

# Right Ventricular Outflow Tract Septal Pacing Is Superior to Right Ventricular Apical Pacing

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**Background**—The effects of right ventricular apical pacing (RVAP) and right ventricular outflow tract (RVOT) septal pacing on atrial and ventricular electrophysiology have not been thoroughly compared.

**Methods and Results**—To identify a more favorable pacing strategy with fewer adverse effects, 80 patients who had complete atrioventricular block with normal cardiac function and who were treated with either RVAP (n=42) or RVOT septal pacing (n=38) were recruited after an average of 2 years of follow-up. The data from electrocardiography and echocardiography performed before pacemaker implantation and at the end of follow-up were collected. The patients in the RVOT septal pacing and RVAP groups showed similar demographic and clinical characteristics before pacing treatments. After a mean follow-up of 2 years, the final maximum P-wave duration; P-wave dispersion; Q-, R-, and S-wave complex duration; left atrial volume index; left ventricular end-systolic diameter; ratio of transmitral early diastolic filling velocity to mitral annular early diastolic velocity; and interventricular mechanical delay in the RVOT septal pacing group were significantly less than those in the RVAP group ( $P<0.05$ ). The final left ventricular ejection fraction of the RVOT septal pacing group was significantly higher than that of the RVAP group ( $P<0.05$ ).

**Conclusions**—Compared with RVAP, RVOT septal pacing has fewer adverse effects regarding atrial electrical activity and structure in patients with normal cardiac function. (*J Am Heart Assoc.* 2015;4:e001777 doi: 10.1161/JAHA.115.001777)

**Key Words:** cardiovascular diseases • electrophysiology • pacemaker

Artificial cardiac pacing refers to a type of treatment that uses electrical impulses to maintain a suitable heart rate and rhythm, primarily in patients with bradyarrhythmias arising from disease in the heart's electrical conduction system, such as the sinoatrial node, atrioventricular node, or His-Purkinje system.<sup>1</sup> Right ventricular apical pacing (RVAP) was established in the mid-1950s and was widely adopted in bradyarrhythmia therapy around the world.<sup>2</sup>

For decades, a growing number of short- and long-term studies have suggested that RVAP may induce or worsen dyssynchronous left ventricle (LV) contraction, LV longitudinal shortening and twist, atrial fibrillation, and heart failure.<sup>3,4</sup> These pathophysiological consequences are now attributed to

the violation of several electrophysiological properties of the myocardium.<sup>5</sup> Electrical conduction in the myocardium is at least 4 times slower than that in the Purkinje system; conduction along muscle fibers is 2 times faster than that with perpendicular activation.<sup>6</sup> In addition, the conduction characteristics between the endocardial and epicardial layers are different.<sup>7,8</sup> In the setting of RVAP, the activation front becomes ellipsoidal, and slowed conduction results, particularly in the intermediate and epicardial layers.<sup>1,5</sup> For these reasons, alternative pacing sites with a more synchronous ventricular activation pattern have been explored.<sup>1,5</sup>

The region and pacing alternatives to RVAP are numerous, including His-bundle pacing and para-His pacing, medial septum, lower septum, right ventricular outflow tract (RVOT) in the septal region, and pulmonary infundibulum.<sup>9,10</sup> Although these strategies are not well defined and standardized because of the ease of catheter implantation and catheter stability in this region, the most preferred area of pacing is the RVOT in the septal region.<sup>11–13</sup> The ideal pacing mode for improving cardiac function requires the participation of Purkinje fibers, an effect that is difficult to establish.<sup>1,5</sup> The RVOT septal region nearest the Purkinje system has been selected as favoring the pacing site<sup>1,5</sup>; however, clinical evidence to support RVOT septal pacing is still rare and confusing.<sup>14–16</sup> Favorable studies have shown that RVOT septal pacing produces interventricular

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synchronous contraction denoted by narrow Q-, R-, and S-wave complexes, preventing the deterioration of LV structure and function.<sup>14,15</sup> In contrast, another study suggested that although RVOT pacing caused more synchronous LV contraction than RVAP, it had no benefit over RVAP regarding the prevention of cardiac remodeling and preserving LV systolic function.<sup>16</sup> These conflicting studies appear to be associated with study designs that were not homogeneous regarding the methodology; judgment criteria; follow-up; and, particularly, statistical power used.<sup>5</sup> In addition, limited studies have focused on the effect of pacing sites on structure and function of the left atrium, findings that may be important for the development of atrial arrhythmias.<sup>15,17</sup> P-wave dispersion (PWD) is a novel prediction index of atrial arrhythmia.<sup>18</sup> Short-term ventricular demand pacing induces prolongation of maximum P-wave duration with increased PWD<sup>19</sup>; however, whether right ventricular pacing sites affect PWD is unclear.

In the present study, to assess whether RVOT septal pacing could maintain more favorable atrial and ventricular electrical activity to reduce the incidence of atrial fibrillation compared with RVAP, cardiac electrophysiology and cardiac structure and function of RVOT septal pacing and RVAP were assessed with 2-year follow-up in 80 Chinese patients who had complete atrioventricular block with normal cardiac function.

## Methods

### Patients

From January 2008 to December 2012, 80 patients who had complete atrioventricular block with normal cardiac function and who received either RVAP or RVOT septal pacing at our cardiology department were enrolled in the study retrospectively. The following exclusion criteria were used: history of atrial fibrillation and/or heart failure; PWD >40 ms before implantation of the pacemaker; severe tricuspid valve disease or tricuspid valve surgery history; and documented coronary artery disease, pulmonary disease, and renal insufficiency. All of the patients underwent electrocardiography, echocardiography, and clinical assessment. The mean follow-up duration was  $2.02 \pm 0.4$  years.

### Ethical Issues

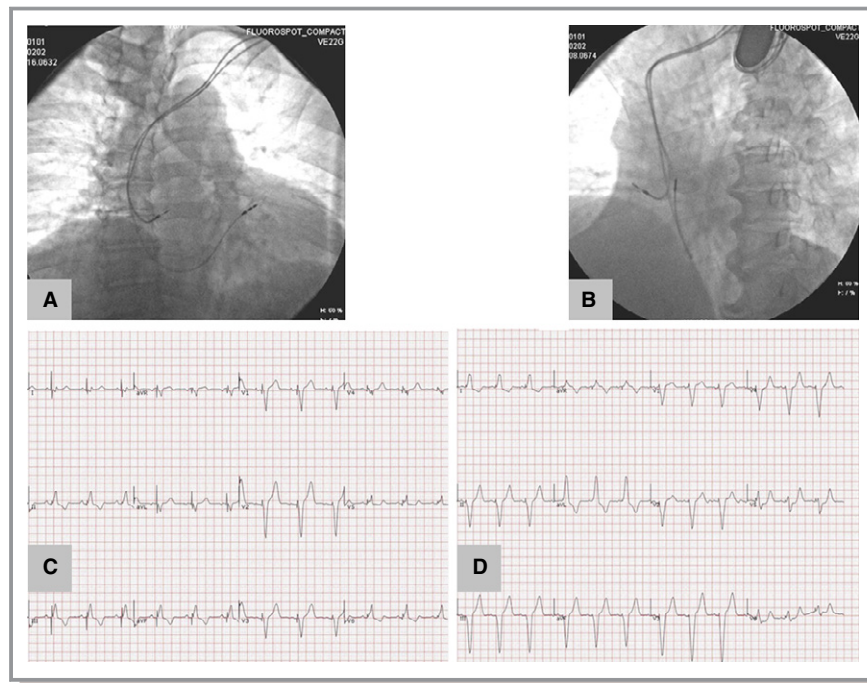
Written informed consent regarding the procedures and medical data to be used was obtained from all patients, according to the guidelines of the Chinese National Ethics Regulation Committee. The review board of the First Affiliated Hospital of Soochow University approved this protocol in accordance with the amended Declaration of Helsinki (reference number: FAHSU 2013–023).

## Pacemaker and Lead Implantation

All implantations were performed in a sterile manner with the patient in a conscious state under local anesthesia and by an experienced operator. In the RVAP group, the passive fixation electrodes were positioned toward the right ventricular apex (Figure). In the RVOT septal pacing group, the active helix electrodes were positioned against the septum of the RVOT (Figure). The RVOT septum was confirmed according to the tip of the electrode directed to the spine  $45^\circ$  to the left anterior oblique view using fluoroscopic radiographs and producing a negative or isoelectric vector in lead I and positive Q, R, and S waves in leads II, III, and aVF in the pacemaker electrocardiogram (ECG) (Figure). In both groups, the passive fixation electrodes were used as a right atrial appendage. After pacemaker implantation, all patients were followed regularly as outpatients. Pacing parameters, including pacing threshold, sensitivity, electrode impedance, and percentage of ventricular pacing, were assessed.

## Twelve-Lead Electrocardiography

A 12-lead surface ECG was obtained after a 20-minute resting period in the supine position at a paper speed of 50 mm/s and 2 mV/cm before pacemaker implantation, immediately after implantation, and at the end of the follow-up. The P-wave duration was measured manually in all of the simultaneously recorded 12 leads of the surface ECG. For greater accuracy, measurements were performed with calipers and a magnifying lens. The onset of the P wave was defined as the point of the first visible upward departure from the baseline for positive waveforms and as the point of the first downward departure from the baseline for negative waveforms. The return to the baseline was considered the end of the P wave. Three continuous sinus P-wave durations in the same lead were measured, and their average value was defined as the same lead P-wave duration. The maximum P-wave duration ( $P_{\max}$ ) measured in any of the 12 leads of the surface ECG was used as the longest atrial conduction time. The difference between the  $P_{\max}$  and minimum P-wave duration was calculated and defined as PWD.<sup>19</sup> The Q-, R-, and S-wave complex duration (QRSd) was calculated using the first to last sharp vector crossing the isoelectric line in all of the leads. Three continuous QRSd values were detected in the lead with the widest QRS complex in the 12 leads, and their average value was defined as QRSd. Twelve-lead electrocardiography was performed and analyzed by the same experienced technician. The final electrocardiographic result for each patient was reviewed by the chief technician. Any uncertainties regarding electrocardiographic results were resolved



**Figure.** Exemplificative diagram for RVOT septal and RVAP pacing. Chest x-ray showing the position of the right ventricular lead at the right ventricular outflow tract septum in right anterior oblique (30°) (A) and left anterior oblique (45°) (B) views. Twelve-lead electrocardiograms indicating the site of RVOT septal pacing (C) and RVAP (D). RVAP indicates right ventricular apical pacing; RVOT, right ventricular outflow tract.

by discussion. Both technicians were blinded to clinical data and group division.

### Echocardiography

A7 transthoracic echocardiographic examination was performed separately at baseline and at the end of follow-up using a Sonos 5500 ultrasound machine (Philips) with a 2.5-Hz transducer. All echocardiographic examinations for each patient were performed and analyzed by the same experienced echocardiographer who was blinded to clinical data and group division. Any uncertainties regarding echocardiographic examination results were resolved by discussion among senior technicians of the department of echocardiography. The measured parameters with the M-mode technique included interventricular septum thickness (normal range: 6 to 10 mm), LV posterior wall thickness (normal range: 6 to 10 mm), LV end-diastolic diameter (normal range: 35 to 56 mm), LV end-systolic diameter (normal range: 20 to 40 mm), transmitral early diastolic filling velocity, and mitral annular early diastolic velocity. The measurement of the LV ejection fraction (LVEF; normal value: >0.50) was performed using the Simpson's biplane method. The ratio of transmitral early diastolic filling velocity to mitral annular early diastolic velocity, designated as the E/Ea ratio (normal value: <8),

represents LV filling pressure and indirect left atrial (LA) pressure. The LA volume was measured at end-ventricular systole from the apical 2- and 4-chamber views immediately before mitral valve opening. The LA volume was indexed to the body surface area to provide the LA volume index (normal value: <24 mL/m<sup>2</sup>).<sup>20</sup>

The intervals between the onset of QRS to the beginning of ejection at the aortic and pulmonary valve levels using pulse-wave Doppler were defined as the aortic and pulmonary pre-ejection intervals, respectively. The interventricular mechanical delay (IVMD) was defined as the difference between aortic and pulmonary pre-ejection intervals.<sup>21</sup> An IVMD value  $\geq 40$  ms was considered an indicator of interventricular desynchrony.

### Statistical Analysis

Continuous data were expressed as mean  $\pm$  SD. Categorical data were summarized as frequencies and percentages, which were analyzed using the chi-square test. Paired Student *t* test analysis was used for comparison within groups, and nonpaired Student *t* test analysis was used for comparison between groups. All tests were 2-sided. A *P* value <0.05 was deemed statistically significant. Statistical analyses were performed using SPSS version 17.0 (SPSS Inc.).

## Results

### Clinical Characteristics of the RVAP and RVOT Septal Pacing Groups

Eighty patients (42 RVAP patients and 38 RVOT septal pacing patients) were included retrospectively in this comparative analysis. As shown in Table 1, there was no statistical difference between the 2 groups regarding age, sex, and rates of patients with hypertension, diabetes mellitus, and/or coronary artery disease. In the RVAP and RVOT septal pacing groups, respectively, 19.1% and 18.4% of patients took  $\beta$ -blocker medication, and 38.1% and 34.1% of patients took angiotensin-converting enzyme inhibitors or angiotensin II receptor blockers. No significant difference was observed regarding this medical history (Table 1).

Most patients had no or trivial mitral regurgitation and/or tricuspid regurgitation. No statistical difference was observed between the 2 groups regarding the rates of patients with moderate and severe mitral regurgitation and/or tricuspid regurgitation (Table 1). The leads were successfully implanted in all patients. No serious complications related to the surgery were detected during implantation or follow-up. Detailed pacing parameters, including the atrial and ventricular pacing threshold, sensitivity, and electrode impedance were recorded, and no intergroup or intragroup difference was significant.

In the RVAP group, 31 patients (73.8%) were implanted with the dual-chamber pacemaker, and 11 patients (26.2%) were implanted with ventricular demand pacing; among these patients, 26 (61.9%) underwent sinus rhythm mapping, and others had atrial pacing (20.5 $\pm$ 4.32%). In the RVOT septal pacing group, 29 patients (76.3%) were implanted with a dual-chamber pacemaker, and 9 patients (23.7%) were implanted with a ventricular demand pacemaker; among these patients, 24 (63.2%) underwent sinus rhythm mapping, and others had atrial pacing (18.9 $\pm$ 5.8%). There were no significant differences in atrial pacing and sinus rhythm mapping between the 2 groups (Table 1). The mean follow-up duration was 2.02 $\pm$ 0.4 years. There was no significant difference in follow-up duration between the RVAP group and the ROVT septal pacing group. During the 2 years of follow-up after implantation, no patients were lost, and 6 patients had new onset of atrial fibrillation in the RVAP group; however, only 1 patient with new-onset atrial fibrillation was observed in the RVOT septal pacing group.

### Cardiac Electrophysiology Evaluation

To compare the difference in the 2 pacing strategies in cardiac electrophysiology, the P-wave and QRS complex

**Table 1.** Summary of Patient Characteristics

Indexes	RVAP Group (n=42)	RVOT Septal Pacing Group (n=38)	P Value
<b>Demographic data and medical history</b>			
Age, y $\pm$ SD	61 $\pm$ 8.5	63 $\pm$ 9.7	0.886
Sex, male/female	23/19	20/18	1.000
Hypertension, n (%)	16 (38.1)	14 (36.8)	0.920
Diabetes mellitus, n (%)	4 (9.5)	5 (13.2)	0.729
Coronary artery disease, n (%)	1 (2.4)	2 (5.3)	0.602
<b>Mitral regurgitation</b>			
No or trivial	28 (66.7)	23 (60.5)	0.740
Mild	13 (31.0)	14 (36.8)	0.752
Moderate	1 (2.4)	1 (2.6)	1.000
Severe	0 (0.0)	0 (0.0)	—
<b>Tricuspid regurgitation</b>			
No or trivial	34 (80.9)	29 (76.3)	0.823
Mild	5 (11.9)	6 (15.8)	0.862
Moderate	3 (7.1)	3 (7.9)	1.000
Severe	0 (0.0)	0 (0.0)	—
<b>Medication use</b>			
$\beta$ -blockers, n (%)	8 (19.1)	7 (18.4)	0.823
ACEI/ARB, n (%)	16 (38.1)	13 (34.1)	0.823
<b>Pacemaker type</b>			
DDD, n (%)	31 (73.8)	29 (76.3)	1.000
VVI, n (%)	11 (26.2)	9 (23.7)	1.000
Sinus rhythm, n (%)	26 (61.9)	24 (63.2)	0.920
Atrial pacing, n (%)	16 (38.1)	14 (36.8)	0.920
Atrial pacing, mean $\pm$ SD	20.5 $\pm$ 4.3	18.9 $\pm$ 5.8	0.719

Continuous variables are presented as mean $\pm$ SD, and categorical data are presented as number (percentage). Differences between the 2 groups were examined using the Student *t* test and chi-square or Fisher exact probability tests according to the characteristics of the data distribution. ACEI/ARB indicates angiotensin-converting enzyme inhibitor/angiotensin receptor blocker; DDD, dual-chamber pacemaker; RVAP, right ventricular apical pacing; RVOT, right ventricular outflow tract; VVI, ventricular demand pacing.

values were checked by ECG. As shown in Table 2, initial  $P_{max}$ , PWD, and QRSd were similar between the RVAP and RVOT septal pacing groups. After a mean follow-up of 2 years, the final PWD and QRSd increased significantly in the 2 groups compared with each baseline level (Table 2). In the RVOT septal pacing group, however,  $P_{max}$ , PWD, and QRSd were significantly shorter than those in the RVAP group ( $P_{max}$ : 107 $\pm$ 8 ms versus 135 $\pm$ 5 ms,  $P=0.043$ ; PWD: 35 $\pm$ 8 ms versus 46 $\pm$ 10 ms,  $P=0.040$ ; QRSd: 130 $\pm$ 12 ms versus 154 $\pm$ 13 ms,  $P=0.048$ ) (Table 2). These data suggest that RVOT septal pacing displays lower  $P_{max}$ , PWD, and QRSd compared with RVAP.



**Table 2.** Electrocardiogram Changes in Both Groups During Follow-up

Electrocardiographic Parameters	RVAP Group (n=42)			RVOT Septal Pacing Group (n=38)			P Value
	Initial	Final	P Value	Initial	Final	P Value	
P <sub>max</sub> , ms	102±11	135±5	0.029	101±10	107±8	0.664	0.043
P <sub>min</sub> , ms	75±9	79±10	0.680	75±9	77±10	0.843	0.843
PWD, ms	27±7	46±10	<0.001	26±9	35±8	0.029	0.040
QRSd, ms	102±11	154±13	0.002	101±10	130±12	0.043	0.048

Data are presented as mean±SD. P<sub>max</sub> indicates maximum P-wave duration; P<sub>min</sub>, minimum P-wave duration; PWD, P-wave dispersion; QRSd, QRS complex duration; RVAP, right ventricular apical pacing; RVOT, right ventricular outflow tract.

### Cardiac Structure and Function Assessment

To monitor cardiac structure and function, echocardiography was performed for all patients before and after pacing. As shown in Table 3, of the patients treated with RVAP, LA volume index, LV end-diastolic diameter, LV end-systolic diameter, E/Ea ratio, and IVMD increased significantly compared with initial levels. Compared with the baseline value, the final LVEF decreased in the RVAP group (from 0.66±0.12 to 0.51±0.10;  $P=0.048$ ). Of the patients treated with RVOT septal pacing, LA volume index, LV end-systolic diameter, and IVMD also increased significantly compared with initial levels (Table 3). Of both groups, interventricular septum thickness displayed no significant change after pacing, and RVOT septal pacing did not alter the LV end-diastolic diameter level (Table 3). Although both RVAP and RVOT septal pacing increased the LAVI and IVMD levels, the increasing rates of the RVOT septal pacing group were significantly lower than those of the RVAP group (Table 3). The final LVEF of the RVOT septal pacing group was 0.62±0.14, a value that was significantly higher than that of the RVAP group (0.51±0.10).

### Discussion

In the present study, we confirmed that RVOT septal pacing induced shorter QRSd and less interventricular mechanical delay than RVAP, features that were associated with fewer adverse effects on LVEF and E/Ea ratio. In addition, our study demonstrated that RVOT septal pacing protected the atrial structure and electroactivity with less increase in LA volume index, P<sub>max</sub>, and PWD than in those with RVAP. These results indicate that RVOT septal pacing maintains a more satisfactory atrial and ventricular electroactivity sequence than RVAP.

Regarding the effect of RVOT pacing on ventricular electroactivity, Leong et al<sup>15</sup> demonstrated that the pacing site directly affects ventricular synchrony. The electrical impulse of RVAP occurs from the right ventricular apex and then propagates through the myocardium instead of the His-Purkinje conduction system, resulting in a wide QRS complex similar to the left bundle branch block.<sup>22,23</sup> The RVOT septal pacing area, however, is close to a physiological conduction system; it would promote depolarization more physiologically through rapid conduction and produces a narrow QRS width on ECG.<sup>24,25</sup> Restoring a narrower QRS duration may, in part, explain why RVOT septal

**Table 3.** Echocardiographic Parameters in the 2 Groups During the Follow-up

Echocardiographic Parameters	RVAP Group (n=42)			RVOT Septal Pacing Group (n=38)			P Value
	Initial	Final	P Value	Initial	Final	P Value	
LAVI, mL/m <sup>2</sup>	24.2±6.3	37.4±11.4	0.001	23.6±7.8	29.8±9.7	0.048	0.047
IVST, mm	10.1±1.6	10.7±1.8	0.648	9.6±2.1	10.4±1.7	0.547	0.826
LVEDd, mm	48.3±6.9	53.8±11.2	0.049	50.3±5.1	52.8±7.2	0.715	0.885
LVESd, mm	29.6±8.1	40.7±10.2	0.014	30.1±6.3	35.2±6.1	0.047	0.049
LVEF	0.66±0.12	0.51±0.10	0.048	0.65±0.09	0.62±0.14	0.722	0.047
E/Ea ratio	7.1±1.4	11.3±2.8	<0.001	6.7±1.2	8.3±3.2	0.111	0.021
IVMD, ms	16.6±5.0	45.2±18.7	<0.001	18.3±4.3	30.4±16.8	<0.001	0.004

Data are presented as mean±SD. E/Ea ratio indicates ratio of transmitral early diastolic filling velocity to mitral annular early diastolic velocity; IVMD, difference between the aortic and pulmonary pre-ejection intervals; IVST, interventricular septum thickness; LAVI, left atrial volume index; LVEDd, left ventricular end-diastolic diameter; LVEF, left ventricular ejection fraction; LVESd, left ventricular end-systolic diameter; RVAP, right ventricular apical pacing; RVOT, right ventricular outflow tract.

pacing is associated with protective effects on interventricular synchrony compared with RVAP. In our study, RVOT septal pacing produced a shorter QRSd than RVAP, whereas the final QRSd of the RVOT septal pacing group was also increased compared with the initial values, and the median average value was >120 ms. This result may be associated with difficulty in obtaining consistent, accurate, and reliable placement of leads in the same position and near the His-Purkinje conduction system. Consequently, the final IVMD in the RVOT septal pacing group was increased compared with the baseline value but was less than that of the RVAP group at the end of follow-up.

Zhang et al compared RVOT pacing with RVAP in elderly patients with normal LVEF.<sup>26</sup> Their results showed deterioration in the LVEF in patients with RVAP but not in those with RVOT pacing, suggesting that RVOT pacing is better than RVAP in terms of preventing cardiac dysfunction. In contrast, in a study by Gong et al,<sup>16</sup> although RVOT pacing caused more synchronous LV contraction than RVAP, no benefit over RVAP was shown regarding the prevention of cardiac remodeling and preservation of LV systolic function after 12 months of pacing in patients with normal cardiac function. In their study, RVOT pacing also produced a wide QRS complex with a median QRSd of 161 ms, a value that was different from that in other studies concerning QRSd produced by RVOT septal pacing. Thus, the precise pacing site might be different from others. In addition, such prolonged QRS duration induced by RVOT septal pacing may increase heart failure risk similar to long-term RVAP.<sup>27</sup> In our study, RVOT septal pacing produced shorter QRSd (median value was 130 ms) and had fewer adverse effects on IVMD, LVEF, and E/Ea ratio than RVAP. Consequently, compared with RVAP, RVOT septal pacing is the better choice to maintain ventricular synchronism.<sup>28</sup>

PWD reflects the heterogeneity of atrial electroactivity.<sup>18</sup> Clinical studies have shown that PWD is a new predictive index of atrial arrhythmia, particularly atrial fibrillation.<sup>29</sup> In the present study, the final PWDs in the RVAP and RVOT septal pacing groups were significantly increased compared with their initial values, but these changes were fewer in the RVOT septal pacing group. In addition, the incidence of new onset of atrial fibrillation was 14.3% (6 of 42) in the RVAP group but only 2.6% (1 of 38) in the RVOT septal pacing group at 2-year follow-up. In the present study, there were no significant differences in the atrial pacing and ventricular pacing percentages between the 2 groups. Consequently, we considered that RVOT septal pacing might have fewer adverse effects on atrial electroactivity compared with RVAP.

P-wave abnormalities include  $P_{\max}$  and PWD, which are detected on ECGs and have been thought to reflect LA enlargement, LA hypertension, and altered conduction.<sup>19</sup> The abnormal ventricular systolic and diastolic dysfunction induced by RVAP results in increased left atrium after-load;

therefore, the left atrium pressure will increase, and the atrial myocardium will be stretched, possibly leading to left atrium remodeling and, later, the development of atrial arrhythmias. A previous study demonstrated that acute RVAP could induce LA enlargement and impaired atrial contractility.<sup>17</sup> Another study documented that long-term RVOT pacing was associated with less adverse left atrium remodeling.<sup>15</sup> These 2 studies, however, did not discuss the effect of different pacing sites on P waves, which represent atrial electroactivity. In our study, we observed the effect of chronic RVAP and RVOT septal pacing on both the left atrium structure and electroactivity. Compared with RVAP, RVOT septal pacing maintained the ventricular synchronism and prevented LV remodeling, resulting in less increase in the LV filling pressure and E/Ea ratio and in left atrium pressure; therefore, the LA volume index,  $P_{\max}$ , and PWD of patients in the RVOT septal pacing group were less than those of the RVAP group.

In the current study, only patients with normal LV function were included. Consequently, whether the findings of this study are applicable to patients with underlying heart disease or LV dysfunction, or both, remains unclear. To control the systematic and accidental errors that can occur during electrocardiographic and echocardiographic data acquisition, all electrocardiographic and echocardiographic examinations were performed by the same experienced technicians of the departments of electrocardiography and echocardiography, respectively. The final result for each patient was reviewed by the chief technicians. Any uncertainties regarding results were resolved by discussion. All technicians were blinded to clinical data and group division. The number of patients in this study was insufficient to determine whether prolonged PWD increases the incidence of atrial fibrillation.

In conclusion, compared with RVAP, RVOT septal pacing showed fewer adverse effects on  $P_{\max}$ , PWD, and QRSd in patients with normal cardiac function, indicating that RVOT septal pacing maintains more satisfying atrial and ventricular electrophysiology.

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## Disclosures

None.

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