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Original article

Predictors of an adverse clinical outcome in patients with long-term right ventricular apical pacing

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ABSTRACT

Background: Right ventricular (RV) apical pacing can result in progressive left ventricular (LV) dysfunction and contribute to the development of heart failure (HF). This study aimed to predict the outcome after long-term RV apical pacing in patients with acquired atrioventricular (AV) block who required permanent pacing.

Methods: We included 247 patients who underwent long-term (>90% ventricular pacing with atrioventricular synchrony for more than 1 year) RV apical pacing for acquired AV block. We excluded patients with a reduced LV systolic function [ejection fraction (EF) <50%]. The paced QRS duration, degree of the axis, clinical characteristics, laboratory findings, and echocardiographic parameters were recorded. We evaluated the mortality and hospitalization due to HF.

Results: The mean follow-up duration was 6.9 years. Mortality and hospitalization due to HF occurred in 8.1% and 17%, respectively. In a multivariate analysis, a wider paced QRS duration and less superior paced QRS axis at the time of the implantation were independent risk factors for adverse events. The patients with a paced QRS duration of ≥163 ms and axis of ≥−65° had a 5.8 times higher risk for adverse events compared to those with a paced QRS duration of <163 ms and axis of <−65°.

Conclusions: The paced QRS duration and axis could help us predict adverse clinical outcomes after permanent RV apical pacing in patients with high-degree AV block.

Introduction

Cardiac pacing is the only effective treatment for symptomatic atrioventricular block (AVB). However, there have been several studies that have indicated that long-term right ventricular (RV) apical pacing is associated with left ventricular (LV) dilatation [1] or a decreased LV ejection fraction [2]. Large-scaled randomized studies also showed an increased hospitalization rate due to heart failure (HF) in patients with long-term RV apical pacing [3,4]. The decreased LV function may be due to abnormal electrical and mechanical activation patterns of the ventricles due to chronic RV pacing [5]. There have been studies that have suggested that the QRS duration and left-axis deviation could be related to the development of HF [6]. Nevertheless, there are no robust electrocardiographic (ECG) features that can predict cardiac outcomes. This study aimed to investigate the paced QRS features that could help predict the cardiac outcome after chronic RV apical pacing in patients with acquired AVB.

Materials and methods

Study population

This study comprised 247 consecutive patients who underwent long-term (>90% ventricular pacing with atrioventricular synchrony for more than a year) RV apical pacing for acquired high degree AVB from October 1995 to December 2012. We only included patients who were implanted with a DDD or VDD pacemaker to maintain atrioventricular sequential pacing. We excluded the patients with an LV ejection fraction of less than 50%, significant valvular disease, or any type of cardiomyopathy or atrial fibrillation before the pacemaker implantation. The demographic characteristics, laboratory findings, ECG findings, and echocardiographic

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findings were collected retrospectively. The current study was conducted in accordance with the declaration of Helsinki, and the Institutional Review Board of the Daegu Catholic University Medical Center approved the study protocol.

Electrocardiography

All ECGs were recorded at a speed of 25 mm/s. The ECG was sequentially obtained right after the implantation of a permanent pacemaker and repeated every year after the implantation. We collected data in terms of the paced QRS duration and axis. The paced QRS duration was assessed at the widest QRS duration at all paced QRS leads. The paced QRS axis was calculated from the voltage of lead I and lead aVF using trigonometry. All interrogated data from the pacemaker, which were obtained every 6 months, were reviewed to confirm the proportion of ventricular pacing.

Echocardiography

Echocardiography was done before the pacemaker implantation and followed up a year after. We also performed echocardiography in situations of cardiac events as well. The LV ejection fraction, LV end-diastolic and end-systolic dimensions, and left atrial diameter were obtained and recorded. The LV ejection fraction was measured by Simpson's method using apical 4 chamber view and apical 2 chamber view.

Pacemaker implantation

All patients were implanted with a pacemaker lead in right ventricular apex. Under fluoroscopic guidance, the pacing lead was inserted to right ventricle via superior vena cava and right atrium. After advancement of lead as far as possible toward the RV apex, we confirmed whether the tip of lead was stably lodged in RV apex in fluoroscopic imaging. ECG was also checked to find out negative QRS axis in lead II, III, and aVF. After the procedure, we checked chest X-ray daily to confirm the position of lead for 5 days. The cardiothoracic ratio in initial chest X-ray was calculated and recorded.

Clinical outcomes

As a primary outcome, the composite of the cardiac events including the all-cause mortality and hospitalization due to HF were evaluated. Further, each of the all-cause mortality and hospitalizations due to HF were analyzed separately as secondary outcomes.

Statistical analysis

The statistical evaluation was performed using SPSS software package version 18.0 for Windows (SPSS Inc., Chicago, IL, USA). A chi-square and t-test were used to compare the demographic characteristic, and laboratory, electrocardiographic and echocardiographic findings between the groups with events and those without events. A logistic regression analysis was used for the detection of the risk factors of cardiac events. A receiver-operating characteristic (ROC) curve was used to determine the cut-off values of the QRS duration and axis for the development of events. We used a Kaplan–Meier curve to describe the event-free survival rate in different risk groups determined by the paced QRS duration and axis deviation. All analyses required a p-value <0.05 for statistical significance.

Results

The follow-up duration was 6.7 ± 3.9 years. Among the patients, 51 (20.6%) developed events that were adjudicated as primary outcomes. Death from any cause occurred in 20 (8.1%) patients, and hospitalization due to HF occurred in 42 (17.0%) patients. The characteristics of the patients who developed events or not are described in Table 1. The age, gender, and presence of hypertension, diabetes, or coronary artery disease did not differ between the groups with events and those without events. There was no significant difference in the medications such as beta blockers, angiotensin-converting enzyme inhibitor, or angiotensin receptor blockers. Among the ECG parameters, the paced QRS duration was longer (167.3 ± 18.2 ms vs. 158.6 ± 17.9 ms, $p = 0.002$) and the degree of the paced QRS axis was less superior (−64.2 ± 7.2 vs. −68.4 ± 10.6, $p = 0.001$) in the patients who developed cardiac events. Among the conventional risk factors for poor cardiovascular outcomes, the LV ejection fraction was confirmed to be an independent risk factor for events. Also, a longer QRS duration and a lesser superior axis were independent risk factors of events (Table 2). The odds ratio of the QRS duration was 1.029 (CI 1.001–1.058, $p = 0.045$) for deaths and 1.047 (CI 1.010–1.053, $p = 0.004$) for hospitalizations due to HF, respectively. The odds ratio of the QRS axis was 1.031 (CI 1.002–1.093, $p = 0.038$) for deaths and 1.054 (CI 1.018–1.091, $p = 0.003$) for hospitalizations due to HF, respectively.

Table 1
The characteristics of patients with cardiac events.

	Event(+) ($n = 51$)	Event(−) ($n = 196$)	p-value
Age at implantation, years	66.4 ± 12.2	65.5 ± 13.5	NS
Female, n (%)	31(60.8)	201(61.2)	NS
Hypertension, n (%)	22(43.1)	83(42.3)	NS
Diabetes, n (%)	11(21.6)	36(18.4)	NS
CAD, n (%)	5(9.8)	15(7.7)	NS
Medication, n (%)			
Beta blocker	7(13.7)	22(11.2)	NS
ACEi/ARB	16(31.4)	45(23.0)	NS
ECG after implantation			
Paced QRS duration, ms	167.3 ± 18.2	158.6 ± 17.9	0.002
Paced QRS axis,°	−64.2 ± 7.2	−68.4 ± 10.6	0.001
Initial cardio-thoracic ratio, %	53.0 ± 4.6	51.8 ± 5.8	NS
Initial echocardiography			
LVEF, %	60.0 ± 7.8	62.9 ± 7.0	0.012
LVEDD, mm	51.9 ± 7.4	50.5 ± 6.1	NS
LVESD, mm	34.7 ± 7.6	32.1 ± 5.6	0.008
LA dimension, mm	41.5 ± 7.7	39.1 ± 6.7	0.023
New onset AF, n (%)	15(28.3)	72(35.3)	NS
Follow-up echocardiography			
LVEF, %	49.9 ± 15.5	58.8 ± 8.9	<0.001
LVEDD, mm	53.6 ± 10.1	48.5 ± 10.0	0.003
LVESD, mm	38.7 ± 11.9	31.9 ± 7.9	<0.001
LA dimension, mm	43.7 ± 7.6	39.2 ± 8.3	<0.001

CAD, coronary artery disease; AF, atrial fibrillation; ACEi, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; LVEF, left ventricular ejection fraction; LVEDD/LVESD, left ventricular end-diastolic/systolic dimension; LA, left atrium.

Fig. 1 describes the ROC curve showing the QRS duration and axis for the development of cardiac events. The best cut-off values of the QRS duration and axis for predicting cardiac events were 163 ms and −65°, respectively. Among the patients with a paced QRS duration of ≥163 ms, 29.3% developed cardiac events. In those with a paced QRS axis of ≥−65°, 28.4% developed cardiac events. The paced QRS duration and axis were also independent risk factors for composite cardiac events including all-cause mortality and hospitalizations due to HF. Using the best cut-off values of the QRS duration and axis for the development of cardiac events, we divided all patients into four different groups: group I, which included patients with a QRS duration of <163 ms and axis of <−65°, group II, which included patients with a QRS duration of <163 ms and axis of ≥−65°, group III, which included patients with a QRS duration of ≥163 ms and axis of <−65°, and group IV, which included patients with a QRS duration of ≥163 ms and axis of

Table 2
The risk factors of cardiac event.

	Univariate analysis			Multivariate analysis		
	OR	CI	*p*-value	OR	CI	*p*-value
Age	1.005	0.981–1.029	NS			
Hypertension	1.033	0.554–1.925	NS			
CAD	1.312	0.453–3.796	NS			
New onset AF	0.724	0.373–1.404	NS			
Initial LVEF	0.947	0.907–0.988	0.013	0.953	0.912–0.996	0.031
Initial LVESD	1.070	1.016–1.126	0.010			
Initial LAD	1.053	1.007–1.102	0.024	1.048	1.001–1.097	0.047
QRS duration	1.030	1.010–1.051	0.003	1.025	1.007–1.043	0.007
QRS axis	1.037	1.008–1.067	0.012	1.012	1.003–1.020	0.007

CAD, coronary artery disease; AF, atrial fibrillation; LVEF, left ventricular ejection fraction; LVESD, left ventricular end-systolic dimension; LAD, left atrial dimension.

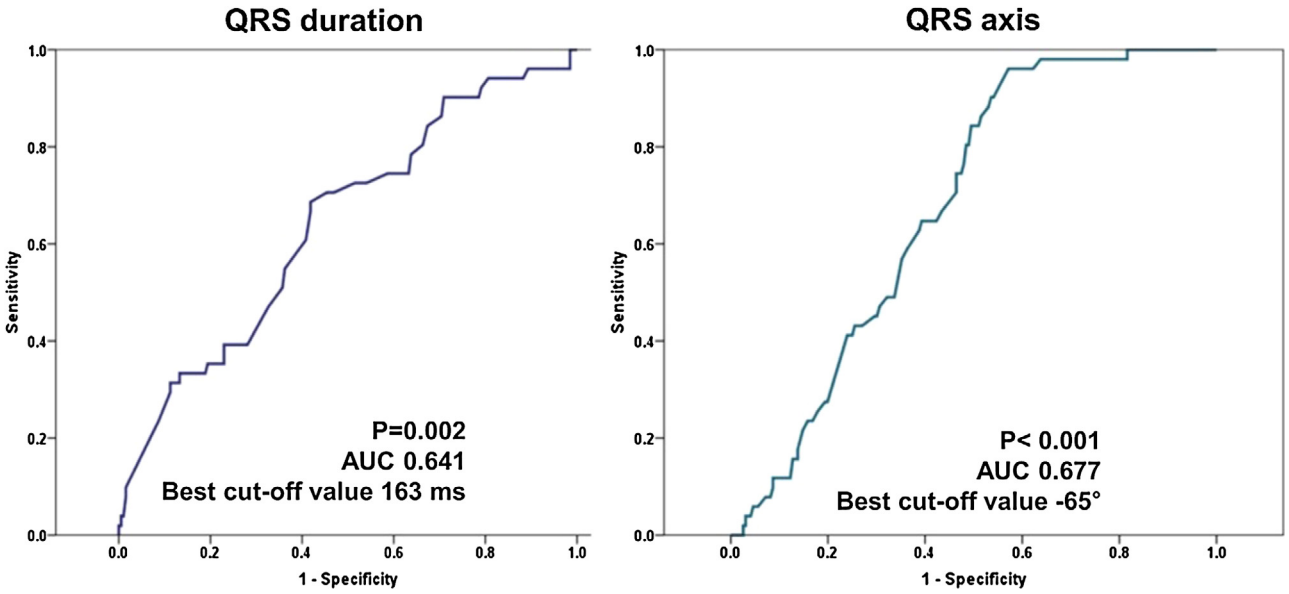

Fig. 1. Receiver-operating characteristic curve of paced QRS duration and QRS axis for cardiac event. The paced QRS duration and QRS axis were related with the composite cardiac events including mortality and admission from congestive heart failure. AUC, area-under-the-curve.

$\geq -65^{\circ}$. The populations of group I to IV were 88, 43, 78, and 38 patients, respectively. There was a significant gradual increase in the risk of cardiac events from group I to group IV (Table 3). In the patients of the paced QRS duration of <163 ms, the less superior QRS axis ($\geq -65^{\circ}$) had higher risk of composite cardiac events by 2.6-fold compared to patents who had the paced QRS duration of <163 ms and the more superior QRS axis ($<-65^{\circ}$). In addition, the patients of the wider paced QRS duration (≥163 ms) and the less superior QRS axis ($\geq -65^{\circ}$) presented increased risk by 5.8-fold compared to the patients with the less wide paced QRS duration (<163 ms) and the more superior QRS axis ($<-65^{\circ}$). A Kaplan–Meier curve demonstrated the difference in the event-free survival rate of the different risk groups using the paced QRS duration and axis as shown in Fig. 2. The event-free survival rate gradually decreased from group I to group IV with statistical significance.

Table 3
Gradual increase of risk according to paced QRS duration and axis.

	OR	CI	*p*-value
Group I (QRSd < 163 ms and axis $<-65^{\circ}$), *n* = 88	1		
Group II (QRSd < 163 ms and axis $\geq -65^{\circ}$), *n* = 43	2.647	0.942–7.440	0.065
Group III (QRSd ≥163 ms and axis $<-65^{\circ}$), *n* = 78	3.448	1.421–8.371	0.006
Group IV (QRSd ≥163 ms and axis $\geq -65^{\circ}$), *n* = 38	5.833	2.187–15.561	<0.001

QRSd, QRS duration.

Discussion

In this study, we found the risk factors that could predict a poor clinical outcome after permanent RV apical pacing in patients with acquired AVB. Because all patients included maintained AV synchrony (DDD or VDD pacing mode) and had a cumulative ventricular pacing rate of >90%, we could provide reliable parameters in terms of pacing rhythm which were related with poor clinical outcome. A paced QRS duration of more than 163 ms and axis of more than -65° for RV apical pacing increased the risk of long-term outcomes by 5.8-fold.

Pacing modes and clinical outcomes

Chronic RV pacing is reported to decrease the LV function [2]. It is possibly due to the non-physiologic activation pattern of the interventricular septum and abnormal electrical and mechanical

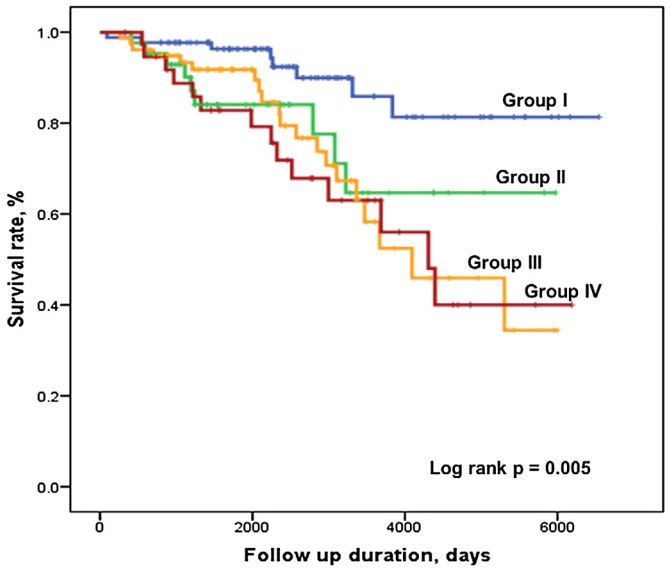

Fig. 2. Event-free survival rate according to QRS duration and axis deviation. The longer paced QRS duration and less superior QRS axis had incremental effect on cardiac events.

activation patterns of the ventricles such as left bundle branch block [5]. The abnormal electrical and mechanical activation causes LV dyssynchrony, which could cause the LV systolic and diastolic function to deteriorate [7]. Andersen et al. [8] demonstrated that physiologic pacing maintaining AV synchrony can significantly reduce the rate of heart failure compared to ventricular pacing in patients with sick sinus dysfunction. However there had been studies showing that physiologic pacing with dual-chamber devices was not related with outcomes [9] or even associated with a higher incidence of new-onset HF [10]. In the present study, we enrolled all patients with only physiologic pacing with dual-chamber devices to avoid the expected bias.

Cumulative ventricular pacing rate

RV apical pacing causes chronic changes in the regional myocardial perfusion [11], cellular structure [12], and ventricular geometry [13] that may impair the ventricular performance such as left bundle branch block. Some clinical trials have reported that a higher cumulative RV pacing rate was associated with an increased risk of HF, especially among patients to whom RV apical pacing is not essential [14,15]. In the sub-analysis of the Mode Selection Trial (MOST) [16], cumulative ventricular pacing of >40% of the time in the dual-chamber pacing mode was associated with a 2.6-fold increased risk of heart failure hospitalizations compared to pacing of <40% of the time. In addition, the MOST trial reported 12% of heart failure hospitalizations among 361 patients with a cumulative ventricular pacing rate of >90% as the same inclusion criteria as the present study. Consistent with the MOST study, the rate of HF hospitalizations was 17% during the mean follow-up of 7.8 years in our study.

Paced QRS duration as a predictor of cardiac events

Permanent RV apical pacing resembles a left bundle branch block pattern and causes prolongation of the QRS duration. A wide QRS width has been suggested as a potential predictor of the development of LV dyssynchrony in cardiac pacing [17] as well as an indicator of LV dyssynchony itself. Miyoshi et al. [18] reported that a prolonged paced QRS duration of ≥190 ms was associated with a significant increase in the overall morbidity of congestive HF during a mean follow-up period of 53 months, in their study which enrolled some elderly patients (mean age 73 years) including single-chamber pacing (14.1%). On the other hand, Zhang et al. [10] demonstrated that a paced QRS duration of ≥165 ms was associated with an increased risk of HF after RV apical pacing in patients with high degree AVB. Consistent with the previous studies, we found that a longer QRS duration was associated with an increased risk of cardiac events.

Paced QRS axis as a predictor of cardiac events

The paced QRS axis could differ according to the location of the RV lead as well. The paced QRS axis and duration were not similar to the original QRS due to the altered intraventricular activation sequence. The development of left-axis deviation has been reported to predict the mortality in patients with left bundle branch block [17]. However, little was known about the mechanism of the outcome from the left-axis deviation and degree of deviation that is related to a poor outcome. A study previously had suggested left-axis deviation as a component of the risk score for interventricular dyssynchrony in patients with chronic RV apical pacing [6]. A lead placement in the RV apex results in a superior paced axis (i.e. negative in the inferior leads), and the degree of the superior paced axis could change according to the location of the RV lead between the septum and free wall of the RV apex. A more superior axis results from being closer to the septum of the RV. We found that the QRS axis as well as the QRS duration was related to the clinical outcome. In the present study, we found that a less superior axis, that is ≥−65°, was associated with cardiac events. Although all patients had left-axis deviation, between −87.3° and −21.0°, because all of them were implanted with a pacemaker lead in the RV apex, we could calculate the best cut-off value of the QRS axis for predicting a poor outcome.

Placement of the RV lead

Recently the placement of pacemaker leads at alternative sites such as RV outflow tract instead of the RV apex has gathered consensus [19]. The rationale for the alternative pacing site is that maintaining AV synchrony could prevent ventricular dyssynchronization. However, there have been data that have shown that a wide QRS duration was associated with poor outcomes also in patients with an implanted pacemaker with a lead in the RV outflow tract [20], and the mortality rate was similar in RV apical pacing and septal pacing [21]. In our study, about 80% of patients with a paced QRS duration of less than 160 ms and an axis of less than −70°, had no adverse events over the long-term follow-up. We can suggest the RV apical pacing would be favorable outcome if the paced QRS duration is narrow and the more superior axis right after lead implantation.

Limitations

The present study was a retrospective study including a relative small number of patients. Because the mean age was 65.7 years, there was a relatively high mortality rate of 8.1%. In some patients the cause of death could not be identified because they died at home without visiting the hospital. However, concerning the odd ratios of deaths and hospitalizations due to HF, the major cause of higher cardiac event rate in patients with wider QRS duration and less superior QRS axis could be considered to result from higher hospitalization rate due to HF. Although we could not find out all causes of deaths in this study, it could provide insight into the long-term outcome in patients with chronic RV pacing and the predictors of the outcome after a pacemaker insertion.

Conclusion

A wide paced QRS duration and less superior axis could help to predict adverse clinical outcomes after permanent RV apical pacing in patients with high degree AVB. So, if the ventricular lead was implanted in the RV apex, the accommodation of the lead position to acquire a narrow QRS duration and a more superior axis would be beneficial for reducing the mortality and hospitalizations due to HF thereafter.

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Disclosures

None of the authors have any potential conflicts of interest associated with this research.

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