Right ventricular apex versus right ventricular outflow tract pacing: prospective, randomised, long-term clinical and echocardiographic evaluation

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Abstract

Introduction: In patients treated with permanent pacing, the electrode is typically placed in the right ventricular apex (RVA). Published data indicate that such electrode placement leads to an unfavourable ventricular depolarization pattern, while right ventricular outflow tract (RVOT) pacing seems to be more physiological.

Aim: To compare long-term effects of RVOT versus RVA pacing on clinical status, left ventricular (LV) function, and the degree of atrioventricular valve regurgitation.

Methods: Patients with indications for permanent pacing, admitted to hospital between 1996 and 1997, were randomised to receive RVA or RVOT pacing. In 2004 during a final control visit in 27 patients clinical status, echocardiographic parameters and QRS complex duration as well as NT-proBNP level were measured. Analysed parameters were compared between groups and in the case of data available during the perioperative period also their evolution in time was assessed.

Results: Out of 27 patients 14 were randomised to the RVA group and 13 to the RVOT group. No significant differences between groups were observed before the procedure with respect to age, gender, comorbidities or echocardiographic parameters. Mean duration of pacing did not differ significantly between the groups (89±9 months in RVA group vs 93±6 months in RVOT group, NS). In the RVA group significant LV ejection fraction decrease was observed (from 56±11% to 47±8%, $p < 0.05$); in the RVOT group LV ejection fraction did not change (54±7% and 53±9%; NS). Progression of tricuspid valve regurgitation was also observed in the RVA group but not in the RVOT group. During the final visit NT-proBNP level was significantly higher in the RVA group: 1034±852 pg/ml *vs* 429±430 pg/ml ($p < 0.05$).

Conclusions: In patients with normal LV function permanent RVA pacing leads to LV systolic and diastolic function deterioration. RVOT pacing can reduce the unfavourable effect and can slow down cardiac remodelling caused by permanent RV pacing. Clinical and echocardiographic benefits observed in the RVOT group after 7 years of pacing are reflected by lower NT-proBNP levels in this group of patients.

Key words: right ventricular apex, right ventricular outflow tract, pacing

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Introduction

In patients treated with permanent pacing, the electrode is typically placed in the right ventricular apex (RVA), whereas the results of clinical and experimental studies indicate that long-term RVA pacing disturbs synchronisation of left ventricular activation (LV), which in turn leads to impairment of its systolic and diastolic function, regional perfusion disorders, deterioration of segmental contractile function and adrenergic innervation [1-6]. There is also some evidence in the medical literature that RVA pacing leads to morphological and histological changes in the heart [7-9].

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Hence, there is a tendency to look for new, "better" regions of pacing. The area of the RV outflow tract (RVOT) seems to be the most interesting, but the results of clinical studies in which RVOT pacing was evaluated are inconsistent [10-17]. Thus de Cock et al. [18] performed a metaanalysis of nine prospective studies evaluating RVOT and RVA pacing effects on LV systolic function. The authors of this metaanalysis indicated moderate (34%), althought statistically significant beneficial haemodynamic effects of RVOT pacing in comparison with RVA pacing in different groups of patients.

There are few reports in the medical literature on long-term RVOT pacing effects [16, 19-22], and the follow-up was limited in these studies. The longest observation – 18 months – was reported by Tse et al. [21]. RVOT and RVA pacing effects on perfusion and LV function were evaluated in this study, but clinical effects of pacing were not assessed.

The aim of the present prospective, randomised study was long-term evaluation of RVOT and RVA pacing effects on clinical status, LV systolic and diastolic function, degree of atrio-ventricular (a-v) valve regurgitation and QRS complex duration in patients treated with permanent ventricular or dual chamber pacing.

Methods

Patients

Patients with indications for permanent pacing, who required VDD, DDD or VVI/R pacemaker implantation, were enrolled in the study. None of the patients before inclusion in the study had been treated with permanent pacing. All patients gave written consent to participate in the study. The study protocol was accepted by the Ethics Committee.

Pacemaker implantation procedure

Patients were randomised to RVOT (RVOT group) or RVA (RVA group) pacing before implantation. Available at the time of the study passive fixation electrodes were used for RVOT and RVA pacing. The atrial lead was fixed for the right appendage. The RVOT lead was introduced through the tricuspid valve into RV and further into the pulmonary trunk. Afterwards, it was withdrawn slowly until the tip of the electrode was placed below the pulmonary valve on the interventricular septum (IVS), two thirds of the way between RVA and pulmonary valve in AP projection and directed towards IVS in LAO projection ("high" IVS). Localisation of the electrode was verified in fluoroscopy (in AP, LAO and RAO projection) and 12-lead ECG recordings.

Electrode localisation, which gave in ECG paced QRS with LBBB-like morphology and QRS electrical axis near 90° in the frontal plane, was assumed an optimal place of RVOT pacing.

Follow-up

Follow-up examination was conducted at 1, 3, 6 and 9 months after implantation, and subsequently once a year. During the final control visit complete analysis was performed; it included the evaluation of patients' clinical status with focus on heart failure symptoms and the presence of permanent atrial fibrillation. Additionally, 12-lead ECG recordings, chest X-ray to confirm localisation of the ventricular electrode (in PA, LAO 60° and RAO 30° projections), echocardiography and NT-proBNP plasma level measurements were performed. Percentage of ventricular pacing was analysed in each patient using Holter pacemaker function. Data from the last three examinations were analysed and the average value was calculated. During the whole follow-up period appropriate medical treatment was used in the patients, depending on their clinical status.

Echocardiographic evaluation

An initial examination was performed within the first month after implantation using Ultramark 4 (ATL company) ultrasound machine with 3.0 MHz transducer. The following parameters were measured using the M-mode technique: LV end-systolic diameter (LVESD), LV end-diastolic diameter (LVEDD), left atrium diameter (LA), interventricular septum (IVS) and posterior wall (PW) thickness, as well as PW/IVS index. In parasternal long axis view RV end-diastolic diameter (RVEDD) was assessed using 2-D technique. Left ventricular ejection fraction (LVEF) was measured according to Simpson's method in apical 4- and 2-chamber views. Early and late mitral peak inflow velocity and E wave deceleration time (DTE) were recorded in apical 4-chamber view using pulsed wave Doppler beam at the level of mitral valve leaflet tips. Isovolumic relaxation time (IVRT) was assessed in apical 5-chamber view by placing the sample volume between LV outflow and inflow tracts, which allows the moment of aortic valve closure and mitral valve opening to be gained simultaneously. During the examination, the values obtained from 3 measurements were averaged. The intensity of mitral and tricuspid valve regurgitation was assessed in apical 4-chamber view using a 4-degree scale. All measurements were obtained during ventricular pacing. In further analysis, the parameters obtained during echocardiographic examination performed within

the first month after implantation were accepted as initial.

Final echocardiographic examination was performed using a Sonos 2000 (Hewlett-Packard company) ultrasound machine with 2.5 MHz transducer. Besides parameters analysed in the initial examination, additional ones were assessed. In order to gain more precise LV diastolic function, the signal from the pulmonary veins was recorded in apical 4-chamber view. Systolic and diastolic velocity time integral (VTI-S and VTI-D) as well as duration (Adur-pulm) and peak velocity (A-pulm) of A wave at pulmonary vein ostia level were measured. The difference (Δ Adur) between A wave duration at the level of transmitral inflow (Adur) and pulmonary vein ostia (Adur-pulm) level was calculated. All measurements were performed according to the pacing program established during previous control visits if the rate of ventricular pacing based on pacemaker Holter monitoring was above 90%.

The QRS complex duration was measured from the peak of ventricular pacing impulse to the last wave of QRS complex in lead II of standard ECG recording at a speed of 50 mm/s. Duration of QRS complexes were measured from ECG performed during the first month after implantation and during the last control examination.

Levels of N-terminal pro-brain natriuretic peptide (NT-proBNP) were measured during the final control visit. Venous blood samples were taken after 30 min at rest in the supine position. NT-proBNP level was assessed with immunochemical method using Elecsys NT-proBNP device (Roche Diagnostics).

Statistical analysis

Continuous variables are expressed as a mean value ± standard deviation. Comparison between RVOT and RVA groups was performed using Student's *t*-test for continuous variables and χ^2 test for dichotomist variables. Two-tailed *t*-test was applied for comparison of parameters evaluated in the initial and final examination in each group of patients. Mann-Whitney test was used to assess the change of the degree of atrio-ventricular valve incompetence. A p value <0.05 was considered significant, but p <0.1 was also given due to the small number of patients in the study groups.

Results

Between January 1996 and June 1997, 120 patients after pacemaker implantation were enrolled in the study. During the final control visit the planned examination was performed in 27 patients: 17 of them were randomised before implantation to RVA pacing (RVA group) and 13 to RVOT pacing (RVOT group). Other patients died or were lost for follow-up.

Initial evaluation

Clinical characteristics of patients. No significant differences in age or gender were observed between the two groups (Table I). We found no significant differences between these two groups with respect to concomitant diseases except for heart failure (HF), which was diagnosed in the RVA pacing group in 3 (21%) patients; p <0.1. In the RVOT group, sinus rhythm was observed more frequently (12 *vs* 9 patients; p <0.1), whereas chronic atrial fibrillation (CAF) was observed in 5 (36%) patients in the RVA group; in three of them ablation of the a-v junction was performed. In the RVOT group CAF was observed only in one patient. As a consequence of different prevalence of CAF, the VVIR pacing system was more frequently implanted in patients in the RVA group (5 *vs* 1 patient; p <0.1).

Echocardiographic examination. In the initial examination (Table II), carried out within the first month after implantation, no significant differences were observed with respect to LA, LV and RVEDD diameters; however, IVS and PW thickness were significantly higher in the RVA group. In both groups global systolic LV function was normal and mean LVEF was similar in RVA and RVOT groups: 56%±11% and 54%±7%, respectively (NS). No differences in mitral and tricuspid valve regurgitation degree or LV diastolic function parameters between groups were noted (Tables V and VI).

Long-term effects of RV pacing

The final control examination was carried out after more than seven years of RV pacing (89±9 months in the RVA group and 93±6 months in the RVOT group, NS). No ventricular electrode dislocation was observed (in either early or late period after implantation). In both groups over 90% of permanent ventricular pacing was observed (Table III).

Patients' clinical status evaluation. Heart failure symptoms were observed in 7 (50%) patients from the RVA group at the final control visit (in comparison with 3 patients with HF in this group before implantation). In the RVOT group symptoms of HF were observed in 2 (15%) patients (before implantation no patient presented with HF in this group). Mean NYHA class in RVA and RVOT groups was 2.2±0.3 and 2.0±0.0 respectively. The number of patients with HF, before and after the procedure, was higher in the RVA group

Table I. Baseline characteristics of patients

	RVA Group (n=14)	RVOT Group (n=13)	p
Age (years)	76 ± 9	69 ± 16	NS
Men	7 (50%)	8 (62%)	NS
Cardiac rhythm			
Sinus	9 (64%)	12 (92%)	<0.1
Chronic atrial fibrillation	5 (36%)	1 (8%)	
Indications for pacemaker implantation			
AV block II°/III°	10 (72%)	13 (100%)	<0.05
including AV node ablation	3	1	
Chronic atrial fibrillation + bradycardia	2 (14%)	0	NS
SSS+RBBB+LAH	1 (7%)	0	NS
SSS	1 (7%)	0	NS
Concomitant diseases			
Hypertension	12 (86%)	9 (69%)	NS
Coronary artery disease	4 (29%)	3 (23%)	NS
Previous myocardial infarction	1 (7%)	3 (23%)	NS
Previous myocarditis	1 (7%)	2 (15%)	NS
Idiopathic dilated cardiomyopathy	1 (7%)	0	NS
Heart failure	3 (21%)	0	<0.1
Pacing mode			
VDD/DDD	9 (64%)	12 (92%)	<0.1
VVI/VVIR	5 (36%)	1 (8%)	

Table II. Parameters assessed in echocardiographic examination performed during the first month after implantation (initial examination)

	RVA Group (n=14)	RVOT Group (n=13)	p
LA (mm)	42±5	40±8	ns
IVS (mm)	12±3	10±2	<0.1
PW (mm)	11±2	10±2	<0.1
PW/IVS	0.94±0.16	0.98±0.04	NS
LVEDD (mm)	49±6	51±5	NS
LVESD (mm)	33±7	35±7	NS
RVEDD (mm)	33±6	30±9	NS
LVEF (%)	56±11	54±7	NS
Mitral valve regurgitation*			
0°	30%	33%	
1°	50%	66%	NS
2°	20%	0%	
Tricuspid valve regurgitation*			
0°	88%	60%	NS
1°	12%	40%	

*LA – left atrial diameter, IVS – interventricular septum thickness, PW – posterior wall thickness, LVEDD, LVESD – end-diastolic and end-systolic left ventricular diameter, RVEDD – right ventricular end-diastolic diameter, LVEF – left ventricular ejection fraction; * – estimated on four-degree scale*

Table III. Characteristics of patients during final control visit

	RVA Group (n=14)	RVOT Group (n=13)	p
Pacing duration (months)	89±9	93±6	NS
Percentage of ventricular pacing (%)	94±13	99±0.7	NS
Cardiac rhythm:			
Sinus rhythm	6 (43%)	12 (92%)	<0.01
Chronic atrial fibrillation (CAF)	8 (57%)	1 (8%)	<0.01
including new CAF	3	0	<0.1
Heart failure	7 (50%)	2 (15%)	<0.05
NYHA class	2.2±0.3	2±0.0	
Heart failure development	4	2	NS
NT-proBNP level (pg/ml)	1034±852	429±430	<0.05

Table IV. Differences of echocardiographic parameters in both groups obtained during initial and final echocardiographic examinations

		Initial examination	Final examination	p
LA (mm)	RVOT	40±8	43±7	<0.05
	RVA	42±5	44±7	NS
IVS (mm)	RVOT	10±2	13±4	<0.05
	RVA	12±3	14±4	NS
PW (mm)	RVOT	10±2	12±2	<0.001
	RVA	11±2	12±2	<0.1
PW/IVS	RVOT	0.98±0.04	0.93±0.21	NS
	RVA	0.94±0.16	0.90±0.17	NS
LVEDD (mm)	RVOT	51±5	50±6	NS
	RVA	49±6	49±8	NS
LVESD (mm)	RVOT	35±7	34±6	NS
	RVA	33±7	32±9	NS
LVEF (%)	RVOT	54±7	53±9	NS
	RVA	56±11	47±8	<0.05

Abbreviations – see Table II

(p <0.1). In 8 (57%) patients treated with RVA pacing CAF was observed, indicating the development of CAF in three new patients in this group during follow-up. For this reason the pacing program was changed to VVI in 2 pts. (before: DDD in one and VDD in another). No patients treated with RVOT pacing developed CAF. Mean NT-proBNP level assessed during the final control visit was significantly higher in the RVA group: 1034±852 pg/ml *vs* 429±430 pg/ml, p <0.05 (Table III).

Final echocardiographic examination. In the final echocardiographic examination, in comparison with the initial results, LA enlargement and increase of IVS thickness were observed in the RVOT group, whereas an increase of PW thickness was noted in both groups, although PW/IVS index did not change (Table IV). A significant decrease of LVEF was found (from 56±11% to 53±8%; p <0.05) in the RVA group, whereas LVEF did not change in patients treated with RVOT pacing (54±7% and 53±9%; NS). The degree of mitral regurgitation did not vary in either group, while in the RVA group increased incidence of tricuspid valve regurgitation and its progression were noted. In the RVA pacing group tricuspid valve regurgitation was not observed in 88% of patients and in 12% of them first degree tricuspid valve regurgitation was identified at the initial examination. In the final examination tricuspid valve regurgitation was observed in all patients in the RVA group: in 75% first degree and in 25% second degree (initial examination *vs* control examination; p <0.05). Analysis of LV diastolic function parameters (Table V) did not reveal differences with respect to mitral flow parameters including: E wave,

Table V. Echocardiographic parameters of LV diastolic function estimated in analysed groups during initial examination (RVOT-1, RVA-1) and during final control (RVOT-2, RVA-2)

	RVOT - 1	RVA - 1	p RVOT-1 *vs* RVA-1	RVOT-2	RVA-2	p RVOT-2 *vs* RVA-2
E (cm/s)	77±34	62±19	NS	75±24	83±26	NS
DTE (ms)	200±64	207±69	NS	245±77	205±61	NS
A (cm/s)	73±26	76±35	NS	80±11	89±13	NS
IVRT (ms)	103±19	97±14	NS	104±17	99±14	NS
VTI-S (cm)				17±6	13±4	<0.1
VTI-D (cm)				9±3	12±5	<0.1
Adur (ms)				159±17	156±19	NS
Adur-pulm (ms)				112±19	136±26	<0.1
ΔAdur (ms)				41±24	19±29	NS
R-R (ms)				853±142	853±103	NS

E – maximum velocity of early mitral flow, DTE – E wave deceleration time, A – maximal velocity of atrial mitral flow, IVRT – isovolumic relaxation time, VTI-S/ VTI-D – systolic and diastolic velocity time integral at pulmonary vein level, Adur – duration of A wave at mitral valve level, Adur-pulm – duration of A wave at pulmonary vein ostia level, ΔAdur – difference of duration of A wave at mitral valve level and pulmonary vein ostia level, R-R – duration of cardiac cycle

Table VI. Echocardiographic parameters of LV diastolic function estimated in analysed groups during initial and final examination

	RVA Group	RVOT Group	p
Initial examination			
Heart rate (/min)	73±4	71±3	NS
QRS (ms)	154±16	133±15	<0.05
Final examination:			
Heart rate (/min)	72±8	67±9	NS
QRS (ms)	178±19	177±21	NS

Duration of paced QRS complexes measured in ECG performed during first month after implantation and during final control visit

DTE, A wave, IVRT and DFP. However, the analysis of flow profile at pulmonary vein level performed during the last examination demonstrated significantly lower VTI-S value, higher VTI-D value and longer Adur-pulm time in patients treated with RVA pacing.

QRS complex duration. The duration of QRS complexes in ECG performed during the first month after implantation was significantly shorter in the RVOT group than the RVA group (133±15 ms *vs* 154±16 ms; p <0.05). During the final control visit there was no difference between the study groups in QRS complex duration (Table VI).

Discussion

In the present study we demonstrate the results of the first randomised prospective study in which the effects of pacing of different RV areas in patients with normal LV function treated for more than 7 years were analysed. The results indicate that RVOT pacing is associated with lower incidence of new CAF and had a protective influence on LV global systolic and diastolic function in comparison with RVA pacing. Significantly lower NT-proBNP levels reflect the haemodynamic benefits of RVOT pacing.

Previous clinical and experimental studies revealed that RVA pacing produces a number of unfavourable effects, due to the change of sequence and synchronic activation in the heart. It was also shown that RVA pacing leads to disturbances of perfusion, adrenergic innervation and segmental LV contractile function, which in turn leads to the deterioration of its systolic and diastolic function [1-3, 5, 6, 8]. Hence, there is a tendency to look for new, alternative RVA places of pacing. The area of the RVOT seems to be the most

interesting, because it is easily accessible during implantation, and the use of an active fixation electrode provides stable localisation and reduces the risk of dislocation [16, 19-23].

However, data from previous studies assessing the effects of RVOT stimulation are inconsistent. Furthermore, the majority of haemodynamic evaluations were performed as an "urgent" procedure, as in the study carried out by Giudici et al. [14]. They found a significant increase in cardiac output during RVOT pacing in the group of 89 patients. In contrast to these results, Buckingham et al. [13] and Blanc et al. [15] observed no differences between RVOT and RVA pacing.

Remarkably there are only a small number of studies in which long-term RVOT pacing effects were assessed; moreover, the follow-up duration was usually limited in these studies. Victor et al. [16] carried out a *cross-over* study in 16 patients with permanent supraventricular tachyarrhythmias and complete A-V block. Among them were 6 patients with LVEF <40% and with NYHA class II or III. The authors did not find significant differences between RVA and RVOT pacing after 3 months with respect to NYHA class, LVEF (assessed using radionuclide angiography), duration of exercise and maximum oxygen uptake (cardiopulmonary stress test). Similar results were found for patients with LVEF ≥40%, and in those with LVEF<40%.

Similar results come from the ROVA study [22], which was designed similarly to the previously mentioned study of Victor et al. [16]. It was a randomised *cross-over* study, which enrolled 103 patients with standard indications for VVIR pacing with HF (NYHA class II or III), LV systolic dysfunction (LVEF ≤40%) and persistent (≥7 days) AF. After randomisation, for the first three months in 37 patients RVA pacing was applied, whereas the other 43 received RVOT pacing. For the next three months the pacing mode was changed for the opposite one. No significant differences were observed between RVOT and RVA pacing with respect to quality of life, NYHA, 6-minute walking test and LVEF as well as degree of mitral regurgitation (estimated on echocardiography).

In contrast to the above-mentioned studies, Mera et al. [19] demonstrated that LV fractional shortening (measured by *M-mode* echocardiography) and LVEF (radionuclide ventriculography) were higher after 2 months of RVOT pacing in comparison with RVA pacing. The study group consisted of 10 patients with mild or moderate LV dysfunction, but mostly with HF symptoms, in whom A-V node ablation due to CAF was performed.

The present study is the only one so far demonstrating effects of long-term (lasting over 7 years) RVOT pacing in comparison with RVA pacing, in patients with initially normal LV function. We showed that RVA pacing led to significant deterioration of LV systolic function (decrease of LVEF) and progression of tricuspid valve regurgitation, and favoured LV diastolic dysfunction development.

These findings are consistent with the results described by Tse et al. [21], who in a perfectly designed and performed study documented that RVOT pacing produces a protective effect on LV perfusion and function in comparison with RVA pacing. The study included 24 patients with sinus rhythm, third degree A-V block and normal LVEF (≥50%). They were randomised to RVA (n=12), or RVOT (n=12) pacing using DDD mode with optimum AV delay. In all patients after 6 and 18 months of pacing cardiac scintigraphy and radionuclide ventriculography were performed. The examinations performed after six months did not reveal differences between groups, whereas after 18 months in patients treated with RVA pacing a significant increase of myocardium perfusion disturbances and a decrease of LVEF were observed. These changes were not observed during RVOT pacing, indicating its protective effect on LV function. The results of our study show that this influence persists during longer treatment, lasting over 7 years.

Tse et al. [21] showed that RVA pacing favours LV diastolic dysfunction and the decrease of maximum velocity of LV inflow was observed just after 6 months of treatment, which was not observed in the RVOT group. In our study the parameters of mitral inflow did not differ between groups in long-term follow-up, whereas the evaluation of inflow on the level of pulmonary vein ostia performed during the final control visit revealed lower VTI – S value and higher VTI – D value, and also longer duration of A wave in the RVA group. These findings indicate greater diastolic dysfunction in these patients.

In the present study it was also observed that RVOT pacing was associated with significant enlargement of LA, IVS and PW diameters, although the PW/IVS ratio did not change. The explanation of this phenomenon is difficult, especially because the two groups did not differ with respect to the prevalence of hypertension, and CAF was diagnosed significantly less frequently in patients with RVOT pacing. The interpretation is hampered also by the small number of patients in the analysed groups. Perhaps "high" localisation of the electrode tip on IVS leads to regional perfusion and contractility changes, resulting in specific remodelling after several years. In their study Tse et al. [21] showed that after 18 months of RVOT pacing the perfusion and contractility within the IVS area were significantly lower, but increased within the posterior wall of LV ($p < 0.05$).

Despite enlargement of LA diameter, the risk of CAF did not coexist in patients with RVOT pacing. On the contrary, it was the RVA pacing which was associated with higher incidence of CAF. In the RVOT group no new cases of CAF were observed, whereas in the RVA group it occurred in three patients. This may indicate that the protective influence of RVOT pacing in comparison with RVA is reflected by antiarrhythmic benefits with respect to CAF.

In papers published so far it has been shown that RVOT pacing can be associated with more synchronic ventricular activation, which is reflected by shorter QRS duration in comparison with RVA pacing [14, 17, 21, 22, 24]. In the present study mean duration of QRS complexes was significantly shorter during RVOT pacing in ECG recordings performed within the first month after the procedure. However, there were no differences found in the analysis of ECG recordings performed during the final control visit – after over 7 years of treatment the QRS complex duration was similar in RVOT and RVA groups. Unfortunately, there are no data on duration and QRS complex morphology before the implantation procedure, complicating the interpretation of these results. Data from the literature indicate that many factors influence synchronisation and activation during ventricular pacing. Of great importance is the fact that at the beginning the electrical impulse spreads as a result of slow muscular conduction and only when the activation wave reaches the ends of the Purkinje fibres it can quickly spread through the His--Purkinje system. The larger area is stimulated as a result of slow muscle conduction, the wider the QRS complexes occur. This phenomenon can be observed in each region of the heart, both in RV and LV, except for His bundle pacing [25]. Experimental studies demonstrated that during IVS pacing, in the area of "fast" conduction, the QRS complexes become shorter in comparison with RVA pacing, whereas our studies indicate that this effect does not remain during further follow-up. Thereby, the permanent RVOT pacing also leads to the change in synchronisation and sequence of ventricular activation; however, as is confirmed by numerous clinical and haemodynamic data, it is less harmful than in the case of RVA pacing.

The results of our study indicate that RVOT pacing results in a protective effect on systolic and diastolic LV function in patients treated with permanent RV pacing. However, both groups initially differed with respect to the type of rhythm (A-V block II°/III°, CAF) and HF presence, which could have influenced the results. The small number of patients in the analysed groups and above-mentioned differences are the limitations of the present study. Admittedly, in none of the patients was percutaneous transluminal coronary angioplasty performed; however, pharmacological treatment in the groups was not compared, which could have been of importance during seven years of treatment. It should be emphasised that initially none of the patients had LV dysfunction, and in these patients even more significant superiority of RVOT pacing over RVA could be expected. In previous studies in which the results of RVOT and RVA pacing were compared, the findings were contradictory [16, 19, 22]. However, the follow-up in these studies was about 2–3 months; thus, as Tse et al. [21] demonstrated, it was too short to reveal significant differences or benefits.

The present study demonstrates also the safety and effectiveness of implantation of passive fixation electrodes in the RVOT region. Barin et al. [23] were the first to demonstrate in a randomised clinical trial that active fixation electrodes can easily be implanted in this location. Following these findings, many investigators used in RVOT pacing screw-in electrodes [16, 19, 20-22, 24]. Schwaab et al. [26], in a large group of 120 patients with follow-up of mean of 14 months, demonstrated that passive fixation electrodes can be easily implanted in the IVS region with maintenance of correct pacing and control parameter without electrode dislocation. Our results confirm the safety and stability of RVOT pacing using similar electrodes during over 7 years of observation.

Conclusions

1. In patients with normal LV function permanent RVA pacing leads to LV systolic and diastolic function deterioration.
2. RVOT pacing can reduce the unfavourable effects of RVA pacing and slow down cardiac remodelling.
3. Clinical and echocardiographic benefits observed in the RVOT group after 7 years of pacing are reflected by lower NT-proBNP levels in this group of patients.
4. Further studies with selected groups of patients and adequately long follow-up are needed to establish which patients benefit most from RVOT pacing.

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Stymulacja wierzchołka lub drogi odpływu prawej komory w obserwacji długoterminowej: prospektywne badanie kliniczne i echokardiograficzne z randomizacją

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Streszczenie

Wstęp: U pacjentów leczonych stałą stymulacją serca typowym miejscem implantacji elektrody jest wierzchołek prawej komory (RVA). Badania sugerują jednak, iż takie jej położenie powoduje niekorzystny tor depolaryzacji komór, natomiast stymulacja okolicy drogi odpływu prawej komory (RVOT) jest bardziej fizjologiczna.

Cel: Celem niniejszego prospektywnego badania z randomizacją była długoterminowa ocena wpływu stymulacji RVOT i RVA na stan kliniczny, funkcję lewej komory (LV) oraz stopień niedomykalności zastawek przedsionkowo-komorowych u pacjentów leczonych stałą stymulacją serca.

Metodyka: W latach 1996–1997 pacjentów ze wskazaniami do stałej stymulacji serca randomizowano do stymulacji RVA lub RVOT. W 2004 r., w czasie ostatniej wizyty kontrolnej u 27 chorych oceniono stan kliniczny, parametry echokardiograficzne, czas trwania zespołów QRS oraz stężenie NT-proBNP. Badane parametry porównywano między grupami RVA i RVOT, a jeśli dostępne były dane w okresie okołozabiegowym, oceniano ich zmianę w czasie.

Wyniki: Spośród 27 przebadanych chorych, 14 było randomizowanych do stymulacji RVA, a 13 do stymulacji RVOT. Przed zabiegiem nie stwierdzano między grupami istotnych różnic odnośnie do wieku, płci, chorób współistniejących czy też parametrów echokardiograficznych. Średni czas stymulacji nie różnił się między grupami (89±9 mies. w grupie RVA *vs* 93±6 mies. w grupie RVOT; NS). Podczas ostatniej kontroli w grupie RVOT nie odnotowano żadnych nowych przypadków utrwalonego migotania przedsionków, natomiast w grupie RVA wystąpiło ono u 3 pacjentów (NS). W grupie RVA stwierdzono znamienny spadek frakcji wyrzutowej LV (z 56±11% do 47±8%; p <0,05), która nie uległa zmianie u chorych leczonych stymulacją RVOT (54±7% i 53±9%; NS). Ponadto w grupie RVA odnotowano progresję stopnia niedomykalności zastawki trójdzielnej, czego nie obserwowano w grupie RVOT. Średnie stężenie NT-proBNP podczas ostatniej kontroli było istotnie wyższe w grupie RVA: 1034±852 pg/ml *vs* 429±430 pg/ml (p <0,05).

Wnioski:

1. U pacjentów z prawidłową funkcją LV długotrwała stymulacja RVA prowadzi do pogorszenia funkcji skurczowej i rozkurczowej LV.
2. Stymulacja RVOT może zmniejszać niekorzystny wpływ i spowalniać proces przebudowy serca powodowany przez stałą stymulację RV.
3. Korzyści kliniczne i echokardiograficzne stwierdzane w grupie RVOT po ponad 7 latach stymulacji znajdują odzwierciedlenie w niższych stężeniach NT-proBNP w tej grupie pacjentów.
4. Konieczne są dalsze badania, przeprowadzone w wyselekcjonowanych grupach chorych oraz z odpowiednio długim okresem obserwacji dla ustalenia, którzy pacjenci odnoszą największe korzyści ze stymulacji RVOT.

Słowa kluczowe: stymulacja wierzchołka prawej komory, stymulacja drogi odpływu prawej komory

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