

Prevalence and clinical, electrocardiographic, and electrophysiologic characteristics of ventricular arrhythmias originating from the noncoronary sinus of Valsalva

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BACKGROUND Idiopathic ventricular arrhythmias (VAs) can be rarely ablated from the noncoronary cusp (NCC) of the aorta.

OBJECTIVE The purpose of this study was to investigate the prevalence and the clinical, electrocardiographic, and electrophysiologic characteristics of idiopathic NCC VAs.

METHODS We studied 90 consecutive patients who underwent successful catheter ablation of idiopathic aortic root VAs (left coronary cusp [LCC] 33, right coronary cusp [RCC] 32, junction between LCC and RCC 19, NCC = 6).

RESULTS NCC VAs occurred in significantly younger patients (all <40 years old) and exhibited a shorter QRS duration (all but one <150 ms), smaller R-wave amplitude ratio in leads II and III (III/II), earlier ventricular activation in the His bundle (HB) region (all but one preceded QRS onset by >25 ms), and larger atrial to ventricular electrogram amplitude ratio (A/V) at the successful ablation site (all but one >1) than the other VAs. QRS morphology of the NCC VAs was similar to that of RCC VAs, but NCC VAs always exhibited a left bundle branch block and left superior (n = 1) or inferior axis (n = 5). All NCC VAs exhibited ventricular tachycardias,

although premature ventricular contractions were dominant in the other VAs.

CONCLUSION NCC VAs were very rare (7%) and occurred in significantly younger patients than those among the other aortic root VAs. In a limited set of six patients, the ECG and electrophysiologic characteristics of NCC VAs were similar to those of RCC VAs but were characterized by narrower QRS duration, smaller III/II ratio, earlier ventricular activation in the HB region, and A/V ratio >1 at the successful ablation site.

KEYWORDS Ventricular arrhythmia; Noncoronary cusp; Prevalence; Characteristics; Radiofrequency catheter ablation

ABBREVIATIONS ASC = aortic sinus cusp; HB = His bundle; LCC = left coronary cusp; LV = left ventricle; NCC = noncoronary cusp; PVC = premature ventricular contraction; RCC = right coronary cusp; RF = radiofrequency; RV = right ventricle; RVOT = right ventricular outflow tract; VA = ventricular arrhythmia; VT = ventricular tachycardia

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Idiopathic ventricular arrhythmias (VAs) often arise from the aortic root.^{1–7} It has been proved that these VAs can be cured by radiofrequency (RF) catheter ablation from above or underneath the aortic sinus cusps (ASCs) of the aorta.^{1–7} On the other hand, it has been recognized that some atrial tachycardias can also be cured by RF catheter ablation within the noncoronary cusp (NCC) of the aorta.^{8–10} Among the three aortic cusps, the NCC is suspended posteriorly across the left ventricular (LV) ostium and is contiguous with

the anterior leaflet of the mitral valve. Because of its anatomic location, most of the NCC is in contact with the interatrial septum, with only the most anterior and rightward extension coming into contact with the ventricle. However, several previous studies have reported successful ablation of idiopathic VAs from within the NCC in isolated cases.^{6–12} Because idiopathic NCC VAs are very rare, information on the prevalence and clinical, ECG, and electrophysiologic characteristics of these VAs is limited. The purpose of this study was to reveal these features of idiopathic NCC VAs.

Methods

Patient characteristics

The study population consisted of 90 consecutive patients from a single center (55 men; mean age 51 ± 16 years, range 13–83 years) with symptomatic idiopathic sustained ventricular tachycardias (VTs; n = 21), nonsustained VTs

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($n = 25$), or premature ventricular contractions (PVCs; $n = 44$), which were successfully ablated at the aortic root. Echocardiography and exercise stress testing or coronary angiography demonstrated no evidence of structural heart disease in any patients. Baseline patient characteristics, including age, gender, nature of the clinical arrhythmia, and 12-lead ECG during the VAs, were recorded. Each patient gave written informed consent, and all antiarrhythmic drugs were discontinued for at least five half-lives prior to the study.

Electrophysiologic study

For mapping and pacing, a quadripolar catheter was positioned via the right femoral vein at the His bundle (HB) region, and a 6Fr or 7Fr deflectable decapolar catheter was placed in the coronary sinus. The coronary sinus catheter was advanced into the great cardiac vein as far as possible until the proximal electrode pair recorded an earlier ventricular activation than the most distal electrode pair during the VAs. Mapping and pacing were performed using a 7F, 4- or 5-mm-tip nonirrigated ablation catheter or a 7.5Fr, 3.5-mm-tip irrigated ablation catheter introduced from the right femoral vein (for the right ventricular outflow tract [RVOT]) or the right femoral artery (for the aortic root). During procedures at the aortic root, intravenous heparin was administered to maintain an activated clotting time > 250 seconds. When few PVCs were observed at the beginning of the electrophysiologic study, induction of VT or PVCs was attempted by burst pacing from the RVOT or apex with the addition of an isoproterenol infusion.

Mapping and RF catheter ablation

Activation mapping was performed in all cases in order to identify the earliest site of ventricular activation during the VT or PVCs. In some patients, when the VT or PVCs were frequent, electroanatomic mapping was performed as previously reported.^{13,14} Pace-mapping was also performed using the distal bipolar electrodes at a pacing cycle length of 500 ms and at the minimum stimulus amplitude required for consistent capture (up to a maximum output of 20 mA and pulse width of 2.0 ms). The score for the pace-mapping was determined as the number of leads with an identical height of the R wave/depth of the S wave (R/S) ratio match (12 = perfect R/S ratio match in all 12 leads), as well as the number of leads with a fine notching match in the 12-lead ECG as previously reported (perfect pace-mapping = 24 points).¹⁵ An excellent pace-map was defined as a pace-map that obtained a score > 20 .

When the earliest ventricular activation in the right ventricle (RV) preceded QRS onset by > 20 ms and was earlier than that recorded in the great cardiac vein, RF catheter ablation was performed at that site when there was confirmation of an excellent pace-map match to the QRS complex of the clinical VAs. When there were no sites with early activation in the RV or when RV catheter ablation was unsuccessful, mapping in the ASCs and LV outflow tract

followed. Nonirrigated RF current was delivered with a target temperature of 55°C and maximum power output of 30 to 50 W. Irrigated RF current was delivered in the power control mode starting at 20 or 30 W, at an irrigation flow rate of 17 or 30 mL/min. RF power was titrated to as high as 40 W, with the goal being a decrease in impedance of 8 to 10 Ω and care taken to limit the temperature to $\leq 40^{\circ}\text{C}$. Before mapping above the aortic valve, selective angiography of the coronary artery and aorta was performed to carefully determine the coronary artery ostium in the ASCs, to precisely define the location of the ablation catheter and avoid arterial injury.^{4,16} Intracardiac echocardiography was performed to identify the precise location of the ablation catheter in the ASCs if needed. RF ablation was applied in the ASCs under continuous fluoroscopic observation with an angiographic catheter positioned within the ostium of the coronary artery. The outline of the ASCs and flow in the coronary artery were observed by hand injections of contrast every 15 seconds. An RF application was never delivered within 5 mm of the coronary artery. When an acceleration or reduction in the frequency of the VT or PVCs was observed during the first 10 seconds of the application, RF delivery was continued for 30 to 60 seconds. Otherwise, RF delivery was terminated, and the catheter was repositioned. The end-point of catheter ablation was elimination and noninducibility of VT or PVCs during an isoproterenol infusion (2–4 $\mu\text{g}/\text{min}$) and burst pacing from the RV (to a cycle length as short as 300 ms).

ECG analysis

Twelve-lead ECGs during the VAs and pace-mapping were recorded digitally at a sweep speed of 100 to 200 mm/s in all patients for offline analysis. QRS morphologies, including the presence of a bundle branch block pattern, the axis, and the configuration in leads I and aVL, were examined. QRS duration and maximal R-wave amplitude in the inferior leads were measured with electronic calipers by two experienced investigators blinded to the site of the origin. QRS duration was measured as the interval between the earliest deflection of the ventricular complex in any of the 12 simultaneous leads to the latest offset in any lead. The ratio of R-wave amplitude in leads III to II (III/II) was also calculated. Any discrepancies between those results were adjudicated by a third investigator.

Follow-up

Follow-up after the procedure included clinic visits with 12-lead ECGs and 24-hour ambulatory (Holter) monitoring, and telephone calls to all patients and their referring physicians. All patients who reported symptoms were given a 24-hour Holter monitor or event monitor to document the cause of symptoms. Successful catheter ablation was defined as no recurrence of any VAs during > 6 months of follow-up.

Statistical analysis

Continuous variables are expressed as group mean \pm 1 SD. Comparisons of continuous variables between the two groups were analyzed using the Student *t* test. When comparisons involved >2 groups, analysis of variance (ANOVA) was used. When group differences were found, a one-way ANOVA was followed by the Fisher least significant difference method to test the significance of the difference among the means in all groups. Categorical variables expressed as numbers and percentages in the different groups were compared by χ^2 test and Yates correction if necessary. An overall Fisher exact test for a $2 \times n$ table was constructed when comparisons involved >2 groups. $P < .05$ was considered significant.

Results

VAs originating from the NCC

Results of the clinical, ECG, and electrophysiologic parameters of the NCC VAs are summarized in [Tables 1](#) and [2](#). Successful ablation was achieved within the NCC in six patients (four men; mean age 23 ± 9 years, range 13–36 years) with symptomatic idiopathic sustained VT ($n = 2$) or nonsustained VT ($n = 4$). These patients never had a recurrence of NCC VAs during mean follow-up of 44 ± 26 months (range 8–89 months). One of these patients experienced presyncope with exercise. Nonirrigation and irrigation catheters were used in five patients and one patient (case 6), respectively. QRS duration of these VAs was <150 ms in all but one case (case 6, 202 ms; [Figure 1](#)). QRS morphologies of these VAs were characterized by a left bundle branch block and left inferior ($n = 5$) or superior ($n = 1$) axis, upright R waves in lead I in all cases, upright R waves dominantly in lead aVL, and frequent presence of S waves in lead III ($n = 3$). The III/II ratio was <0.65 in all but one case (case 6, 0.765). In one of these cases, activation mapping was not available because of infrequent PVCs, and only pace-mapping was used for identifying the site of ablation (case 2). Pacing within the NCC did capture the ventricle in this case, whereas it captured the atrium in the remaining cases. During the NCC VAs, local ventricular activation with a far-field electrogram in the HB region always preceded QRS onset (-15 to -43 ms; [Figure 2](#)), and three-dimensional activation maps revealed a relatively wide area with early activation around the HB region ([Figure 3](#)). Catheter ablation was unsuccessful in the RV despite an excellent pace-map in three cases (cases 3, 4, and 6; [Figure 3](#)). In these cases, RF applications in the RV resulted in suppression of the VAs, a slight change in QRS morphology with prolongation of QRS duration, and attenuation and delay of the high-amplitude near-field ventricular electrogram in the RV HB region ([Figure 2](#)). A low-amplitude far-field ventricular electrogram preceding QRS onset was then separated from the near-field ventricular electrogram following QRS onset in the RV HB region. Local ventricular activation at the successful ablation site within the NCC was always earlier than that in the RV HB

region. The atrial and ventricular electrogram amplitude ratio (A/V) at the successful ablation site within the NCC was >1 in all but one case (case 6). No HB electrograms were recorded at the successful ablation sites. Selective angiography of the coronary artery and aorta clearly identified the site of successful ablation within the NCC in five cases ([Figure 4](#)). However, intracardiac echocardiography was performed in one case because the successful ablation site was located within the NCC at the junction with the right coronary cusp (RCC; [Figure 5](#)).

Comparison of NCC VAs with VAs originating from other aortic root locations ([Tables 1](#) and [2](#)).

The VA origin was determined to be in the left coronary cusp (LCC) in 33 (36.7%) patients, RCC in 32 (35.6%), junction of LCC and RCC (L-RCC) in 19 (21.1%), and NCC in 6 (6.7%). Patients with NCC VAs were significantly younger than those with any other aortic root VAs ($P < .0001$). All the NCC VAs exhibited VTs, whereas PVCs were dominant in the other VAs. There were no significant differences in gender among patients with aortic root VAs, and men were more prevalent than women in each of the aortic root VAs.

QRS duration during the NCC VAs was significantly shorter than that during LCC and L-RCC VAs ($P < .05$), but there were no significant differences in QRS duration between NCC and RCC VAs. Some of the LCC, RCC, and L-RCC VAs exhibited a right bundle branch block pattern and/or right-axis deviation, whereas none of the NCC VAs did. The precordial transition zone was most frequently observed between leads V_2 and V_3 in all of the aortic root VAs. In the QRS morphology of lead I, an R wave was dominant in RCC and NCC VAs, whereas an rS pattern was dominant in LCC VAs. In lead aVL, large S waves were observed in all of the LCC and L-RCC VAs and most of the RCC VAs, whereas those were very rare in NCC VAs. The amplitude of the R waves in the inferior leads was significantly smaller during NCC VAs than that during LCC and L-RCC VAs ($P < .001$), but there were no significant differences in amplitude between NCC and RCC VAs. The R-wave amplitude ratio in leads III to II (III/II) was significantly smaller during NCC VAs than that during any other aortic root VAs ($P < .0001$). Comparing aortic root VA origins, QRS duration <150 ms and III/II ratio <0.65 predicted an NCC origin with sensitivity of 83% and 83%, specificity of 86% and 89%, positive predictive accuracy of 29% and 36%, and negative predictive accuracy of 99% and 99%, respectively.

There were no significant differences in electrophysiologic parameters such as local ventricular activation time relative to QRS onset or amplitude of ventricular electrograms at the successful ablation sites, or duration or number of RF applications needed for successful ablation among the sites of the VA origin in the aortic root. Local ventricular activation time relative to QRS onset at the RV HB region was significantly earlier for the VAs with NCC origins than for those with LCC, RCC, and L-RCC origins ($P < .0001$). Comparing the aortic root VA origins, local ventricular

Table 1 Clinical and ECG characteristics

Origin	Age (Years)	Gender (M/F)	Type (NSVT/SVT/PVC)	QRS						
				Duration (ms)	Morph.	Transition	Lead I	Lead aVL	R amp (mV) in inferior leads	III/II ratio
NCC (n = 6)	23 ± 9*	4/2	4/2/0	155 ± 23 [†]	LL 6	V1-2;1 V2-3;3 V3-4;1 V4;1	R;6	R;4 rS;1 rsr;1	1.1 ± 0.4 [#]	0.4 ± 0.3*
LCC (n = 33)	50 ± 15*	17/16	7/3/23	173 ± 19	LL 8 LR 13 RL 1 RR 11	<V1;8 V1-2;1 V2;1 V2-3;16 V3;5 V3-4;1 V4-5;1	rS;22 R;2 rsr';2 rSr';5 QS;2	QS;21 rS;12	1.9 ± 0.6 [#]	1.1 ± 0.1*
RCC (n = 32)	57 ± 15*	21/6	6/5/8	163 ± 17	LL 13 LR 5 RL 2	V1;1 V1-2;4 V2;5 V2-3;15 V3;2 V3-4;3 V4;1 V4-5;1	R;22 rS;4 Rr';3 rsr';3	rS;17 QS;9 rsr';3 R;2 qr;1	1.4 ± 0.5	0.8 ± 0.3*
L-RCC (n = 19)	51 ± 15*	13/6	6/5/8	173 ± 19 [†]	LL 13 LR 6	V1-2;7 V2;2 V2-3;7 V3;3	rS;10 R;7 rsr';1 qrS;1	QS;10 rS;9	1.8 ± 0.5 [#]	1.0 ± 0.2*

Amp = amplitude; F = female; inf. = inferior; LCC = left coronary cusp; LL = left bundle block (LBBB) + left inferior axis; L-RCC = the junction of LCC and right coronary cusp (RCC); LR = LBBB + right inferior axis; M = male; NCC = noncoronary cusp; NSVT = nonsustained ventricular tachycardia (VT); PVC = premature ventricular contraction; RR = right bundle branch block + right inferior axis; SVT = sustained VT.

*P < .0001 vs each of the other sites.

[†]P < .05 vs each of the other sites.

[#]P < .0001 vs each of the other sites.

Table 2 Electrophysiologic characteristics

Origin	V-QRS (ms)		ABL site			RF duration (min)	No. of RF lesions
	ABL site	HB region	A (mV)	V (mV)	A/V		
NCC (n = 6)	-35 ± 7	-30 ± 12 [#]	0.34 ± 0.22 [#]	0.17 ± 0.06	2.10 ± 1.21 [#]	1.9 ± 0.9	1.7 ± 0.8
LCC (n = 33)	-26 ± 9	37 ± 13 [#]	0.08 ± 0.12 [#]	0.60 ± 0.60	0.22 ± 0.25 [#]	2.0 ± 1.0	2.1 ± 1.0
RCC (n = 32)	-31 ± 13	-10 ± 8 [#]	0.03 ± 0.06 [#]	0.42 ± 0.28	0.07 ± 0.15 [#]	2.2 ± 0.9	2.0 ± 0.8
L-RCC (n = 19)	-29 ± 8	27 ± 10 [#]	0.05 ± 0.05 [#]	0.59 ± 0.59	0.12 ± 0.16 [#]	2.4 ± 1.0	2.3 ± 1.2

A = atrial electrogram; ABL = ablation; A/V = the amplitude ratio of atrial and ventricular electrograms; HB = His bundle; RF = radiofrequency; V = ventricular electrogram; V-QRS = the local ventricular activation time relative to QRS onset.

[#]P < .0001 vs each of the other sites. Other abbreviations as in previous table.

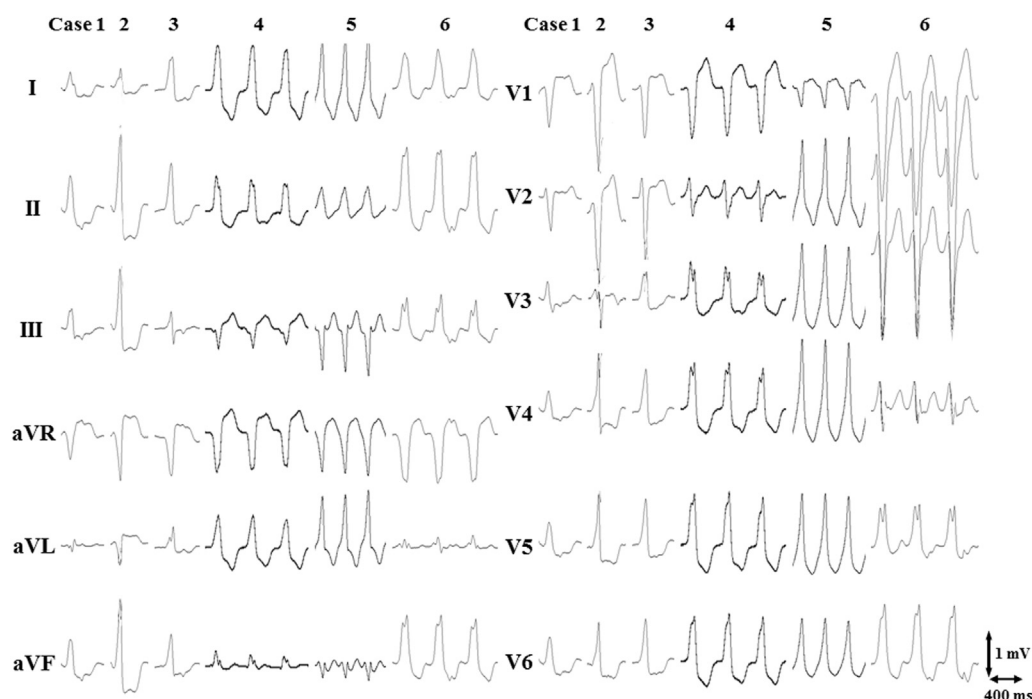


Figure 1 Twelve-lead ECGs of ventricular arrhythmias originating from the noncoronary cusp of the aorta. Note that S waves in lead III were recorded in three cases.

activation time relative to QRS onset at the RV HB region < -25 ms predicted an NCC origin with sensitivity of 83%, specificity of 98%, positive predictive accuracy of 71%, and

negative predictive accuracy of 99%. The amplitude of the atrial electrograms and amplitude ratio in the atrial and ventricular electrograms at the successful ablation sites were significantly greater for NCC VA origins than for any other aortic root VA origin ($P < .0001$).

Complications

Transient sinus bradycardia followed by complete AV conduction block occurred in one patient with PVCs with an RCC origin. Both sinus node function and AV conduction recovered soon after termination of RF energy delivery in this patient. No other complications occurred.

Discussion

Idiopathic NCC VAs were very rare (7% of the idiopathic aortic root VAs) and always occurred in young patients. NCC VAs always exhibited VT, whereas PVCs were dominant in the other aortic root VAs. These clinical characteristics of NCC VAs were consistent with previous isolated case reports.^{8–10}

Considering the anatomic configuration of the NCC, the ECG and electrophysiologic characteristics of NCC VAs observed in this study seemed to be reasonable. Anatomically, among the ASCs, the NCC is most inferiorly, posteriorly, and medially located.¹⁶ The RCC is located anterior to the NCC and attached to the interventricular septum; thus, the ECG and electrophysiologic characteristics of NCC VAs were similar to those of RCC VAs. Nevertheless, the QRS morphologies of NCC VAs were characterized by a left bundle branch block and left-axis QRS morphology and upright R waves in lead I in all cases, upright R waves dominantly in lead aVL, and frequent presence of S waves in lead III (50%). QRS duration of NCC

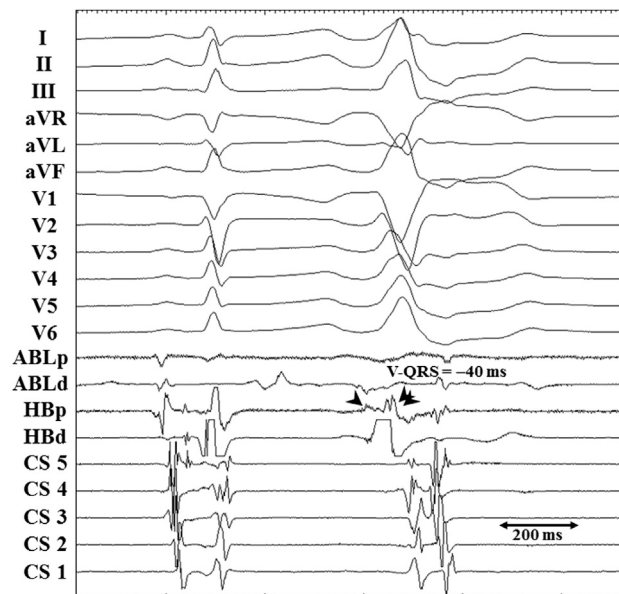


Figure 2 Cardiac tracings exhibiting the successful ablation site within the noncoronary cusp (NCC) in case 6. The first beat is a sinus, and the second beat is a premature ventricular contraction (PVC). During the PVCs, a far-field electrogram (single arrowhead) followed by a near-field electrogram (double arrowheads) preceded QRS onset in the right ventricular His bundle (HB) region. Local ventricular activation at the successful ablation site within the NCC was earlier than that in the right ventricular HB region. At the successful ablation site, the amplitude of the ventricular electrogram was slightly larger than that of the atrial electrogram. ABL = ablation; CS = coronary sinus; CS 1 to 5 = first (most distal) to fifth (most proximal) electrode pairs of the CS catheter; V-QRS = local ventricular activation time relative to QRS onset; d, p = distal and proximal electrode pairs of the relevant catheter, respectively.

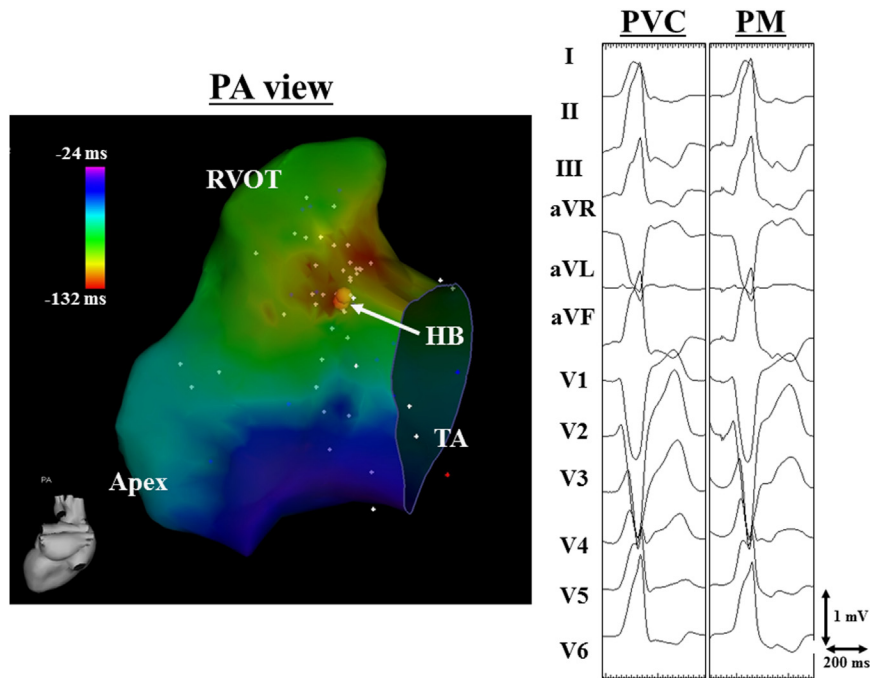


Figure 3 Activation map obtained in the right ventricle during noncoronary cusp ventricular arrhythmias (**left**) and an excellent pace-map (PM) obtained in the right ventricle (**right**) in case 6. Note that the activation map revealed a relatively wide area with early activation around the His-bundle (HB) region. PA = posteroanterior; PVC = premature ventricular contraction; RVOT = right ventricular outflow tract; TA = tricuspid annulus.

VAs was typically narrower (<150 ms), and III/II ratio typically was <0.65 . These ECG characteristics predicted an NCC origin among the aortic root VAs sites with a high accuracy.

The most inferior portions of the NCC and RCC are in contact with the membranous portion of the interventricular septum, where it comes in close apposition to the penetrating HB (Figure 6).¹⁶ Because of this close proximity, a catheter recording HB activation across the tricuspid annulus typically recorded an early ventricular activation during NCC and RCC VAs. Although the local ventricular activation time relative to QRS onset at the RV HB region was also early during RCC VAs, if the activation time was <-25 ms an NCC origin was predicted with high accuracy. For most of its anatomic course, and slightly more superiorly, the NCC is attached to the interatrial septum.¹⁶ It is well known that

atrial tachycardias arising from the roof of the interatrial septum can be ablated only within the NCC.^{8–10} Therefore, a catheter positioned in the NCC usually records a large atrial electrogram and a much smaller ventricular electrogram. In this study, the amplitude ratio in the atrial and ventricular electrograms at the successful ablation sites of the NCC VAs typically was >1 .

Although the proximity of the NCC to the atrial septum has been well appreciated, the relation of this structure to the ventricular myocardium has been less discussed. Anatomically, the NCC is attached to the anterior leaflet of the mitral valve from its most leftward extent in continuity with the LCC commissure. This contact of the NCC with the anterior leaflet of the mitral valve extends approximately 70% of its width until it makes contact with the interventricular septum near its junction with the RCC (Figure 6A). Because the

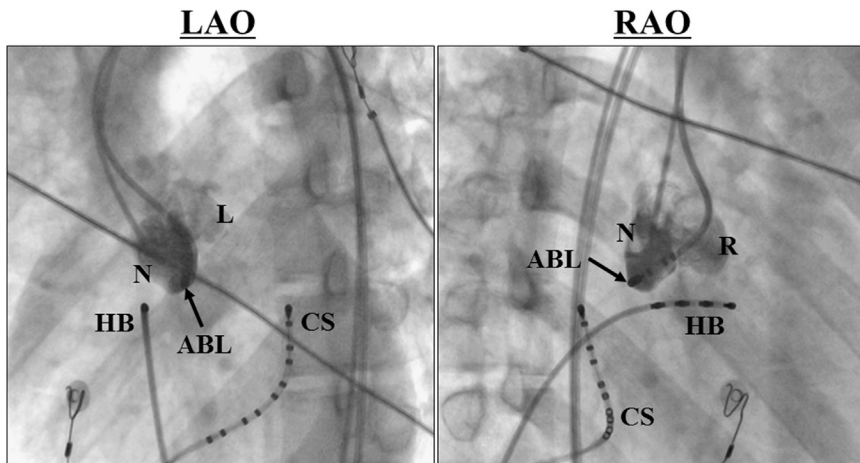
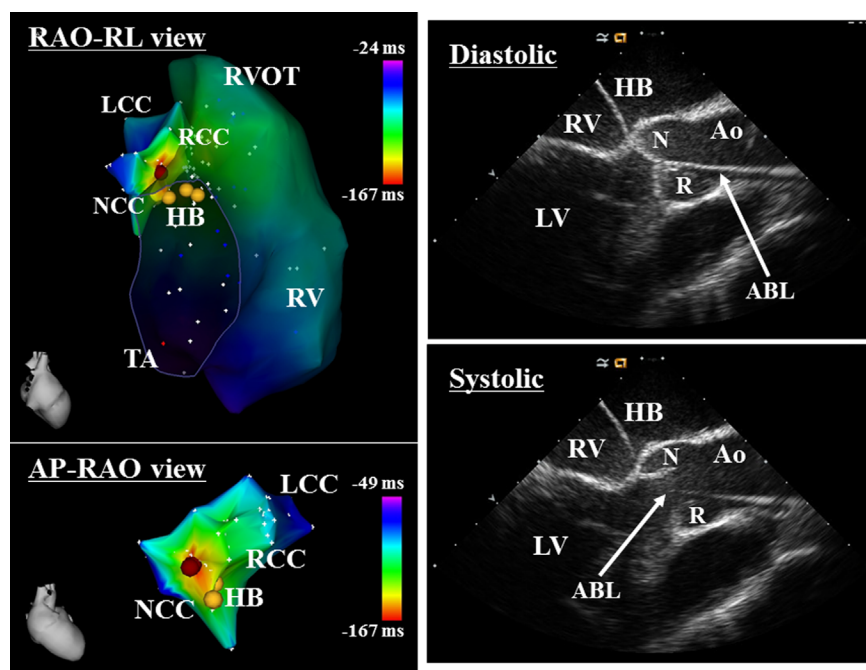


Figure 4 Fluoroscopic images showing the successful ablation site within the noncoronary cusp in case 2. Aortography assured that the ablation catheter was positioned within the noncoronary cusp. L = left coronary cusp; LAO = left anterior oblique view; N = noncoronary cusp; R = right coronary cusp; RAO = right anterior oblique view. Other abbreviations as in previous figures.

Figure 5 Activation maps (left panels) and intra-cardiac echocardiograms (right panels) showing the successful ablation site within the noncoronary cusp (NCC) at the junction with the right coronary cusp (RCC) in case 6. Red and yellow tags indicate the successful ablation site and the sites where HB electrograms were recorded. Ao = aorta; AP = anteroposterior; LCC = left coronary cusp; LV = left ventricle; RL = right lateral; RV = right ventricle. Other abbreviations as in previous figures.



anterior leaflet of the mitral valve is attached to the posterior portions of the NCC and LCC including their junction (intervalvular trigone), it seems that there is little likelihood that NCC VAs could arise from the posterior portion of the NCC. The membranous septum is located anterior to the

anterior mitral leaflet and underneath the RCC and NCC. Therefore, the membranous septum and anterior mitral leaflet completely separate the NCC from the LV myocardium. In addition, the RV myocardium is also very close to the NCC (Figure 6B). These findings might suggest that

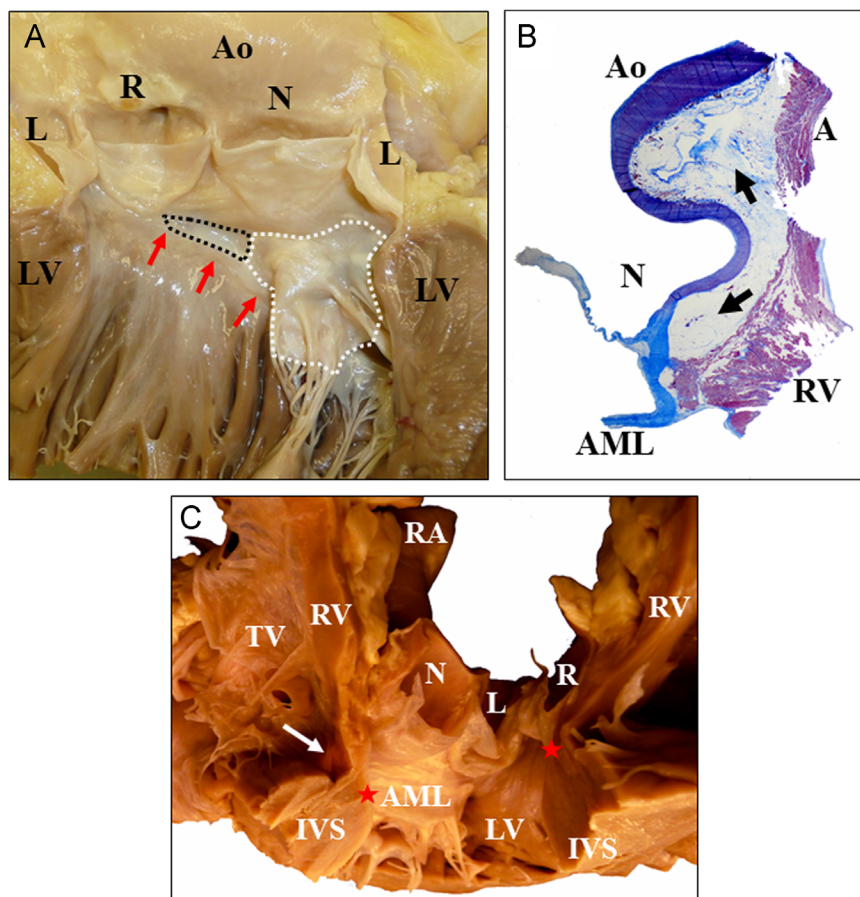


Figure 6 Autopsy hearts showing anatomic relationships between the noncoronary cusp (NCC) and its adjacent structure (A, C) and trichrome staining of histologic sections through the midpoint of the NCC (B; magnification 1×). **A:** Anatomically, the anterior leaflet of the mitral valve (AML) is attached to the NCC from the left coronary cusp commissure to a site between approximately 70% of its distance toward its junction with the right coronary cusp. The NCC is not attached to the left ventricular myocardium because the membranous septum (black dotted circle) and the anterior leaflet of the mitral valve (white dotted circle) separate the NCC from the left ventricular myocardium. Red arrows indicate superior end of left ventricular myocardium. **B:** Right ventricular myocardium is attached to base of the NCC and anterior mitral leaflet. Adipose tissue (black arrows) separates the aortic wall from right atrial and ventricular myocardium. **C:** White arrow indicates membranous septum. Red stars indicate superior end of left ventricular septum. The left ventricular septum is attached to the right coronary cusp, whereas it is not to the NCC. The NCC is typically attached to the interventricular septum (IVS) closer to the right ventricular endocardium than the left ventricular endocardium. A = atrial myocardium; RA = right atrium; TV = tricuspid valve; V = ventricular myocardium. Other abbreviations as in previous figures.

NCC VAs arise from the RV rather than the LV. The clinical observation that a noncoronary sinus of Valsalva aneurysm can rupture into the RV as well as the right atrium supports this possibility.^{8–10} However, the findings that RV electrograms near the HB were far-field and that ablation in the peri-HB region was not effective for these VAs suggest that the origin is not in the RV. Rather, VAs that can be ablated from within the NCC VAs likely arise from the interventricular septum rather than the RV myocardium. The junction between the RCC and NCC typically is attached to the interventricular septum (Figure 6C), although there could be minor anatomic variations among individuals. Therefore, it might be appropriate to conclude that NCC VAs arise from the interventricular septum closer to the RV endocardium than the LV endocardium.

During NCC VAs, far-field early ventricular electrograms reflecting activity of VA origin usually were recorded at the RV HB region. These early far-field ventricular electrograms could be easily recognized when they were separated from the later near-field electrograms reflecting activity of the RV HB region. When conduction from the NCC VA origin to the RV endocardium was rapid, far-field early ventricular electrograms might be missed because they overlapped with near-field early ventricular electrograms. In such cases, when RF ablation eliminated the RV breakout sites, delaying the near-field ventricular activity in the HB region, the far-field early ventricular electrograms were separated from the near-field ventricular electrograms. When these electrophysiologic findings are obtained in the RV HB region, mapping in the NCC as well as the RCC should be added to accurately identify the site of origin.

Study limitations

In this study, the value of statistical analysis in such a small case series may be questioned. The significance of the observed findings will need to be revisited in a larger number of patients in the future.

Conclusion

This study revealed that among the aortic root VAs, NCC VAs were very rare (7%) and occurred in significantly younger patients. In a limited set of six patients, the ECG and electrophysiologic characteristics of NCC VAs were similar to those of RCC VAs but were characterized by a narrower QRS duration (typically <150 ms), smaller III/II ratio (typically <0.65), earlier ventricular activation in the HB

region (typically < –25 ms), and A/V ratio > 1 at the successful ablation site. The findings in this study will be helpful for improving the outcome of catheter ablation of idiopathic VAs originating from the aortic root and HB region.

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