Idiopathic ventricular arrhythmias originating from the right coronary sinus: Prevalence, electrocardiographic and electrophysiological characteristics, and catheter ablation

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BACKGROUND Ventricular arrhythmias (VAs) of the right coronary cusp (RCC) are not fully characterized.

OBJECTIVES To investigate the electrocardiographic and electrophysiological characteristics, mapping and ablation of RCC-VAs.

METHODS Among 256 consecutive patients undergoing electrophysiological evaluation and ablation of VAs of ventricular outflow tract origin, data were compared among 27 RCC-VAs, 50 VAs of the septal aspect of right ventricular outflow tract (RVOT), including from pulmonary artery, and 9 VAs of left coronary cusp (LCC).

RESULTS The only electrocardiographic characteristic that differentiated VAs originating from the RCC and RVOT was the amplitude of the R wave in lead I. During VAs of the RCC, the earliest activation site (EAS) in the right ventricle was localized in the middle-posterior septal region of the RVOT. The distance between the His bundle and the EAS in the RVOT in the RCC group was shorter than that in the RVOT and LCC group; the distance ≤ 29.4 mm, which rules out an RVOT and LCC origin, had 92.6% sensitivity and 100% specificity for RCC-origin speculation. Double or complex potentials were recorded in RVOT middle-posterior septal area surrounding the EAS in 20 of 27 RCC-VA patients (70%). Most of the successful ablation sites (24/27) were located in the anterior and upper margin of the RCC, close to the middle-posterior septal region of the RVOT. The prepotential (P1) amplitude and the P1-to-QRS complex interval may be indicators of successful RCC-VA ablation sites.

CONCLUSIONS RCC-VAs are not uncommon and have unique electrocardiographic and electrophysiological characteristics that distinguish an RCC origin of VA from RVOT and LCC origins. Most RCC-VAs were ablated successfully in the anterior and upper aspects of the RCC.

KEYWORDS Catheter ablation; Echocardiography; Electrophysiology; Right coronary cusp; Ventricular arrhythmias

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Introduction

Idiopathic ventricular arrhythmias (VAs), which are commonly treated with radiofrequency catheter ablation, most often arise from the right ventricular outflow tract (RVOT) or left ventricular outflow tract (LVOT). Although differences in electrocardiographic characteristics of VAs originating from RVOT and aortic sinus cusp (ASC) regions have been documented,1–3 there are limited published data on electrocardiographic and electrophysiological characteristics of VAs originating from the right coronary sinus (RCC), which is the subject of this study.

Methods

Study population

The study population consisted of 27 consecutive adult patients (11 men, 16 women; mean age 44 ± 24 years) with VAs originating from the RCC. Of 27 patients, 13 had frequent premature ventricular contractions (PVCs), and 14 of 27 manifested both PVCs and ventricular tachycardia. The 27 patients were derived from a series of 256 consecutive patients undergoing electrophysiological evaluation and ablation of idiopathic VAs from the ventricular outflow tract. The remaining patients had VAs from RVOT (VAs from pulmonary artery could be included) (n = 158), LVOT (n = 25), left coronary cusp (LCC) (n = 35), noncoronary cusp...
(n = 1), the distal great cardiac vein (n = 2), proximal anterior interventricular vein (n = 1), left ventricular summit (n = 4). Left ventricle (LV) epicardial origin was suspected after failed ablation in the remaining 3 patients. The study was approved by the institutional review board of Beijing Anzhen Hospital, and affiliate of Capital Medical University.

Surface electrocardiogram and electrophysiological study
For comparison, electrocardiograms (ECGs) were analyzed from 50 consecutive patients with idiopathic VAs arising from the septal aspect of the RVOT (VAs from pulmonary artery could be included). Relevant ECG data known to differentiate between RVOT and LVOT VAs, such as R-wave amplitude in lead I,4 V2 transition ratio,5 precordial transition zone,6 and V2S/V3R,7 were collected for analysis.

Mapping and ablation
If the VAs showed left bundle-branch block QRS morphology, activation and pace mapping were initially performed in the RVOT (pulmonary artery also might be included) with NaviStar or SmartTouch catheter (CARTO, Biosense Webster, Irvine, CA). Whenever RVOT mapping failed to identify an early activation site or ablation failed, mapping of the ASC and LVOT was performed retrogradely. Pace mapping was performed at a stimulus amplitude of 1 mA greater than the diastolic threshold before ablation. RVOT mapping data in the RCC group were subsequently compared with the previously mentioned 50 consecutive VAs from the RVOT (RVOT group) and 9 VAs from the LCC (LCC group). The distance of the EAS in RVOT to His bundle (HB) in these 3 groups was measured using CARTO. The distance between a successful ablation site at the RCC and the distance from a successful ablation site at the RVOT mapping and electrophysiological characteristics of the RVOT group and the LCC group were calculated with previously mentioned 50 consecutive patients (Figure 1). Only 5 patients had previously undergone ablation in the RVOT. Most ECG parameters that were used to predict the site of origin in LVOT did not show great sensitivity and specificity in these 2 groups (Supplementary Table S1). In contrast, the R-wave amplitude in lead I was higher in the RCC than in the RVOT group (0.52 ± 0.35 mV vs 0.19 ± 0.18 mV, P < .05), and it showed the greatest sensitivity (81.5%) and specificity (84.6%).

Analysis of electrograms in the ASC
Quantitative analysis of electrograms recorded by ablation catheters in the ASC was performed. Using electronic calipers of Bard LabSystem (Boston Scientific, Marlborough, MA), measurements of activation time and electrogram amplitude were performed. Analysis of electrograms during VAs in the ASC consisted of the following measurements: incidence of presystolic potential preceding QRS complex (P1 potential), P1 potential amplitude, and interval from P1 potential to QRS complex onset. The incidence of potential following the QRS complex (P2 potential) recorded during sinus rhythm was also analyzed. The electrograms recorded during VAs at successful ablation sites were compared with the electrograms recorded during VAs at all the unsuccessful ablation sites.

Statistical analysis
Statistical analysis was performed using SPSS version 19 (IBM, Armonk, NY). Continuous variables are expressed as mean plus or minus standard deviation, and they were compared using the t test. A receiver operating characteristic analysis was used to calculate sensitivity and specificity. The optimal cutoff value of the distance between the HB and the EAS in the septal RVOT in the RCC group vs those in the RVOT group and the LCC group were calculated with the greatest area under the curve. Differences were considered statistically significant when P < .05.

Results
Patient characteristics and ECG analysis
During VAs, the surface ECG showed a typical left bundle-branch block morphology with an inferior axis in all 27 patients (Figure 1). Only 5 patients had previously undergone ablation in the RVOT. Most ECG parameters that were used to predict the site of origin in LVOT did not show great sensitivity and specificity in these 2 groups (Supplementary Table S1). In contrast, the R-wave amplitude in lead I was higher in the RCC than in the RVOT group (0.52 ± 0.35 mV vs 0.19 ± 0.18 mV, P < .05), and it showed the greatest sensitivity (81.5%) and specificity (84.6%).

RVOT mapping and ablation
RVOT mapping and electrophysiological characteristics of the 27 VAs from the RCC group were compared with those of 50 VAs from the RVOT group and 9 VAs of the LCC group. In 27 patients from the RCC group, the RVOT EAS was located in the middle-posterior septal area, preceding the onset of the QRS complex by 28 ± 9.8 ms (range 19–30 ms) (Figures 2, 3A, and 4A). The local potential in the EAS was double or complex in 20 of 27 patients (70%); in addition, a small area of double or complex potentials was recorded in the posterior-middle septal RVOT surrounding the EAS, a finding that was rarely observed in the RVOT and the LCC groups (Figure 2). The activation map of the RVOT septal area revealed that activation propagated from the middle septal toward the superior and inferior septal regions.
Pace mapping was performed in 19 of these 27 patients. In contrast to a good pace mapping match in most patients in the RVOT group (45/50 patients, 90%), a poor pace mapping match was obtained in the EAS of RVOT in 15 of 19 patients (78.9%) in the RCC group (Figure 3) and in 9 of 9 patients (100%) in the LCC group. The distance between the HB and the EAS in the septal RVOT in RCC group (Figures 4A and 4B) was shorter than that in the LCC group (Figures 4C and 4D) (25.9 ± 4.3 mm vs 42.5 ± 7.6 mm, \( P \leq 0.05 \)) and the RVOT group (Figure 4E) (25.9 ± 4.3 mm vs 47.7 ± 9.3 mm, \( P \leq 0.05 \)). As a consequence, distances ≤29.4 mm had 92.6% sensitivity and 100% specificity for...
the RCC origin. Therefore, a cutoff value of \( \leq 29.4 \) mm rules out RVOT or LCC origin (Figure 4F). The distance between the HB and the EAS in the septal RVOT was observed only in 2 patients in the RCC group. An early potential was recorded in the left pulmonary sinus cusp in 1 of the 2 patients, and ablation at this site was transiently effective, but a poor pace mapping match was obtained. Finally, an early and perfect potential was recorded in the RCC, and ablation was successful.

Ablation was attempted in the EAS of the RVOT in 9 patients and was transiently effective in 6 patients. VAs were terminated within 10 seconds during RF ablation and recovered in 5 ± 3 minutes (range 2–8 minutes) in all 6 of these patients. Several additional RF ablation procedures were attempted; however, VAs were not abolished completely in all 6 patients, whereas VAs were completely abolished in the RCC. No ablation was performed in 18 patients, including 5 patients who had previously undergone ablation in the RVOT. In 2 of these 5 patients with previous ablation, prior successful ablation was achieved in the RVOT, and the VAs recurred after 2 weeks.

ASC mapping and ablation

In 24 of 27 patients with VAs originating from the RCC, the VAs were successfully ablated in the anterior aspect of the RCC with an average and standard deviation of 2.8 ± 3.0 ablation attempts (range 1–9 attempts); the earliest activation in a successful site preceded QRS onset by 31 ± 13.1 ms, and distance between the EAS of the RVOT and a successful ablation site in the RCC was only 12.3 ± 2.6 mm as determined using CARTO (Figures 4G and 4I), whereas the distance was significantly greater in the LCC group (22.3 ± 5.9 mm) (Figures 4H and 4I). Compared with the EAS recorded in the RVOT in the RCC group, the EAS preceding QRS complex onset in the target recorded in the RCC was not obviously earlier (Figures 3A and 3C). Pace mapping was performed at the EAS before RF ablation in all 24 patients, but a perfect pace mapping could not be achieved in most patients (20/24), and a longer pacing stimulus–QRS interval was observed in 10 of 24 patients (Figure 3D). The anatomic position of the anterior aspect of the RCC was determined using a combination of electroanatomic mapping, fluoroscopic angiography, and ICE. In the RAO projection, the ablation catheter was typically located anterior and inferior to the right coronary artery (RCA) ostium (Figure 5A). In the LAO projection, the typical ablation site was more leftward in the cusp than the RCA ostium and superior to the RCC nadirs (Figure 5B). The average and standard deviation of the distance from a successful RF site to the RCA ostium was 12.2 ± 3.2 mm and to the bottom of the RCC was 7.2 ± 0.8 mm in the RAO and LAO views. ICE further confirmed that the ablation catheter tip was located in anterior and upper aspects of RCC (Figure 5C) in 3 patients. In these 24 patients, no His potentials were found at successful sites. However, distinct His potentials were recorded at the posterior portion or base of RCC in 13 patients.

In 3 patients, an early near-field presystolic potential (P1) could be recorded in RCC and several ablations suppressed
the VAs but did not eliminate it. Additional mapping and ablation then was performed below the aortic valve and the VAs was abolished successfully (Figures 6A–6C). ICE results (Figure 6D) and CARTO results (Figures 6E and 6F) confirmed that the ablation catheter tip was located below the valve at almost the same level of the ablation sites of the RCC.

**Figure 4** Mapping and measurement in the right ventricular outflow tract (RVOT) and aortic sinus cusp (ASC) in different groups. The distance between the His bundle (HB) and the earliest activation site (EAS) in the septal RVOT was A and B: 22.8 mm in the right coronary cusp (RCC) group, C and D: 55 mm in the left coronary cusp (LCC) group, and E: 33.8 mm in the RVOT group. F: A cutoff value of ≤29.4 mm rules out RVOT and LCC origin, and had 92.6% sensitivity and 100% specificity for RCC origin. G: The distance between the EAS of the RVOT and successful ablation site at ASC was shorter in RCC group compared with that in the H and I: LCC group. Yellow point: His bundle. White point: RCC. Deep blue point: LCC. Pink point: noncoronary sinus. Light blue point in RVOT: EAS. Light blue point in ASC: the successful ablation site. n = 27 in the RCC group. n = 50 in the RVOT group. n = 9 in the LCC group.

**Figure 5** Angiogram of the coronary artery and aorta and intracardiac echocardiography showing catheter tip at successful ablation site in the right coronary cusp (RCC). The distance from the successful ablation site to the right coronary artery ostium was 13.2 mm and to the bottom of RCC was 7.1 mm in the A: right anterior oblique (RAO) and B: left anterior oblique (LAO) views. C: Intracardiac echocardiogram confirmed that the ablation catheter tip was located at anterior and upper aspects of the RCC. NCC = noncoronary sinus; PA = pulmonary artery.
Analysis of electrograms in the ASC

Two activation components were found at the successful site in all 24 patients with VAs of RCC origin. The first component was near-field presystolic potential (P1) with high frequency. The second component was far-field ventricular potential (Figures 3C and 7D). During sinus rhythm, an obvious late P2 potential was recorded in the RCC in only 10 patients, whereas in most patients, a P2 potential was always infused in the ventricular potential during sinus rhythm, and spontaneous reversal during VAs was recorded (Figures 3C and 7D) in most patients.

The P1 potential was found at 24 of 24 successful ablation sites (100%), but it was also recorded at 46 of 60 unsuccessful ablation sites (76.6%) in 24 RCC patients. The interval from P1 potential to QRS complex onset at successful ablation sites was 31 ± 13.1 ms (Figures 3C and 7D). However, P1 potential amplitude was obviously higher at successful ablation sites under the circumstance of approximately the same interval from P1 potential to QRS complex onset, compared with that of unsuccessful ablation sites (Figures 7A–7D).

P1 potential was also found at 9 successful ablation sites in all 9 patients with VAs of LCC origin. However, there were significant differences in P1 potential amplitude at successful ablation sites between the RCC and LCC groups (0.352 ± 0.155 mV vs 0.012 ± 0.007 mV, P < .05) (Figure 7E).

Complications and follow-up

No stroke, cardiac perforation, coronary arterial damage, or damage of the aortic valve was seen in any case. In addition, all 27 patients were free of arrhythmias without antiarrhythmic drugs during 9 ± 3 months of follow-up.

Discussion

Main findings

The major findings of this study were as follows. First, when mapping in the RVOT for VAs of RCC origin, the earliest right ventricular activation was localized in the middle-posterior septal RVOT, and the distance between the EAS and HB was relatively shorter. Second, for VAs of RCC origin, double or complex potentials may be recorded in the middle-posterior septal RVOT surrounding the EAS. Third, most successful ablation sites were located in the anterior aspect of the RCC and most were above the RCC base. Fourth, P1 amplitude and P1-to-QRS complex interval may be indicators of successful ablation sites for RCC-VAs. Fifth, the anterior aspects of the RCC are close to the middle-posterior septal area of the RVOT, and ablation in the RVOT may transiently suppress the VAs of RCC origin. Sixth, when ablation was not successful in the RCC, further mapping below the aortic valve was useful.
ECG analysis

Yamada et al. described a qR pattern in leads V1–V3 for VAs originating from the LCC-RCC junction in 5 patients. In a study by Bala et al., a QS morphology in lead V1 with notching on the downward deflection was present in 15 of 19 VAs originating from the RCC-LCC commissure. In the present study, most VAs also were confirmed to originate from the anterior, upper margin of the RCC, near or at the RCC-LCC junction. These VAs were defined as RCC-VAs, because all could be ablated successfully in the RCC. However, a qR pattern and QS morphology with notching on downward deflection in lead V1 were present in only 4 of 27 and 5 of 27 VAs from the RCC, respectively. The discrepancy may be attributed to the relatively small sample sizes, different definition, and cardiac spatial orientation. In the present study, among ECG parameters analyzed, only R amplitude in lead I was significantly different between VAs of the RVOT and the RCC, as Ebrille et al. had described.

Figure 7  Comparison of electrograms from ablation sites in the aortic sinus cusp. P2 potential (arrowhead) was infused in the ventricular potential in sinus rhythm, and spontaneous reversal could be recorded during ventricular arrhythmias, which was defined as P1 potential (arrows) at that time. The interval from P1 potential to QRS complex onset was A: 22 ms, B: 44 ms, and C: 44 ms at 3 unsuccessful ablation sites in the right coronary cusp (RCC), respectively. P1 potential amplitude was A: 0.134 mV, B: 0.154 mV, and C: 0.247 mV at 3 unsuccessful ablation sites in the RCC, respectively. D: P1 potential amplitude (0.371 mV) was obviously higher at successful ablation sites under the circumstance of approximately the same interval (44 ms) from P1 potential to QRS complex onset, compared with that of unsuccessful ablation sites. E: The interval from P1 potential to QRS complex onset was 44 ms at successful ablation sites in the left coronary cusp. In contrast, P1 potential amplitude at successful ablation sites in LCC was 0.095 mV, which was obviously lower than that in the RCC group.
ROVT mapping, electrograms and ablation

Yamada et al\(^1\) reported VAs originating from the RCC and the noncoronary sinus, with earliest right ventricular activation recorded in the HB region. In the present study, in contrast to the study by Yamada et al,\(^3\) the earliest activation of the right ventricle was recorded mostly in the posterior and middle septal RVOT, which was superior to the HB. The difference might be explained by the VAs in Yamada’s study possibly mostly originating from the posterior margin of RCC near the noncoronary sinus, which was a very rare phenomenon in the present study and was excluded according to QRS morphology of outflow tract VAs. Recently, Acosta et al\(^8\) described an algorithm based on the EAS-pulmonary valve (PV) distance to predict LVOT vs RVOT origin of outflow tract VAs. This method has 60.8% sensitivity to predict an LVOT site of origin. Some VAs originating from the LCC and RVOT could not be differentiated because the distance of the EAS to the PV was measured in a direction parallel to the longitudinal axis of the RVOT, and the origins of the RVOT and the LCC are both higher than the RCC. The method might be good at differentiating RCC origin; however, the study included only 5 VAs of RCC origin, which precluded further assessment. Moreover, the definition of PV by voltage mapping using an electroanatomic map is greatly influenced by contact force. Myocardial extension showed normal voltage above the PV, which might lead to an inability to delineate PV annulus accurately by voltage map.\(^9\) In addition, the 3 pulmonary semilunar valvar cusps are not at the same level, and definition of PV by voltage mapping using an electroanatomic map is laborious. One may posit that the HB location is simpler, convenient and less affected by catheter contact. In the present study, patients with VAs originating above and below the PV were all included in the RVOT group. The average and standard deviation distance of the EAS to the HB in the septal RVOT was 25.9 ± 4.3 mm in the RCC group, which was shorter than that in the RVOT and LCC groups, and the distances found in the LCC and RVOT groups were not significantly different from each other. Findings in the current study suggest that the distance between the EAS and the HB in the septal RVOT provides useful information. When this distance is <29.4 mm, an RCC origin should be considered, and careful mapping in the ASC might be necessary. Knowledge of the anatomy of the left and right outflow tracts helps us to understand these electrophysiological data. The posterior (septal) aspect of the RVOT is essentially in continuity with the RCC and the anterior aspect of the LCC, and the LCC is situated superior to the RCC. The RCC directly abuts the inferior septal aspect of the RVOT that is more adjacent to the HB.\(^10\) Moreover, for some rare cases with anatomic variation, the EAS in the RVOT in RCC-VAs could be recorded in a superior site.

The activation sequence in the RVOT of RCC-VAs has been unclear. The study of Yamada et al\(^3\) showed that the activation wave front from the left RCC spreads first toward the posterior side along the ostium of the left ventricle and thereafter turns anteriorly to the RVOT via the ventricular septum. The activation of the septal RVOT was from bottom to top in the study of Yamada et al.\(^3\) In the present study, EAS activation in the RVOT was recorded almost simultaneously with local activation of a successful ablation site in the RCC. The activation map of the septal RVOT revealed that the activation propagated from middle septal toward the superior and inferior septal regions rather than from bottom to top. The average and standard deviation distance between the EAS of the RVOT and successful ablation site in RCC was only 12.3 ± 2.6 mm as measured using CARTO. According to these findings, one may posit that the earliest ventricular electrograms of the septal RVOT may be the far-field potential of the RCC-VA activation site. Sometimes such far-field electrograms are difficult to recognize and look like high-frequency potential because of overlap with near-field electrograms owing to relatively fast transseptal conduction. Moreover, another possible interpretation might be the existence of an epicardial or subendocardial preferential muscular fiber connection between the origin of VAs and the EAS of the RVOT. Ablation in the RVOT was transiently effective in 6 of 9 (67%) patients, and ventricular arrhythmia was terminated within 10 seconds during RF ablation in all 6 patients, which may also be explained by a very short distance from the RVOT to the RCC. Therefore, when ablation is transiently effective in the RVOT, an RCC origin should be highly suspected. In 2 patients of the RCC group, prior successful ablation was achieved in the RVOT and the VAs recurred after 2 weeks, underscoring that ablation in the RVOT should not be performed before RCC mapping when an RCC origin is suspected.

ASC mapping, electrograms, and ablation

In 24 of 27 patients, the successful ablation site was located at the anterior and upper margins of the RCC by fluoroscopic angiography and CARTO. Ablation in the RCC usually requires coronary angiography in both RAO and LAO projections for accurate identification of the catheter relative to the RCA ostium. In 24 of 27 patients, no His potentials were found at successful sites, and distinct His potentials were recorded at the posterior portion or base of the RCC in 13 patients. This may be explained by anatomic location. The posterior part of the RCC is adjacent to the central fibrous body, which carries within it the penetrating portion of the HB.\(^10\) Because RCC-VAs tend to originate in the anterior portion of the RCC, catheter ablation poses a lesser risk of damage to the His and atrioventricular conduction system.

Pace mapping in the RCC did not well replicate the morphology of the QRS, which may be related to several factors, such as the distance between the pacing site and real myocardial origin of VAs, the different preferential conduction exits of VAs, the size of regional myocardium, and the output voltage of pacing. The ablation is more accurately guided by recording the earliest site during the VAs than by pace mapping.

Late potentials (P2) have been reported at successful ablation sites in the ASC during sinus rhythm.\(^1\) The presence of a presystolic potential (P1) at the site of successful ablation also
has been reported in ASC VAs. In this study, a P1 potential was often recorded at anterior margin of the RCC, and no significant difference was observed in incidence between successful and unsuccessful ablation sites. Therefore, the presence of P1 or P2 potential does not help to identify successful ablation sites. The higher P1 potential amplitude appeared more important under the circumstance of approximately the same interval from P1 potential to QRS complex onset. The P1 amplitude and P1-to-QRS complex interval may be indicators of successful ablation sites for RCC-VAs. When pacing mapping is performed on the RCC, precise pace mapping cannot be achieved, because high-output pacing is needed, and this method unfortunately captures a relatively large area. In this study, ablation in RCC was effective but unsuccessful in 3 patients, and PVCs were successfully ablated below the aortic valve, which indicated that some presumed RCC-VAs might indeed originate instead from the LV ostium emphasized by Yamada et al. Ablation in the RCC might cause thermal lesions to the actual origin. One should keep in mind that when mapping and ablation are not ideal in RCC, further mapping should be performed below the aortic valve.

Limitations
This study has several limitations. First, VAs from the RCC-LCC junction were also defined as RCC-VAs and were not analyzed separately in this study, because most VAs were confirmed to originate from the anterior, upper margin of the RCC near or at the RCC-LCC junction and all could be ablated successfully in the RCC. Second, VAs were not further precisely divided into 2 groups originating above and below the PV in the RVOT group. Finally, although successful ablation was performed in the RCC, the actual pathologic anatomy of the VAs remains to be further confirmed and explored.

Conclusions
VAs originating from the RCC have unique electrocardiographic and electrophysiologic characteristics. Most RCC-VAs could be ablated successfully in the anterior and upper aspects of the RCC. P1 amplitude and P1-to-QRS complex interval may be indicators of successful ablation sites for RCC-VAs. When mapping and ablation were not ideal in the RCC, further mapping below the aortic valve was useful.

Appendix
Supplementary data
Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrthm.2017.09.008.

References