Radiofrequency catheter ablation (RF-CA) is established as an effective and curative therapy for ventricular tachycardias (VTs) or symptomatic premature ventricular contractions (PVCs) originating from the outflow tract in the structurally normal heart. Most of these arrhythmias have their origin in the right ventricular outflow tract (RVOT), but VT originating from the pulmonary artery (PA) have been recently reported, although the characteristics of such tachycardias have not been sufficiently clarified.

We describe a case of symptomatic PVC with typical ECG characteristics of a tachycardia originating from the RVOT in which RF-CA at sites in the RVOT resulted in changes in the QRS morphology but did not completely eliminate the PVC. The need for careful and detailed mapping to find the optimal ablation site is highlighted by this case.

**Case Report**

A 27-year-old woman was admitted for RF-CA of symptomatic idiopathic ventricular contractions (PVCs). RF energy applications at 2 sites in the right ventricular outflow tract (RVOT), where both the earliest ventricular activation and near-perfect pace mapping were obtained, did not abolish the PVC but resulted in changes in the QRS morphology of the PVC. Complete elimination of the PVC was achieved with RF energy application at a site within the pulmonary artery 13 mm above the pulmonary valve, which was greater than 20 mm away from the failed ablation sites within the RVOT.

**Key Words:** Catheter ablation; Idiopathic ventricular contractions; Pulmonary artery; Ventricular arrhythmia

A patient underwent radiofrequency (RF) catheter ablation of symptomatic idiopathic ventricular contractions (PVCs). RF energy applications at 2 sites in the right ventricular outflow tract (RVOT), where both the earliest ventricular activation and near-perfect pace mapping were obtained, did not abolish the PVC but resulted in changes in the QRS morphology of the PVC. Complete elimination of the PVC was achieved with RF energy application at a site within the pulmonary artery 13 mm above the pulmonary valve, which was greater than 20 mm away from the failed ablation sites within the RVOT.

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Fig 1. (A) Twelve-lead ECGs in the patient with symptomatic idiopathic premature ventricular contractions. (B) Pace mapping from the septal aspect of the right ventricular outflow tract (RVOT). (C) Intracardiac recordings from the septal aspect of the RVOT before radiofrequency (RF) energy application. The local ventricular activation recorded by the distal electrode pair of the ablation catheter (Abl) precedes the onset of the QRS complex by 28 ms. Unipolar recording of the ablation catheter (uni) shows a QS pattern. GCV-AIV, bipolar recordings at the transitional area from the great cardiac vein to the anterior interventricular vein; HRA, high right atrium; prox, proximal.

Fig 2. Radiograms obtained in the right anterior oblique (RAO 35°; Upper panels) and left anterior oblique (LAO 45°; Lower panels) projections showing the ablation sites. (A) First ablation site. (B) Ablation site after the change in QRS morphology. (C) Final ablation site. The ablation catheter was positioned 13 mm above the base of the pulmonary valve (arrows), which was more than 20 mm away from the ablation sites in the right ventricular outflow tract. G-A, 2Fr octapolar catheter with an interelectrode spacing of 5 mm (PATHFINDER, Cardima, Fremont, CA, USA). Other abbreviations as in Fig 1.
Fig 3. (A) Premature ventricular contraction after RF catheter ablation at the septal RVOT. (B) Pace mapping near the posterior attachment in the RVOT. (C) Intracardiac recordings before the applications of RF energy at this site. The local ventricular activation recorded by the distal electrode pair of the Abl preceded the onset of the QRS complex by 7 ms. Other abbreviations as in Fig 1.

Fig 4. (A) Premature ventricular contraction after the second change in the QRS morphology following catheter ablation. (B) Pace mapping from the pulmonary artery at a site 18 mm above the base of the pulmonary valve. (C) Intracardiac recordings during sinus rhythm at the successful ablation site before and after RF energy delivery. A sharp potential following a tiny ventricular activation was recorded 13 mm above the pulmonary valve before ablation (arrow). After ablation, this potential disappeared. Other abbreviations as in Fig 1.
amount or the site from which a perfect pace map could be obtained, mapping in the PA was carried out and during sinus rhythm a sharp potential following a tiny ventricular activation, which has been reported as the characteristic ablation site of VT originating from the PA, was found 13 mm above the pulmonary valve (Figs 2C, 4C). Perfect pace mapping was obtained at this site (Fig 4B) and the sharp potential disappeared with RF energy delivery at this site, nor could the PVC be induced during isoproterenol infusion. The patient was discharged without the need for medications and there has been no recurrence of PVC during a 6-month follow-up period.

Discussion

Recently, VT originating from the PA was reported as a distinct subgroup of outflow tract tachycardias. It is believed that ectopic impulse formation starts in the PA followed by conduction over a myocardial sleeve to the right ventricle. Therefore, as has been previously suggested, interruption of the conduction by catheter ablation either at its beginning (in the PA) or at its insertion into the RVOT may be curative, as it is for an accessory atrioventricular pathway, with ablation at the atrial or ventricular end. However, in the present case, RF energy delivery at 2 sites in the RVOT where both the earliest ventricular activation was recorded and perfect or near-perfect pace mapping were obtained could not abolish the PVC completely, but instead produced PVC with different QRS morphologies. Complete elimination of the PVC was finally achieved with an RF energy application within the PA 13 mm above the pulmonary valve, where perfect pace mapping was obtained, and this site was more than 20 mm away from the sites within the RVOT where RF energy was delivered. Two different morphologic PVCs appeared after RF ablation in the RVOT that had never been observed before the ablation procedure, and no PVC could be induced after RF ablation within the PA. These results indicate that the origin and exit of the tachycardia were present in the PA and RVOT, respectively, and that RF ablation at the origin within the PA cured the PVC completely. Therefore, when a tachycardia originates from the PA and its activation is conducted to the RVOT through a myocardial connection between the focus within the PA and the RVOT, it may be necessary to ablate the tachycardial origin or the myocardial connection near the origin. Conversely, when RF energy is delivered at the exit site of the PVC within the RVOT, it may be difficult to cure the tachycardia because of changes in the exit point or activation of an alternative pathway between the focus and the exit point. In a previous report, successful ablation could not be obtained with RF energy deliveries at the RVOT, but the arrhythmia was ablated by RF energy delivery at a focus within the PA. Although repetitive ventricular responses appeared and ceased during RF energy application and were considered to be suggestive of successful ablation of the outflow tract tachycardia, this finding may not be helpful in predicting successful ablation of tachycardias of this kind.

The characteristic ECG findings of a tachycardia originating from the RVOT and the left ventricular outflow tract and of tachycardias with their origin in a left ventricular epicardial site that can be ablated from the aortic sinus of Valsalva have been reported. In such tachycardias, the origin can be identified with moderate accuracy by ECG analysis. In the present study, all 3 different morphologic PVCs observed during the ablation procedure had an LBBB QRS morphology and an inferior axis, and all were classified as tachycardias originating from the RVOT by ECG analysis. None of the specific ECG findings were found in the tachycardia originating from the PA. A previous study also demonstrated that the ECG of a PA VT in 100% of patients was an LBBB pattern with an inferior axis, and that the ECG characteristics of the VT could not be distinguished between an RVOT and PA origin of the VT. Because the RVOT is the exit site of the tachycardia originating from the PA, differentiation between the tachycardia originating from the RVOT and PA can be difficult. Therefore, a tachycardia originating from the PA should be considered whenever it has ECG characteristics of a tachycardia originating from the RVOT or it shows an LBBB QRS morphology with an inferior axis. When neither early activation time during the VT or PVCs, nor an optimal pace map are found in the RVOT, detailed mapping of the PA including the pulmonary valve should be performed. At the time of a repeat ablation procedure in patients with a previously unsuccessful RVOT ablation, careful mapping of the PA should be performed.

In the present study, complete elimination of the PVC was achieved with an RF energy application within the PA where the sharp potential following a tiny ventricular activation was recorded during sinus rhythm. Perfect pace mapping was obtained during pacing from this site. After an RF energy delivery, the sharp potential disappeared, and no PVC could be induced with or without isoproterenol infusion. Similar findings were observed in a previous study, which also demonstrated that this potential preceded both the QRS onset and the ventricular activation during the tachycardia. Therefore, the presence of this potential during sinus rhythm and tachycardia may indicate an origin in the PA. These findings may help to identify the optimal ablation site within the PA. Furthermore, elimination of this potential may also indicate successful elimination of this type of tachycardia.

The results obtained from the present case and previous studies cannot determine why myocardial tissue is present in or around the pulmonary artery. Both the embryonic avian and mammalian outflow tracts, as well as the outflow tract in adult primitive fish (called conus) and amphibians (called bulbus cordis), are surrounded by myocardium. In animal hearts, the distal part of the outflow tract loses its myocardial cuff and becomes the proximal part of the ascending aorta and pulmonary trunk during embryogenesis. In contrast to the myocardium of the atrial and ventricular chambers, this myocardium retains its embryonic features (ie, slow propagation of the depolarizing impulse owing to the poor intercellular coupling of the cardiac muscle cells). Myocardial regression in the outflow tract continues until after birth, as revealed by the disappearance of the myocardial cuff surrounding the semilunar sinuses. Incomplete retraction of this myocardium may be the reason for the presence of a myocardial sleeve in the main stem of the PA, which is connected to the RVOT and remnants persist that may provide the substrate for the arrhythmia.

Variations in QRS morphology of VT or PVCs following catheter ablation as in the present case have been reported in patients with idiopathic VT. A change in the exit point or activation of alternative pathways between the tachycardia focus and exit point is a plausible reason...
for the change in the QRS morphology following catheter ablation. In the present case, RF energy applications near but not at the tachycardia focus might have resulted in both changes in the QRS morphology. Therefore, careful and detailed mapping should be performed to find the optimal ablation site, and RF energy delivery should be delivered at the site where the earliest activation time is obtained during the tachycardia and/or a perfect pace map is obtained. Another possible explanation for the change in the QRS morphology of the PVCs after the RF ablation is the appearance of new foci of the PVCs recorded before the RF ablation. However, because the ECGs recorded before the RF ablation always exhibited a single monomorphic QRS morphology, the appearance of new, unsuppressed foci may be a more plausible explanation than a change in the exit point or activation of alternative pathways between a single PVC focus and an exit point. Therefore, the ECGs recorded before the ablation procedure always exhibited a single monomorphic QRS morphology and the QRS morphology of the PVCs recorded was identical, we believe that all PVCs recorded during the tachycardia and/or a perfect pace map is obtained. Another possible explanation for the change in the QRS morphology following catheter ablation, requiring additional radiofrequency ablation at a different point in the tachycardia. Circulation 1992; 20: 1397–1404.


