Combined deficiency of alpha and epsilon sarcoglycan disrupts the cardiac dystrophin complex. Hum Mol Genet, 2011; 20:4644–4654.
5. Nguyen HH, Wolfe JT III, Holmes DR Jr, Edwards WD. Pathology of the cardiac conduction system in myotonic dystrophy: a study of 12 cases. J Am Coll Cardiol, 1988; 11: 662–671.
6. Gregoratos G, Abrams J, Epstein AE et al. ACC/AHA/NASPE 2002 guideline update for implantation of cardiac pacemakers and antiarrhythmia devices: Summary article: A report of the American college of Cardiology/American Heart Association Task Force on Practice Guidelines (ACC/AHA/NASPE Committee to Update the 1998 Pacemaker Guidelines). Circulation, 2002; 106: 2145–2161.
7. Russo V, Rago A, D'Andrea A, Politano L, Nigro G. Early onset "electrical" heart failure in myotonic dystrophy type 1 patient: the role of ICD biventricular pacing. Anadolu Kardiyol Derg, 2012; 12: 517–519.
8. Russo AD, Mangiola F, Della Bella P et al. Risk of arrhythmias in myotonic dystrophy: trial design of the RAMYD study. J Cardiovasc Med (Hagerstown), 2009; 10: 51–58.
9. Cudia P, Bernasconi P, Chiodelli R et al. Risk of arrhythmia in type I myotonic dystrophy: the role of clinical and genetic variables. J Neurol Neurosurg Psychiatry, 2009; 80: 790–793.
10. Russo V, Rago A, Politano L et al. The effect of atrial preference pacing on paroxysmal atrial fibrillation incidence in myotonic dystrophy type 1 patients: a prospective, randomized, single-bind cross-over study. Europace, 2012; 14: 486–489.
11. Lazarus A, Varin J, Babuty D et al. Long-term follow-up of arrhythmias in patients with myotonic dystrophy treated by pacing: a multicenter diagnostic pacemaker study. J Am Coll Cardiol, 2002; 40: 1645–1652.
12. Sweeney MO, Hellkamp AS, Ellenbogen KA et al. Adverse effect of ventricular pacing on heart failure and atrial fibrillation among patients with normal baseline QRS duration in a clinical trial of pacemaker therapy for sinus node dysfunction. Circulation, 2003; 23: 2932–2937.
13. Sweeney MO, Bank AJ, Nsah E et al. Minimizing ventricular pacing to reduce atrial fibrillation in sinus-node disease. N Engl J Med, 2007; 357: 1000–1008.
14. Silberbauer J, Veasey RA, Freemantle N et al. The relationship between high-frequency right ventricular pacing and paroxysmal atrial fibrillation burden. Europace, 2009; 11: 1456–1461.
15. Lazarus A, Varin J, Onnoughene Z et al. Relationships among electrophysiological findings and clinical status, heart function and extent of DNA mutation in myotonic dystrophy. Circulation, 1999; 99: 1041–1046.
16. Nigro G, Russo V, Politano L et al. Right atrial appendage versus Bachmann's bundle stimulation: a two-year comparative study of electrical parameters in myotonic dystrophy type-1 patients. Pacing Clinical Electrophysiol, 2009; 32: 1191–1196.
17. Nigro G, Russo V, Politano L et al. Does Bachmann's bundle pacing prevent atrial fibrillation in myotonic dystrophy type 1 patients? A 12 months follow-up study. Europace, 2010; 12: 1219–1223.
18. Camm AJ, Sulke N, Edvardsson N et al. Conventional and dedicated atrial overdrive pacing for the prevention of paroxysmal atrial fibrillation: the AFTherapy study. Europace, 2007; 9: 1110–1118.
19. Sulke N, Silberbauer J, Boodhoo L et al. The use of atrial overdrive and ventricular rate stabilization pacing algorithms for the prevention and treatment of paroxysmal atrial fibrillation: the Pacemaker Atrial Fibrillation Suppression (PAFS) study. Europace, 2007; 9: 790–797.
20. Veasey RA, Arya A, Freemantle N et al. The usefulness of minimal ventricular pacing and preventive AF algorithms in the treatment of PAF: the "MinVPace" study. J Interv Card Electrophysiol, 2010; 28: 51–57.
21. Sopher SM, Camm AJ. Atrial pacing to prevent atrial fibrillation? J Intervent Card Electrophysiol, 2000; 4 (suppl. I): 149–153.
22. Mehra R, Hill MRS. Prevention of atrial fibrillation/flutter by pacing techniques. In: Saksena S, Luderlitz B eds. Interventional electrophysiology: a textbook. 2nd Ed. Futura Publishing, Armonk, NY 1996: 521–540.

Czy większy odsetek stymulacji prawej komory wpływa na częstość występowania epizodów migotania przedsionków u chorych z dystrofią miotoniczną typu 1?

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Streszczenie

Wstęp: Napadowe tachyarytmie przedsionkowe występują często u chorych na dystrofię miotoniczną typu 1 (MD1). Stymulatory serca, wszczepiane w celu leczenia bradyarytmii, są wyposażone w funkcje umożliwiające szczegółową diagnostykę i mogą ułatwić rozpoznawanie i leczenie przypadków częstego napadowego migotania przedsionków (AF), które mogły zostać niewykryte w czasie rutynowej oceny klinicznej. Wpływ stymulacji prawej komory na występowanie AF nadal jest przedmiotem dyskusji.

Cel: Celem badania była ocena wpływu wysokiego odsetka stymulacji prawej komory na AF u chorych z MD1 w okresie rocznej obserwacji.

Metody: Do badania włączono 70 chorych z MD1 (wiek 51,3 ± 5; 32 kobiety), którym wszczepiono dwujamowy stymulator serca. Po 12 miesiącach obserwacji badaną populację podzielono na 3 grupy w zależności od odsetka stymulacji przedsionków i komór: grupa ASVS (*atrial sensing ventricular sensing*, wyczuwanie przedsionkowe, wyczuwanie komorowe; n = 22; wiek 52 ± 7,7; 8 kobiet), w której odsetek stymulacji przedsionków i komór był mniejszy niż 50%; grupa ASVP (*atrial sensing ventricular pacing*, wyczuwanie przedsionkowe, stymulacja komór; n = 24; wiek 50,5 ± 7,6; 13 kobiet), w której odsetek stymulacji przedsionków był mniejszy niż 50%, a odsetek stymulacji komór większy niż 80%; grupa APVP (*atrial pacing ventricular pacing*, stymulacja przedsionków, stymulacja komór; n = 24; wiek 56 ± 4,3; 11 kobiet), w której odsetek stymulacji przedsionków i komór był większy niż 80%. Określono liczbę epizodów arytmii przedsionkowej, które wystąpiły w okresie obserwacji i czas trwania każdego epizodu.

Wyniki: Wykazano statystycznie istotną różnicę w liczbie i czasie trwania epizodów AF między trzema grupami w okresie 12-miesięcznej obserwacji. W grupie ASVP stwierdzono większą liczbę (253 ± 30 *vs.* 80 ± 27 *vs.* 53 ± 32; $p < 0,03$) i dłuższy czas trwania epizodów AF (8700 ± 630 *vs.* 4480 ± 975 *vs.* 3853 ± 870 min; $p < 0,03$) niż w grupach ASVS i APVP. Parametry odprowadzeń nie zmieniły się z upływem czasu; nie stwierdzono również przemieszczenia się elektrod po implantacji.

Wnioski: Na podstawie porównania danych z rocznej obserwacji wykazano statystycznie istotne zwiększenie częstości epizodów napadowego AF u chorych z MD1, u których występował duży odsetek stymulacji prawej komory i mniejszy odsetek stymulacji przedsionków.

Słowa kluczowe: stymulacja komór, dystrofia miotoniczna, stymulator serca, zaburzenia rytmu, migotanie przedsionków

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