

EDITORIAL COMMENT

Ablation of Ventricular Arrhythmias Arising From the Pulmonary Artery*



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The right ventricular outflow tract (RVOT) is the most common site for ventricular arrhythmias (VAs) in patients with no overt structural heart disease. VAs that originate from the RVOT exhibit a signature left bundle branch block (LBBB)–inferior axis electrocardiographic (ECG) pattern. Most VAs arising from the RVOT are benign premature ventricular contractions (PVCs), but up to 10% can be life-threatening arrhythmias, including ventricular tachycardias/ventricular fibrillations (VTs/VFs) that can lead to syncope or cardiac arrest (1). Over the past decade, the incidence of these arrhythmias has increased, in more and more patients who either have had a high burden of symptomatic PVCs or VT that require compulsory treatment (2).

Over the past 2 decades, catheter ablation has gained popularity as a substitute for medical therapy (eg, β -blockers, antiarrhythmic drugs) because several studies have shown that ablation is superior to medical therapy with respect to clinical outcomes. In addition, the RVOT is an easily approachable and amendable area for mapping and ablation, which can be carried out at a relatively low risk. However, the long-term success of ablation treatment for VAs that have an LBBB–inferior axis ECG pattern is only 82%, which is far from ideal (2).

The results of VA ablation that originates from the RVOT is expected to be on par with supraventricular tachycardia ablation (ie, cavo-tricuspid, isthmus-dependent atrial flutter) (>95%). The main reason the results are different is because the anatomy of the outflow tract and its connection to the neighboring structure is more intricate than it appears (3). In short, the distal RVOT and pulmonic valve are close to the aortic valve. Myocardial extensions from the outflow tract are common into the great arteries above the semilunar cusps and often are the origin of the VAs. Several studies have demonstrated that arrhythmia foci of VAs with LBBB and inferior axis morphology do not always originate from the RVOT but may emanate from other neighboring areas contiguous to the RVOT. Thus, operators should realize that the site of the arrhythmia may be at the semilunar cusps or pulmonary artery (PA). In the past, many operators believed that VAs with LBBB and inferior axis came from the RVOT, and they started ablating the earliest site in the RVOT before mapping other areas (ie, semilunar cusps or PA) to make sure the RVOT was the earliest site of the origin of the VA. As a result, ablations at this RVOT site sometimes failed to eliminate the arrhythmias and sometimes affected the morphology of VAs, which created confusion and raised the possibility that VAs had multifocal origins, as shown in the nice example by Yagishita et al (4) in this issue of *JACC: Case Reports*.

It was during a case of LBBB–inferior axis VT that had a transitional zone at lead V_4 that caused the investigators to initially search at the RVOT free wall. After they found the RVOT earliest, where pace mapping produced the same morphology as clinical PVCs/VT, they proceeded to ablate at this site. However, RVOT ablation did not abolish the VAs but instead produced serial changes of PVC morphologies, prompting Yagishita et al (4) to search other sites in the PA, where they found the true earliest site of VT origin.

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Many other investigators have shared the same experience in the literature, that ablations at the RVOT site they presumed to be the VA foci often only slightly changed the morphology of the VA without abolishing the arrhythmias (5). When this scenario happens, the site of the arrhythmia origin is almost always elsewhere in neighboring areas mentioned previously. As in the Yagishita et al (4) case, the site of the VA origin was in the PA. The reason the PVC morphology changed after ablations in the RVOT free wall was probably caused by the effects of ablation on the exit sites of the arrhythmias from the PA to RVOT. Other educational features of this case include how they mapped and ablated the arrhythmogenic site.

Yagishita et al (4) showed the usefulness of using circular multipole catheters to record beautiful PA potentials from above the pulmonic valve, which was the earliest activated site during VT. This bipolar PA potential spike was activated late during sinus rhythm but reversed to become the earliest spike during VT. Hence, this PA potential represents the near field of local activation of the muscle sleeve that extends from the RVOT to the PA. Although this phenomenon of the far field–near field reversal when sinus rhythm changes into VT has been described before (6), Yagishita et al (4) should be commended for clearly demonstrating the usefulness of multiple recordings simultaneously and circumferentially around the PA by the circular catheter. They delineated the early activation around this PA site (see Figures 4 and 5 in Yagishita et al [4]) and showed the likelihood that there were probably multiple arrhythmogenic sites in the PA. This observation, in turn, suggested reentry as the mechanism of the VA in this patient. Furthermore, the manner in which these distinct PA potentials caused VA was similar to pulmonary vein potentials that triggered atrial fibrillation. They were the arrhythmogenic potentials that originated the tachyarrhythmias.

The most noticeable and important feature of this case is how the investigators ablated the arrhythmogenic site. They elected to ablate below the pulmonic valve where the earliest PA potential was recorded because they were concerned about potential complications from ablation in the PA. Some parts of the PA right above the valve are close to the

coronary ostium, and ablation in such areas may cause collateral damage to the artery. However, if they had established that the VT foci was far from the coronary ostium, ablation at the PA arrhythmogenic site could have been as effective and safe. Because the investigators did not use 3D mapping at the time of the procedure and did not have the tool to tag the earliest site of the PA potential during VT, the circumstance perhaps forced them to leave the circular catheter in the PA as a landmark for ablation catheter positioning. So, it was logical that they carried out the ablations beneath the valve just below the arrhythmogenic sites. In essence, they applied a similar strategy as pulmonary vein isolation for treatment of atrial fibrillation. The ablations were performed during sinus rhythm with the target sites being spike sharp PA potential sites. Yagishita et al (4) were able to successfully isolate the PA and eliminate the VA without having to circumferentially ablate around the PA. Their results and observations not only confirmed that there were existing arrhythmogenic myocardial extensions into PA, but also suggested that the areas of myocardial extensions were not capacious.

This reported case is instructive and educational. The lessons learned from this case include the following. 1) The preconceived notion that PVCs/VTs with LBBB morphology and an axis always emanate from the RVOT should be reconsidered; operators should be cognizant of the potential site of origin from the PA or aortic cusp. 2) Thorough mapping in the PA and sometimes in the aortic cusp may be required. 3) Using a multipole catheter for mapping should be considered. Whether to use a circular multipole catheter to map routinely is determined by the operator's experience, because a circular mapping catheter poses a risk of the catheter being entrapped in the valve structure. In a case like Yagishita et al (4), it is best to map using 3D electroanatomical mapping, which allows merging and integrating of the 3D map to imaging from either x-ray or computed tomography of the heart. 4) PA isolation can be done successfully for treatment of VAs arising from the PA. Finally, Yagishita et al (4) should be congratulated for showing a compelling example to help with mapping and ablation of patients who have VAs arising from the PA. Future research is warranted to determine the

value and limitations of PA isolation in treating these arrhythmias.

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