Ventricular premature complexes successfully ablated from the non-coronary cusp: a case report

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Background	Ventricular premature complexes (VPCs) occasionally originate from the aortic sinus of Valsalva. Because the anterior part of the left coronary cusp (LCC) and right coronary cusp (RCC) are connected through the ventricular musculature at their bases, VPCs are more common in the LCC and the RCC than in the non-coronary cusp (NCC). We herein report a case in which VPCs were successfully ablated from the NCC, which is considered rare.
Case summary	A 30-year-old woman was admitted to our hospital for the ablation of VPCs, which comprised 43% of the total heart beats. The clinical VPCs had an inferior axis and left bundle branch block morphology with a precordial transition between V4 and V5. Three-dimensional mapping of the target VPCs indicated that the earliest activation site was RCC. After radiofrequency (RF) energy application at the RCC, VPCs were temporally suppressed but recurred after 24 min. Remapping of the recurrent VPCs revealed that the earliest activation site shifted from the RCC to the His region. To avoid the risk of atrioventricular block, RF energy was applied from the NCC, which resulted in successful elimination of the VPCs without any complications.
Discussion	The present case suggests that RF energy application from the NCC may be a safe and effective option for the ablation of VPCs with the earliest activation at the RCC and His region.
Keywords	Ventricular premature complexes • Non-coronary cusp • Right coronary cusp • His region • Radiofrequency catheter ablation • Case report
ESC Curriculum	5.1 Palpitations • 5.6 Ventricular arrhythmia

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Learning points

- Idiopathic ventricular arrhythmias (VAs) from the aortic sinus of Valsalva are more common in the left coronary cusp and the right coronary cusp (RCC), and rarely arise from the non-coronary cusp (NCC).
- Electrocardiography and the electrophysiological characteristics of NCC VAs were similar to those of RCC VAs.
- Radiofrequency energy application from the NCC may be a safe and effective option for the ablation of ventricular premature complexes with the earliest activation at the RCC and His region.

Timeline

1 year before	Palpitations and fatigue appeared
	Premature ventricular complexes (VPCs) were diagnosed
3 months before	Palpitations and fatigue worsened
	Holter ECG—VPCs comprised 43% of the total heart beats
	Bisoprolol 2.5 mg daily was started
1 month before Drug treatment for 2 months	Holter ECG—VPCs comprised 44% of the total heart beats
	Her symptoms were not resolved
Day of ablation	Ablation of VPCs was performed as follows:
	9:05 Femoral vein puncture
	9:32 RVOT mapping
	9:48 Femoral artery puncture
	9:53 Aortic cusps mapping
	10:21 VPCs elimination after RCC ablation
	10:45 VPCs recurrence
	10:52 RVOT and aortic cusps remapping
	11:30 VPCs elimination after NCC ablation
	11:53 Ablation was finished
3 days later	Discharged without any complications
1 month later Post-ablation follow-up	Holter ECG—no recurrence of the VPCs
	Palpitations and fatigue disappeared
1 year later Post-ablation follow-up	Palpitations and fatigue disappeared

Introduction

Approximately 70% of idiopathic ventricular arrhythmias (VAs) originate from the right ventricular outflow tract (RVOT).[1] Other origins of idiopathic VAs include the aortic sinus of Valsalva, the left ventricular outflow tract (LVOT), great cardiac veins, epicardial myocardium, aorta-mitral continuity, and rarely the pulmonary artery. Right ventricular outflow tract VAs typically present between the ages of 20 and 50 years and more frequently occur in women.[1] Recent progress of three-dimensional mapping technology has increased the success rates of VAs ablation remarkably. The success

rates of RVOT VAs catheter ablation are reported to be >95% in patients without structural heart disease when performed by experienced operators.[1] Therefore, according to the current European Society of Cardiology (ESC) guidelines,[1] catheter ablation of RVOT VAs is ranked as Class I and recommended in symptomatic patients and/or in patients with a failure of antiarrhythmic drug therapy or in patients with a decline in their left ventricular function due to the RVOT VAs burden.

Ventricular arrhythmias originating within the aortic sinus of Valsalva account for ∼20% of idiopathic VAs, most from the left coronary cusp (LCC), followed by the right coronary cusp (RCC), and rarely the fibrous non-coronary cusp (NCC).[1] The main complication from ablation within the aortic cusps is the acute occlusion of the left main coronary artery. Therefore, the current ESC guidelines[1] rank catheter ablation of aortic cusp VAs as Class IIa and it should be performed by experienced operators in symptomatic patients after the failure of one or more sodium channel blockers (Class IC agents).

Because aortic cusp VAs from the NCC are considered to be rare, we herein report a case of idiopathic ventricular premature complexes (VPCs) that were successfully ablated from the NCC without any complications.

Case presentation

A 30-year-old woman visited our hospital with a chief compliant of palpitations and fatigue. No significant abnormal findings were found on physical examination. Electrocardiography (ECG) revealed that the rhythm was sinus with frequent VPCs. Holter ECG demonstrated that the VPCs comprised 43% of the total heart beats. Echocardiography indicated no evidence of clinically overt structural or organic heart disease. Because she had no particular medical history, bisoprolol 2.5 mg daily was started. However, since bisoprolol did not either alleviate her symptoms or reduce the frequency of VPCs, the drug was discontinued and radiofrequency (RF) catheter ablation was instead recommended. The clinical VPCs had an inferior axis and left bundle branch block morphology with a precordial transition between V4 and V5 (*Figure 1A*).

Radiofrequency catheter ablation was performed in accordance with our protocol for VPC ablation (*Figure 2*). In brief, under local anaesthesia, a 20-polar electrode monorail catheter (Inter-Nova, Tokyo, Japan) was positioned percutaneously through the coronary sinus (CS) into the anterior interventricular vein (AIV) using a 0.014 guidewire. Since the surface ECG morphology suggested that the clinical VPCs were of an RVOT origin, the VPC mapping was first obtained at the RVOT using a THERMOCOOL SMARTTOUCH®
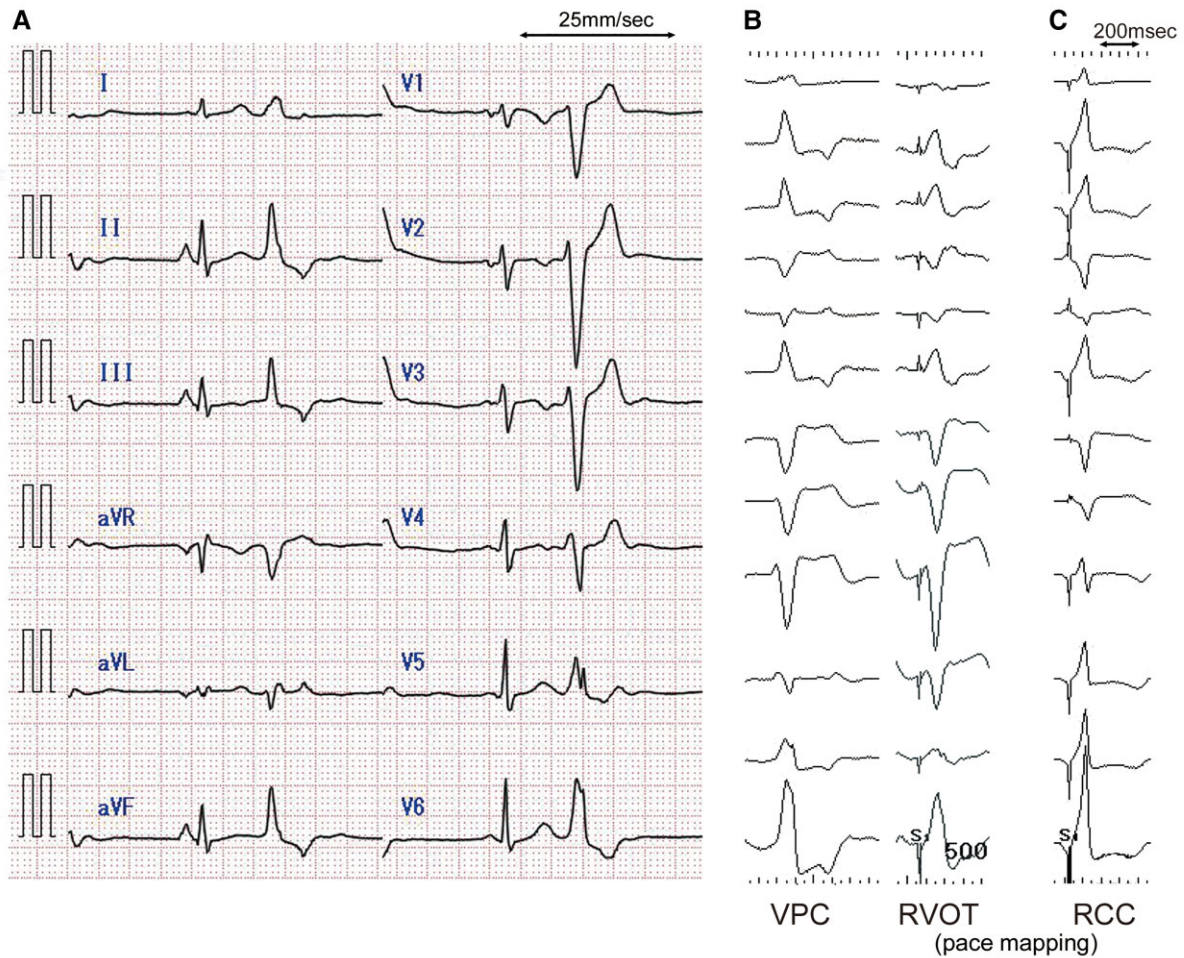
Figure 1 (*A*) Twelve-lead electrocardiography of the ventricular premature complexes. Clinical ventricular premature complexes had an inferior axis and left bundle branch block morphology with a precordial transition between V4 and V5. The QRS morphology has the left bundle ranch block, left axis, and upright R waves in Lead I with the QRS duration of 128 ms and III/II ratio of 0.62. (*B*) Pace mapping at the right ventricular outflow tract septum. No good pace mapping was obtained at the right ventricular outflow tract (8 of 12 leads were identical to the clinical ventricular premature complexes). (*C*) Pace mapping at the right coronary cusp. Pacing at the earliest activation site was similar to target ventricular premature complexes, but a perfect pace map was not obtained (10 of 12 leads were identical to the clinical ventricular premature complexes). RCC, right coronary cusp; RVOT, right ventricular outflow tract; VPCs, ventricular premature complexes.

ablation catheter (Biosense Webster, Irvine, CA, USA). We prefer to use the ablation catheter for RVOT VA mapping because of its manoeuvrability and lower incidence of catheter-induced VPCs compared with multipolar catheters. The earliest activation was recorded at the septal side of the RVOT and preceded surface ECG by 11 ms. However, no good pace mapping was obtained at the RVOT (8 of 12 leads were identical to the clinical VPCs, *Figure 1B*). Therefore, the VPC mapping was performed at the LVOT using a multi-electrode catheter PENTARAY® (Biosense Webster) after aortic angiography (*Figure 3A*). The geometry of the aortic cusp was reconstructed by CARTOSOUND® (Biosense Webster) (*Figure 4A*). Three-dimensional mapping by the CARTO 3 system (Biosense Webster) demonstrated that the earliest activation was at the RCC and preceded surface ECG by 28 ms (*Figures 2, 4B*, and *5A*). Pacing at the earliest

activation site provoked a waveform similar to the target VPCs, but no perfect pace mapping was obtained (10 of 12 leads were identical to the clinical VPCs, *Figure 1C*). Radiofrequency energy was applied at the earliest site of the RCC with a maximal temperature of 42°C, a maximum power of 35 W, and an irrigation rate of 12 mL/min for 120 s. The VPCs were suppressed in 3 s but recurred after the cessation of RF energy application. After the third energy application, the VPCs were finally eliminated.

However, the VPCs recurred 24 min after the last energy application. Although the morphology, QRS transition, and rate of the recurrent VPCs were almost the same as the target VPCs, additional ablation from the RCC did not diminish the recurrent VPCs. Remapping of the recurrent VPCs using CARTO3 system indicated that the earliest activation site was not at the RCC but below the
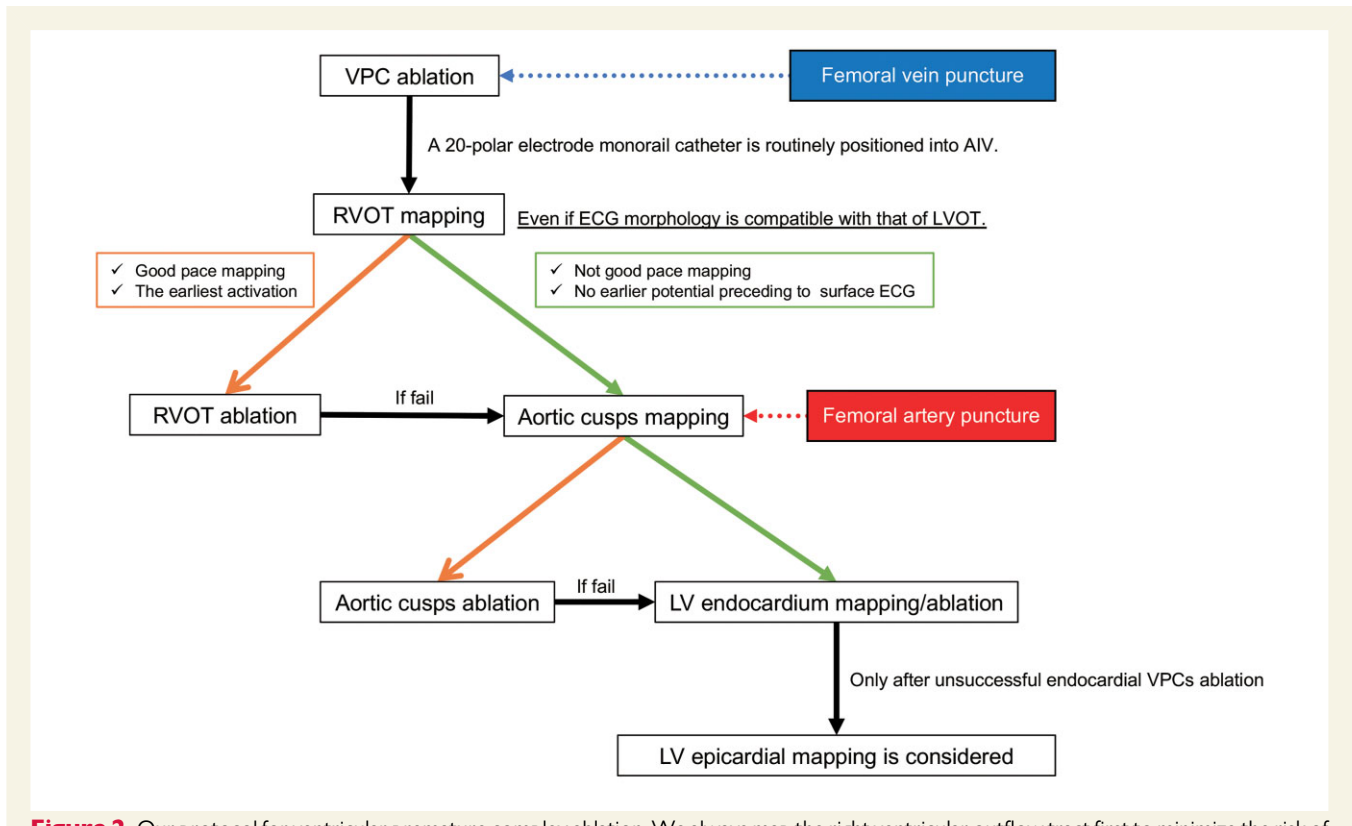
Figure 2 Our protocol for ventricular premature complex ablation. We always map the right ventricular outflow tract first to minimize the risk of complications, even if the electrocardiography morphology is compatible with that of the left ventricular outflow tract. If no good pace mapping or earlier potential preceding to surface electrocardiography at the right ventricular outflow tract is obtained, then we map the aortic cusps and the left ventricular outflow tract. A 20-polar electrode monorail catheter is routinely positioned into the anterior interventricular vein for monitoring the great cardiac vein and anterior interventricular vein. Only after unsuccessful left ventricle endocardial ventricular premature complex ablation, left ventricle epicardial mapping may thus be considered. AIV, anterior interventricular vein; ECG, electrocardiograms; GCV, great cardiac vein; LV, left ventricle; LVOT, left ventricular outflow tract; RVOT, right ventricular outflow tract; VPCs, ventricular premature complexes.

aortic valve (*Figure 3B*), where the His potential was recorded during sinus rhythm. The earliest activation preceded surface ECG by 16 ms. Mapping of the RVOT revealed that the earliest activation site was in the His region (*Figure 3C*), where local potentials preceded surface ECG by 34 ms during VPCs, and a clear His potential was recorded during sinus rhythm (*Figure 5B*). To avoid the risk of atrioventricular (AV) block, we preferred to apply energy from the aortic valve cusps. Because RF energy application at the RCC had failed to diminish the VPCs, RF energy was delivered from the NCC where the His potentials were neither sharp nor split (*Figure 3D*). Because far-field His potentials were recorded at the NCC, CS pacing (120 b.p.m.) was continued during RF energy application to monitor AV conduction. Although the local potentials at the NCC preceded surface ECG by only 22 ms (*Figure 5C*), RF energy delivery from the NCC successfully eliminated the VPCs within 5 s. Energy application for 120 s was repeated several times to secure the elimination of the VPCs. The session was finished without any complications. The total procedure time was 2 h 48 min.

Echocardiography after ablation indicated no evidence of either aortic valve injury or regurgitation. Holter ECG at 1 month after catheter ablation revealed no recurrence of the VPCs. The patient never complained of either palpitations or fatigue after undergoing catheter ablation.

Discussion

Idiopathic VAs often originate from the RVOT and sometimes from the aortic sinus of Valsalva.[1] Idiopathic VAs from the aortic sinus are more common in the LCC than in the RCC and rarely arise from the NCC.[2–5] Because the anterior part of the LCC and RCC is connected with the ventricular musculature at their bases,[6,7] it has been recognized that some VAs were curatively treated by RF catheter ablation from the LCC and RCC. In contrast, the NCC is rarely a target of VAs, because the NCC is not in direct contact with the ventricular musculature and is exclusively surrounded by fibrous walls.[2,7,8] Yamada *et al.*[4] reported that NCC VAs were rare (7%) among 90 cases of idiopathic aortic root VAs and occurred in significantly younger patients in comparison to patients with other aortic root VAs.

Previous studies reported that an early QRS transition (V1–2) predicts VAs with an origin in the sub-valvular LVOT or aortic sinus cusps, while a late QRS transition (V4–5) predicts those with an origin in the RVOT or His region.[3,5,6,9] Since the NCC is located posterior to the RCC which is attached to the interventricular septum, ECG and the electrophysiological characteristics of NCC VAs were similar to those of RCC VAs.[2,9] However, Yamada *et al.*[4] reported that among six cases with NCC VAs, the QRS morphologies
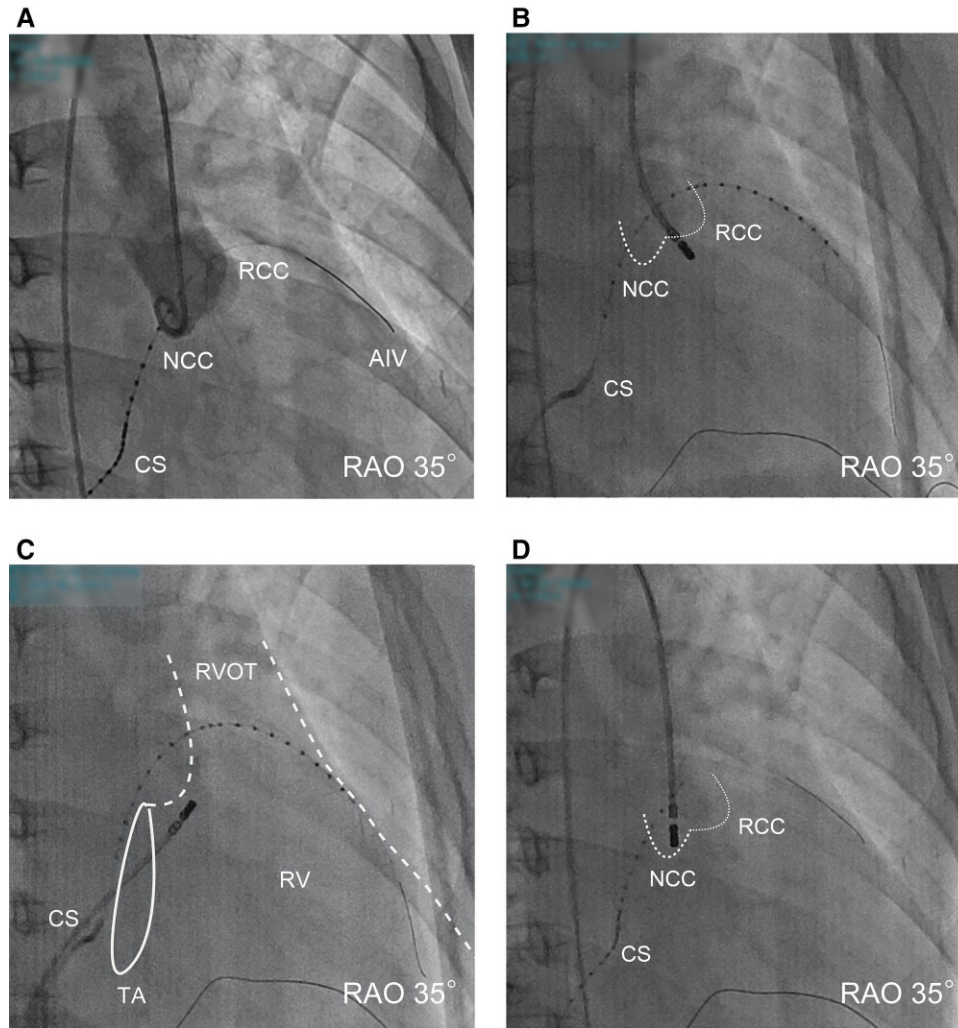
Figure 3 (*A*) Angiography of the left ventricular outflow tract in the right anterior oblique view. The 20-polar electrode catheter was positioned percutaneously through the coronary sinus into interventricular vein. (*B*) Fluoroscopic image of the earliest activation site of recurrent ventricular premature complexes in the left ventricular outflow tract in the right anterior oblique view. The ablation catheter was located at the right coronary cusp. The diagnostic catheter was placed in the coronary sinus-interventricular vein. (*C*) Fluoroscopic image of the earliest activation site of recurrent ventricular premature complexes in the right ventricular outflow tract in the right anterior oblique view. The ablation catheter was in the His region. (*D*) Fluoroscopic image of the successful ablation site in the right anterior oblique view. Energy application to the non-coronary cusp successfully eliminated the ventricular premature complexes. AIV, anterior interventricular vein; CS, coronary sinus; LVOT, left ventricular outflow tract; NCC, non-coronary cusp; RCC, right coronary cusp; RV, right ventricular; RVOT, right ventricular outflow tract; TA, tricuspid annulus; VPCs, ventricular premature complexes.

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 the frequent presence of S waves in Lead III (50%). The QRS duration of NCC VAs was typically narrower (<150 ms), and the III/II ratio typically was <0.65. The A/V ratio was >1 at the successful ablation site. These ECG characteristics may predict an NCC origin among aortic root VAs sites with high accuracy. In the present case, the QRS morphology was a left bundle branch block and upright R waves in Lead I and a small R wave in Lead aVL. The QRS duration was 128 ms (<150). The III/II ratio was 0.62 (<0.65). The A/V ratio at the successful ablation site was >1. These findings were consistent with the QRS

morphology of the NCC VAs reported by Yamada et al.[4] except left-axis QRS morphology and S waves in Lead III.

Previous studies reported that in all VA patients with an RCC or NCC origin, the earliest RV activation preceding the QRS onset was recorded in the His bundle region, and some VAs with the earliest ventricular activation site at the His bundle region were successfully ablated from the NCC.[3,5,6] In the present case, energy application from the NCC resulted in the successful elimination of the VPCs, while the first energy application from the RCC might have blocked the exit of the VPCs towards the RCC without eliminating the VPCs. Therefore, we think that the origin of the VPCs in the present case may have been located at the NCC or the NCC–RCC commissure.
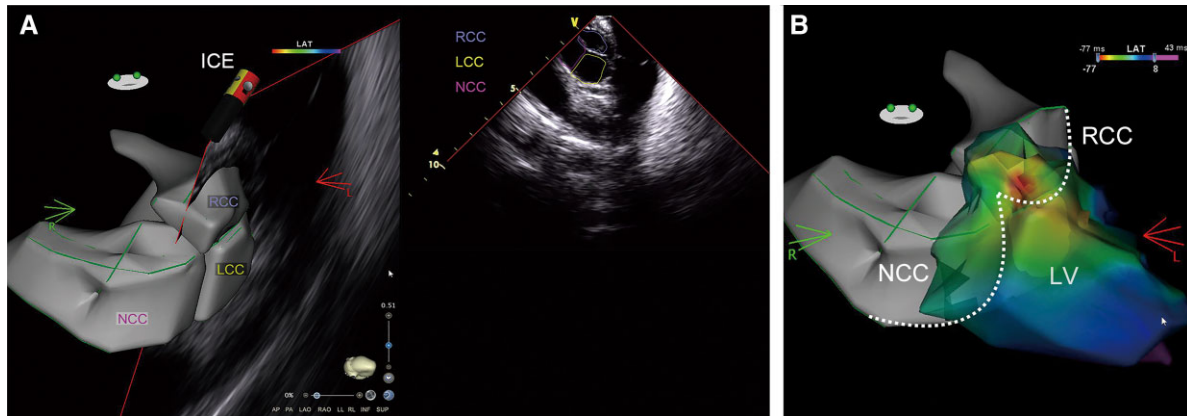
Figure 4 (*A*) CARTOSOUND and intracardiac echocardiography images of the aortic cusp. (*B*) Activation map of ventricular premature complexes. The geometry of the aortic cusp was reconstructed by CARTOSOUND. Three-dimensional mapping using CARTO 3 revealed the earliest activation at the right coronary cusp. ICE, intracardiac echocardiography; LV, left ventricle; NCC, non-coronary cusp; RCC, right coronary cusp; VPCs, ventricular premature complexes.
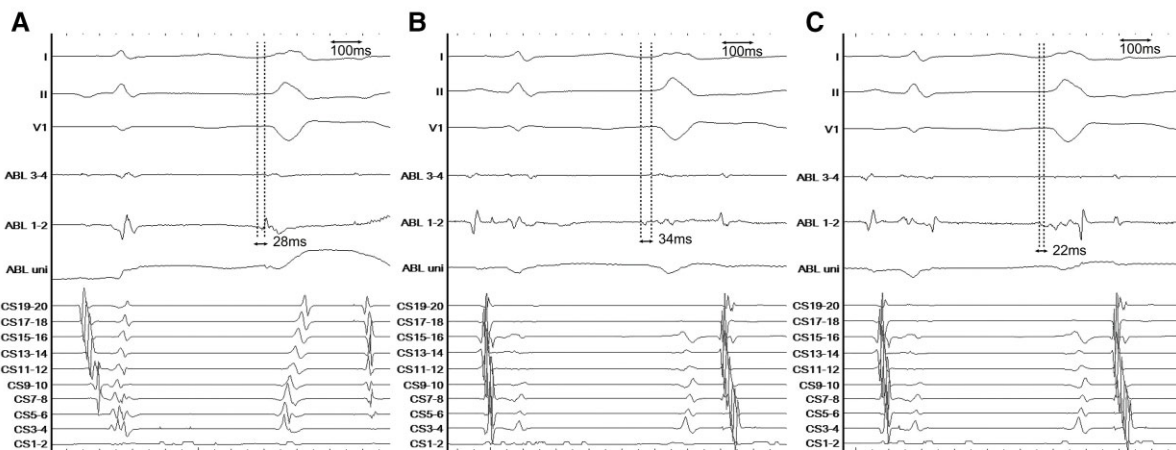
Figure 5 (*A*) Electrograms of target ventricular premature complexes were obtained by the ablation catheter. The local potential at the right coronary cusp preceded surface electrocardiography by 28 ms. Electrograms of the coronary sinus (coronary sinus; 19–20 proximal and 1–2 distal) are shown from top to bottom at a paper speed of 100 mm/s. (*B*) Electrograms of the earliest activation site of the recurrent ventricular premature complexes in the right ventricular outflow tract. The pre-potential preceded surface electrocardiography by 34 ms during ventricular premature complexes, and a clear His potential was recorded during sinus rhythm. (*C*) The electrograms of the recurrent ventricular premature complexes were obtained at the non-coronary cusp. The local potential at the non-coronary cusp preceded the surface electrocardiography by 22 ms; however, that was not the earliest. Far-field His potentials were recorded at the non-coronary cusp during sinus rhythm. CS, coronary sinus; ECG, electrocardiograms; NCC, non-coronary cusp; RCC, right coronary cusp; RVOT, right ventricular outflow tract; VPCs, ventricular premature complexes.

According to the current ESC guidelines,[1] ablation of aortic cusp VAs should only be performed at highly experienced ablation centres after the failure of at least one Class IC agent.[1] Our hospital, which has two electrophysiology (EP) rooms with 16 cardiologists including three board-certified EP consultants, performed 1675 cases of ablation in the last 5 years, of which 112 cases were VAs. Ablation at the aortic sinus of Valsalva can involve greater procedural complexity as well as some periprocedural risk (stroke, aortic valve cusp injury, or coronary artery injury) when compared with ablation at RVOT.

Aortography to detect the coronary artery and/or intracardiac echocardiography monitoring was recommended.

Ventricular arrhythmias arising from the RV septum near the His bundle can be successfully ablated in ~70–90% of patients, with several series reporting a higher likelihood of abandoning attempts at ablation due to concerns about inducing AV block.[10] Therefore, catheter ablation of VAs from para-Hisian sites should be considered only in patients with the symptomatic disease for whom antiarrhythmic medications are ineffective, not tolerated, or not the patient's preference.[10]

Contrary to the current ESC guidelines,[1] we did not use any Class IC agents before performing ablation. Because the surface ECG morphology had suggested that the clinical VPCs were of an RVOT origin, we therefore used β-blockers instead. As the drug therapy for 2 months did not alleviate her symptoms, the patient was reluctant to continue the drug therapy. Therefore, we performed catheter ablation, in which RF delivery at the NCC eventually proved to be effective. If VPCs had an obvious aortic cusp origin before the ablation, then we used Class IC agents first.

We experienced a case in which VPCs were successfully ablated from the NCC, which is considered rare. Radiofrequency energy application from the NCC may be a safe and effective option for the ablation of VPCs with the earliest activation at the RCC and His region.

Lead author biography

Atsushi Tanaka graduated from School of Medicine, Hokkaido University, Sapporo, Japan, in 2004, and earned his PhD degree from Graduate School of Medical Science, Kyushu University, Fukuoka, Japan, in 2013. He is interested in arrhythmia and heart failure and is working in the Department of Cardiology at Saiseikai Fukuoka General Hospital, Fukuoka, Japan.

Supplementary material

Supplementary material is available at European Heart Journal – Case Reports online.

Slide sets: A fully edited slide set detailing these cases and suitable for local presentation is available online as Supplementary data.

Consent: The authors confirm that written consent for publication of this case report including images and text has been obtained from the patient in line with COPE guidance.

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