Journal of Arrhythmia 28 (2012) 214–218

Contents lists available at SciVerse ScienceDirect

Journal of Arrhythmia

journal homepage: www.elsevier.com/locate/joa

Original Article

Long-term effectiveness of right septal pacing vs. right apical pacing in patients with atrioventricular block

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ARTICLE INFO

Article history:
Received 17 November 2011
Received in revised form
17 January 2012
Accepted 19 January 2012
Available online 24 April 2012

Keywords:
Right septal pacing
Right apical pacing
Heart failure
Paced QRS interval

ABSTRACT

Background: Long-term right ventricular apical (RVA) pacing increases the risk of heart failure (HF) by inducing ventricular dyssynchronization. Although recent studies suggest that right ventricular septal (RVS) pacing results in improved short-term outcomes, its long-term effectiveness remains unclear.
Methods and results: This study investigated 149 consecutive patients who underwent implantation of a dual chamber pacemaker for atrioventricular block with either RVS-pacing between July 2007 and June 2010 or RVA-pacing between January 2003 and June 2007. The endpoint was defined as death and hospitalization due to heart failure (HF). The rates of mortality and hospitalization due to HF were significantly lower in the RVS-pacing group than that in the RVA-pacing group (event free RVS: 1 year, 98% and 2 years, 98%; RVA: 1 year, 85% and 2 years, 81%; $p < 0.05$). None of the patients died from HF in the RVS-pacing group, while 4 patients died from HF in the RVA-pacing group within 2 years after pacemaker implantation. The paced QRS interval was significantly shorter with RVS pacing than with RVA pacing at different times after pacemaker implantation (RVS: immediately 157.8 ± 24.0 ms, after 3 months 157.3 ± 17.5 ms, after 6 months 153.6 ± 21.7 ms, after 12 months 153.6 ± 19.4 ms, after 24 months 149.3 ± 24.0 ms vs. RVA: immediately 168.3 ± 23.7 ms, after 3 months 168.7 ± 26.0 ms, after 6 months 168.0 ± 22.8 ms, after 12 months 171.2 ± 22.3 ms, after 24 months 176.1 ± 25.5 ms; $p < 0.05$).
Conclusions: RVS pacing is feasible and safe with more favorable clinical benefits than RVA pacing.

1. Introduction

Right ventricular apical (RVA) pacing can result in ventricular dyssynchrony and decreased ejection fraction (EF), thereby leading to increased hospitalizations and mortality [1–4]. In contrast, pacing at the right septum and right ventricular outflow tract (RVOT) may result in more physiologic pacing [5]. While echocardiographic studies suggest that right ventricular septal (RVS) pacing can improve short-term outcomes and left ventricular systolic performance [6–8], there is no definitive evidence that RVS pacing is superior to RVA pacing in the long term. This may be because there is no consistency in lead placement among studies. Most studies are potentially flawed in that the leads were positioned in the RVOT and not necessarily in the septal position [6,7–11]. Furthermore, many short-term studies have reported a physiologic benefit of RVS pacing, and the negative remodeling effect of RVA pacing can take a year or more to manifest in patients with previously normal or near-normal ventricles. Some studies have investigated the long-term effect of RVS pacing, but the duration of follow-up was limited to 18 months, and the patient population included patients with atrial fibrillation or patients with ventricular pacing. Another reason for the reluctance to accept RVS pacing among physicians may be their concerns regarding long-term lead performance (e.g., R-wave sensing, stimulation threshold, and impedance) and complications. Thus, the goal of this study is to compare the long-term effectiveness of RVS pacing vs. RVA pacing in patients with atrioventricular block.

2. Methods

This observational study was performed at Showa University Hospital. Subjects included patients with second-degree or higher-degree atrioventricular block who exhibited clinical symptoms accompanied by bradycardia. All patients underwent

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http://dx.doi.org/10.1016/j.joa.2012.04.001

implantation of a dual-chamber pacemaker. Seventy-nine consecutive patients with RVS lead implantation were considered eligible for study participation from July 2007 to June 2010. Another 70 consecutive patients who underwent RVA lead implantation from January 2003 to June 2007 were used for comparison. All patients were followed up at an outpatient clinic in Showa University Hospital every 3–6 months after pacemaker implantation. The primary endpoint was death caused by heart failure (HF) and hospitalization for HF. The secondary endpoint was all-cause mortality and hospitalization for any cause. The following exclusion criteria were applied: (1) cumulative percent ventricular pacing (cumulative %VP) of less than 90% or recovery of atrioventricular conduction block, as assessed every 3–6 months in the clinic; (2) atrial fibrillation; (3) left ventricular ejection fraction (LVEF) $\leq 35\%$; (4) severe congestive heart failure (CHF), defined as New York Heart Association (NYHA) class III or IV, and (5) development of acute coronary syndrome within 3 months before.

2.1. Implantation procedure and determination of the pacing site

The right ventricular lead was placed via the subclavian vein. A screw in the lead was positioned onto the right ventricular septum using a hand-shaped stylet. A curve in the stylet was created using the distal 5–6 cm of wire. Then, the terminal 2 cm was bent to create a swan-neck deformity, similar to the design suggested by Vlay [12]. The superior margin of the RVS-pacing site can be considered as a line drawn from the superior tricuspid annulus at the level of the His bundle. This can be represented fluoroscopically as a multipolar catheter passing through the summit of the tricuspid valve by recording the His potential [13] (Fig. 1A). The screw-in lead was advanced into the pulmonary artery and withdrawn into the RV septum. The pacing site in the ventricular septum was determined by fluoroscopy. The posteroanterior (PA) view was used to guide the lead into the RVOT and mid RV (Fig. 1B). The 40° right anterior oblique (RAO) projection was used to prevent inadvertent positioning in the coronary sinus and great cardiac vein. Septal and free-wall sites were determined by a leftward orientation of the lead tip in the 40° left anterior oblique (LAO) view, as proposed by Mond [14] (Fig. 1C). The septal positioning was confirmed by 2 fluoroscopic views: PA and 40° LAO.

Once the lead tip made contact with the septal wall, the screw was deployed. The ventricular stimulation threshold at a 0.4 ms pulse width, R-wave amplitude, and lead impedance were measured several minutes after screw deployment.

By contrast, a tined lead was placed at the RV apex guided by fluoroscopy in the RVA-pacing group. We used a screw-in lead in only 7 patients in the RVA-pacing group, because a tined lead was unable to pace the ventricle stably.

2.2. Electrocardiogram and pacemaker follow-up

ECGs were recorded daily after pacemaker implantation until discharge and every 3–6 months at an outpatient clinic. The QRS interval, defined as the length of time from the beginning of the pacing spike until the end of the QRS complex, was automatically measured using FDX-6500 from recordings obtained from the filing system, FCP-2000A. The sampling rate of FDX-6500 was 1000 Hz, and the bandwidth was 0.05–100 Hz [15].

The measurement of the bipolar ventricular stimulation threshold at a 0.4 ms pulse width and the R-wave amplitude was performed immediately after pacemaker implantation and every 3–6 months at the outpatient clinic. The pacing output was programmed 2–3 times as the pacing threshold.

The cumulative %VP was determined from diagnostic data stored on the pacemaker at each follow-up visit. When the cumulative %VP of the patient was less than 90% or when there was a recovery of atrioventricular conduction, the patient was excluded from the study.

Lead dislodgement was defined as movement of the lead requiring another procedure for repositioning.

2.3. Evaluation of underlying heart disease and cardiac function

Cardiac function and underlying heart disease were assessed in all patients by echocardiography within 1 week prior to pacing. We also assessed cardiac function and mitral regurgitation by echocardiography immediately after implantation. A cardiac electrophysiological study was performed to determine the blocked site within the atrioventricular conduction system. Some diagnoses (e.g., dilated cardiomyopathy, myocarditis, and other cardiomyopathies) were made after cardiac catheterization and myocardial biopsy. HF was defined by clinical symptoms, greater than NYHA class II, or radiological evidence of pulmonary congestion.

2.4. Statistical analysis

Continuous data are expressed as the mean $\pm$ standard deviation. Data were compared using Student's *t*-test for paired or unpaired samples; categorical variables were analyzed with the χ^2 test. The Kaplan–Meier survival techniques were used to

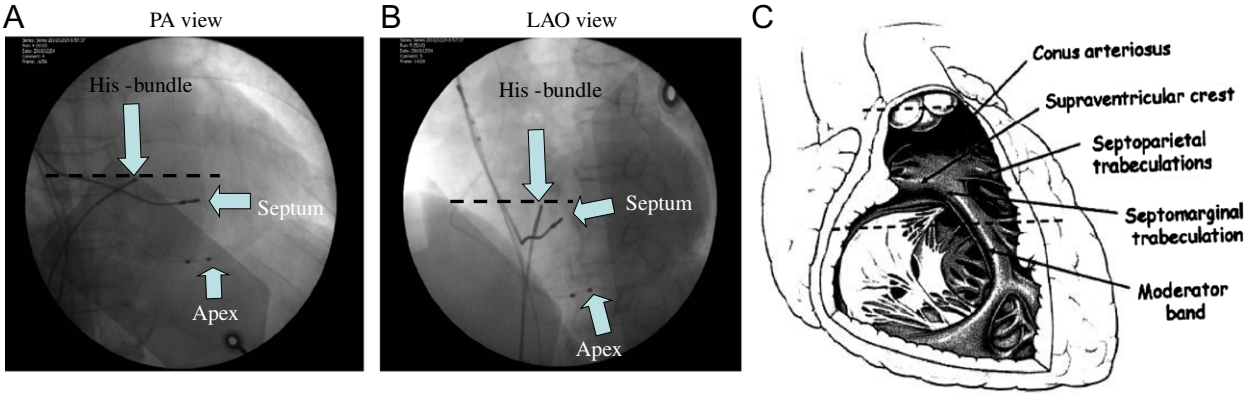

Fig. 1. Right ventricular pacing site. The dotted lines represent the boundary between the RV outflow tract (above) and the mid septum. This line represents a His-bundle catheter passed across the roof of the tricuspid valve. Radiographic views: posterior–anterior (PA) and 40° left anterior oblique (LAO). The superior lead is in the RV septum and the inferior lead is in the RV apex. The RV lead was placed deep in the RVA guided by fluoroscopy in the right anterior oblique (RAO) 30° projection. Most of the patients in the RVA group had tined leads. We only used screw-in leads in 7 patients in the RVA.

determine distributions. Survival was estimated from the date of pacemaker implantation to that of death or hospitalization; living patients were censored at the last date of follow-up. All probability values were deemed statistically significant at a level of 0.05 or below.

3. Results

3.1. Patient characteristics

Patient background data are summarized in Table 1. The mean follow-up period for patients undergoing RVS pacing was 23.5 ± 12.4 months. The average ages of the 79 patients with RVS pacing and the 70 patients with RVA pacing were 79.7 ± 8.4 years and 76.0 ± 13.0 years, respectively. The differences in age, gender, history of HF, underlying heart disease, EF, diagnosis of AVB on electrocardiogram, blockage site of the atrioventricular conduction system, QRS duration, morphology of the escaped beat, or medication at the time of pacemaker implantation between the 2 groups were not significant.

Table 1
Patient characteristics.

Pacing site	RVS pacing	RVA pacing	*p*
N	79	70	
Age (mean ± SD)	79.7 ± 8.4	76.0 ± 13.0	ns
Sex, *n* (%)			
Male	49(62.0)	48(68.6)	ns
Female	30(38.0)	22(31.4)	ns
Heart failure before implantation, *n* (%)	26(32.9%)	25(35.7)	ns
Underlying heart disease, *n* (%)			
Ischemic heart disease	8(10.1)	11(15.7)	ns
Cardiomyopathy	7(8.9)	0(0)	ns
Valvular heart disease	4(5.1)	5(7.1)	ns
Ejection fraction (%) (mean ± SD)	61 ± 8.6	58 ± 13.3	ns
Medications at implantation, *n* (%)			
β-Blocker	2(2.5)	4(5.7)	ns
Angiotensin-converting enzyme inhibitor	6(7.6)	8(11.4)	ns
Angiotensin II receptor blockers	30(37.9)	29(41.4)	ns
Furosemide	25(31.6)	28(40)	ns
Spironolactone	20(25.3)	25(35.7)	ns

RVS: right ventricular septal; RVA: right ventricular apical.

3.2. Mortality and hospitalization

Fig. 2 shows hospitalization and mortality in the RVS-pacing group and the RVA-pacing group within 2 years after pacemaker implantation. Fig. 2A indicates that the hospitalization from HF in the RVS-pacing was significantly lower than that in the RVA-pacing group (% event free RVS: 1 year, 99% and 2 years, 98% vs. RVA: 1 year, 85% and 2 years, 81%; $p < 0.05$). In addition, none of the patients died from HF in the RVS-pacing group, while 4 patients died from HF in the RVA-pacing group within 2 years after pacemaker implantation (Fig. 2B).

3.3. Pre- and postoperative QRS interval

The preoperative QRS interval was 122 ± 30 ms in the RVS-pacing group and 129 ± 29 ms in the RVA-pacing group, which was not significantly different. However, the paced QRS interval was significantly shorter with RVS-pacing than with RVA-pacing at different times after pacemaker implantation (RVS: immediately 157.8 ± 24.0 ms, after 3 months 157.3 ± 17.5 ms, after 6 months 153.6 ± 21.7 ms, after 12 months 153.6 ± 19.4 ms, after 24 months 149.3 ± 24.0 ms vs. RVA: immediately 168.3 ± 23.7 ms, after 3 months 168.7 ± 26.0 ms, after 6 months 168.0 ± 22.8 ms, after 12 months 171.2 ± 22.3 ms, after 24 months 176.1 ± 25.5 ms; $p < 0.05$) (Fig. 3) (Table 2).

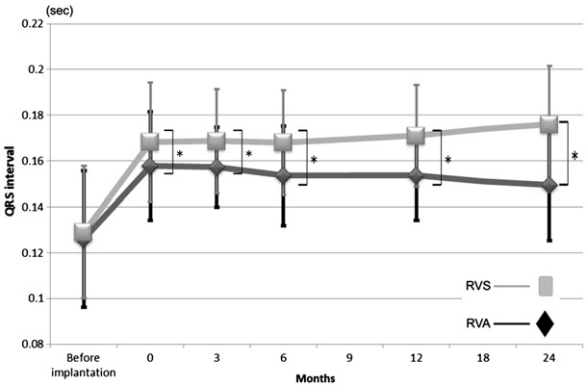

Fig. 3. Paced QRS width. The paced-QRS interval was shorter with right ventricular septal (RVS) pacing than with right ventricular apical (RVA) pacing at different times after pacemaker implantation.

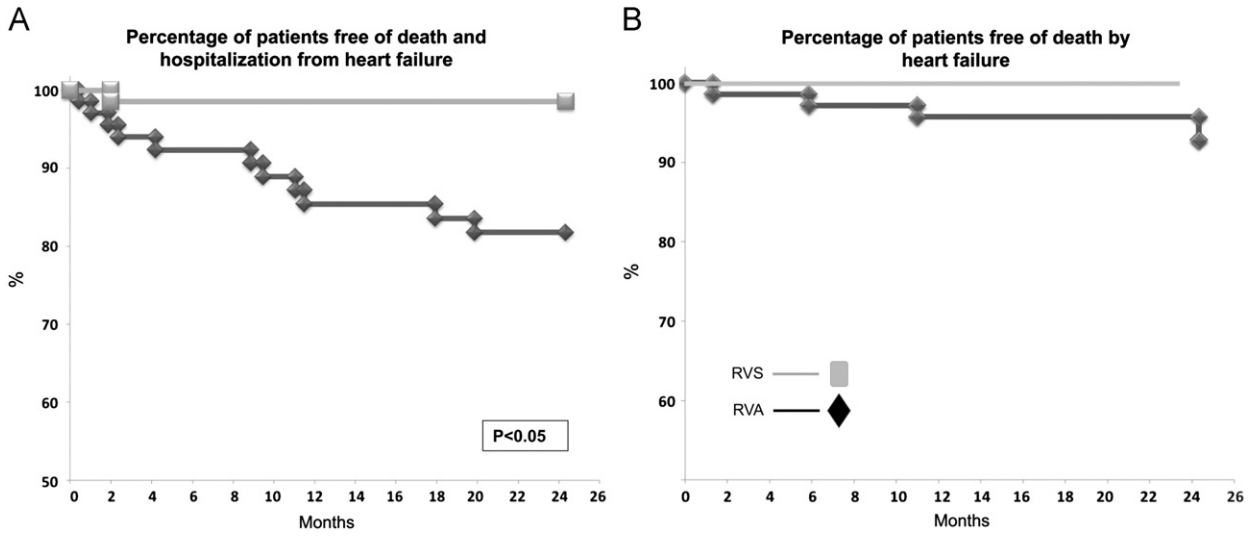

Fig. 2. Event-free survival curve. Only 2 patients developed congestive heart failure in the RVS-pacing group. The incidence of congestive heart failure was significantly lower in patients undergoing right ventricular septal (RVS) pacing than in those undergoing right ventricular apical RVA pacing ($p < 0.05$).

Table 2
Electrocardiogram characteristics.

Pacing site	RVS pacing	RVA pacing	*p*
N	79	70	
Diagnosis, *n* (%)			
II degree	7(8.9)	2(2.9)	ns
Advanced	29(36.7)	18(25.7)	ns
Complete	42(53.1)	49(70)	ns
Block site			
AH block	13(16.4)	12(17.1)	ns
HH′ block	7(8.9)	8(11.4)	ns
HV block	23(29.1)	23(32.9)	ns
Escaped beat duration before implantation (mean ± SD)	122 ± 30 ms	129 ± 29 ms	ns
Morphology of escaped beat			
CRBBB	22(27.8)	28(40)	ns
CLBBB	6(7.6)	10(14.3)	ns

Table 3
Characteristics of patients with and without heart failure.

	Developed CHF	Non-developed CHF	*p*
n	21	128	ns
Age (mean ± SD)	79.4 ± 9.5	77.7 ± 11.2	ns
Sex, *n* (%)			
Male	16(76.2)	81(63.2)	ns
Female	5(23.8)	47(36.8)	ns
Heart failure before implant disease	8(38.1)	43(33.5)	ns
Disease			
Ischemic heart	4(19.0)	15(11.7)	ns
Cardiomyopathy	0(0)	7(5.5)	ns
Valvular heart diseases	3(14.3)	6(4.68)	ns
Ejection fraction (%)	59 ± 11.6	59 ± 9.4	ns
QRS duration before implantation	130 ± 28 ms	124 ± 29 ms	ns
Pacing site			
RVA	19(90.4)	51(39.8)	$p < 0.05$
RVS	2(9.6)	77(60.2)	$p < 0.05$
Paced QRS duration (mean ± SD)	170 ± 20 ms	159 ± 23 ms	$p = 0.056$
Diagnosis, *n* (%)			
2 degree	0(0)	9(7.0)	ns
Advanced	3(14.3)	44(34.3)	ns
Complete	18(85.7)	73(58.7)	ns
Block site			
AH block	2(9.5)	23(18.0)	ns
HH block	1(4.8)	14(10.9)	ns
HV block	7(33.3)	39(30.4)	ns
Morphology of escaped beats			
CRBBB	9(42.9)	41(32.0)	ns
CLBBB	5(23.8)	11(8.6)	ns
Medications at implant, *n* (%)			
β-Blocker	2(9.5)	5(3.9)	ns
Angiotensin-converting enzyme inhibitors	4(19.0)	10(7.8)	ns
Angiotensin II receptor blockers	9(42.8)	50(39.0)	ns
Furosemide	6(28.6)	47(36.7)	ns
Spironolactone	6(28.6)	39(30.5)	ns

CHF: congestive heart failure: RVS: right ventricular septal; RVA: right ventricular apical.

3.4. Paced QRS interval with CHF

Within 2 years after pacemaker implantation, 21 patients developed CHF. Background variables of the patients with CHF are compared with the patients without CHF in Table 3. As mentioned before, the incidence of CHF was significantly lower in the RVS-pacing group than in the RVA-pacing. In addition, the paced-QRS interval immediately after pacemaker implantation tended to be longer in patients with CHF than in the patients without CHF (170 ± 20 ms vs. 159 ± 23 ms; $p = 0.056$). However, there was no significant difference in the other background variables between patients with CHF and patients without CHF.

3.5. Pacing threshold, R-wave sensing, and lead impedance

There were no significant differences in mean pacing threshold, R-wave sensing, or lead impedance at the different time points (6 months, 12 months, 18 months, and 24 months after implantation; Table 3). The number of lead dislodgements requiring surgical revision was 6 in the RVS-pacing group and 5 in the RVA-pacing group.

4. Discussion

The main finding in this study was that RVS-pacing is superior to RVA-pacing with regard to prevention of CHF in patients with atrioventricular block. The second finding was that patients who developed CHF after pacemaker implantation had longer QRS intervals than patients without CHF.

The present study demonstrated that the QRS interval measured at different times after pacemaker implantation was significantly shorter in the RVS-pacing group than in the RVA-pacing group, which probably indicates that RVS-pacing was associated with reduced electrical dyssynchrony. The clinical implications of these findings were reflected in the decreased rate of hospitalization for CHF and the decreased CHF-related mortality in the RVS-pacing group when compared with the RVA-pacing group. Furthermore, this study demonstrated that pacing parameters (e.g., R-wave sensing and impedance) were stable over time in the RVS pacing group and that both RVS- and RVA-pacing groups had similarly low rates of lead dislodgement. These data indicate that RVS pacing has long-term efficacy and safety.

Previous reports have suggested that prolongation of the QRS interval results in decreased LVEF and a higher risk of CHF [15,16]. Thus, there has been increasing interest in RV pacing sites that are associated with more favorable physiologic function. Some studies suggest that pacing from a septal stimulation site may produce such favorable physiologic atrioventricular conduction [6,7]. However, the absence of definitive data showing the superiority of RVS-pacing over RVA-pacing has limited the adoption of this strategy.

Durrer et al. [17] reported that ventricular depolarization begins in the LV septum, which suggests that initiating pacing from regions close to this area (e.g., RV septum) may produce a physiologic contraction pattern. In contrast, the free wall of the RV is the last zone to be depolarized. Thus, it is important to distinguish septal positioning from other RV sites. Although a number of studies describe RVS pacing [18,19], they do not provide specific details regarding the position of the leads or confirmation of septal site placement. In this study, we clearly demonstrated right ventricular septal pacing anatomically under fluoroscopy. The pacing of this site produces a narrower QRS than the pacing of the right ventricular apex. These findings suggest that right ventricular septal site may be more optimal than the right ventricular apex in patients who need continuous ventricular pacing. Moreover, the difference in QRS interval between the 2 groups became significant at different times after implantation in this study.

The negative remodeling effects of RVA-pacing may take years to manifest. Thus, results from acute studies of RVA-pacing cannot be generalized to outcomes in patients undergoing chronic

Table 4
Comparison of pacing threshold, R-wave amplitude, and pacing impedance.

	Implant	6 months	12 months	18 months	24 months
RVS-pacing					
R wave (mV)	11.8 ± 5.5	11.8 ± 4.4	12.4 ± 6.3	11.9 ± 4.8	12.4 ± 6.6
Threshold (volts)	0.88 ± 0.4	0.9 ± 0.4	0.82 ± 0.4	0.90 ± 0.4	0.92 ± 0.3
Impedance (Ω)	680 ± 220	610 ± 180	536 ± 242	544 ± 18	581 ± 334
RVA-pacing					
R wave (mV)	12.6 ± 6.6	15.2 ± 9.2	15.6 ± 10.4	16.7 ± 11.0	15.3 ± 9.1
Threshold (V)	0.50 ± 0.3	0.68 ± 0.4	0.68 ± 0.3	0.64 ± 0.3	0.62 ± 0.3
Impedance (Ω)	974 ± 406	778 ± 263	760 ± 232	789 ± 363	800 ± 397

pacing. Xue-Hua Z [20] reported that RVA pacing with >90% ventricular pacing was associated with HF in 26% of patients over a median follow-up period of 7.8 years. They also reported that an elderly age at the time of implantation predicted new onset of HF. Our study found that 27.2% of RVA-paced patients had HF only 2 years after implantation. The 2 studies differed in the age of patients at the time of implantation. The mean age was 76.0 ± 13 years in our study, while it was 68.2 ± 14.9 years in that of Xue-Hua et al. Therefore, an elderly age may predict HF after implantation.

The use of RVS pacing has been limited by concerns regarding procedural complication and long-term electrical performance, such as R-wave sensing and stimulation threshold. Most studies of RVS pacing have had a relatively small sample size, limited follow-up duration, and include relatively few data concerning the electrical performance and complications associated with these septal pacing sites [21]. Lead dislodgment was the most common adverse event in previous studies of RVS pacing, even when RV apex pacing was employed. In order to further explore these issues, prospective long-term comparative studies should be conducted that include strict definitions of RVS pacing and that exclude patients with atrial fibrillation. The present study is the first study to meet these criteria.

4.1. Study limitations

There are several limitations of this study. Firstly, it was a retrospective analysis and was subjected to selection bias. Secondly, this study was performed in a small number of patients within the limited facilities of our hospital. Thirdly, since implantation for apical pacing was performed during the period from January 2003 to June 2007 and implantation for septal pacing was performed during the period from July 2007 to June 2010, systematic cohort bias may have occurred. Therefore, the results of this study should be interpreted with some caution. Finally, longer-term randomized multicenter controlled studies with a larger number of patients are needed (Table 4).

5. Conclusions

RVS pacing was feasible and safe and produced more favorable clinical outcomes than RVA pacing.

Conflict of interest statement

The authors declare no conflicts of interest.

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