EDITORIAL COMMENTARY

Pulmonary sinus of Valsalva arrhythmias: A new twist to an old story

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Idiopathic ventricular arrhythmias with a left bundle branch block and inferior axis morphology typically occur in patients with structurally normal hearts and commonly originate from the right ventricular outflow tract (RVOT).[1] Ventricular arrhythmias originating in the pulmonary artery have also been described in small retrospective series/case reports but were considered to be relatively uncommon (4%–16%).[2–4] Such arrhythmias have been thought to arise from remnants of muscular sleeves that extend above the pulmonary valve and may be ablated in the pulmonary artery.

Myocardial recordings and voltage extensions beyond the pulmonary valve annulus have been demonstrated in 92% of controls and 88% of patients with RVOT arrhythmias.[5] Similar to the left ventricular outflow tract, ventricular myocardial extensions beyond the ventricular-arterial junction have also been described in the RVOT, as both regions were derived from the embryonic infundibulum. Since there is no true valvular "annulus," the semilunar valve leaflets are attached directly to the right ventricular (RV) myocardium.[6] The junction between the RVOT and the pulmonary trunk is complex, with the hinge line of the valve leaflets repeatedly crosses the ringlike (planar) junction between the RVOT and the vessel wall. The valve sinus is therefore bounded by cardiac muscle in its depths and by the vessel wall more superiorly and proximal to the sinotubular junction[6–8] (Figure 1).

Analogous to idiopathic ventricular arrhythmias originating from the aortic sinus of Valsalva,[9] the presence of electrogram recordings within the pulmonary sinuses was also highly predictive of a successful ablation outcome.[2,10] A "late" potential was often evident in the valve sinuses during sinus rhythm that became the "early" electrogram during ectopic beats, reflecting recordings from the distal ventricular myocardial extensions connecting RVOT to the pulmonary sinus cusps.

In this issue of **Heart**Rhythm, Zhang et al[11] publish their single-center experience of mapping and ablation of idiopathic RVOT arrhythmias. The study enrolled a nonselected group of 90 patients with ventricular arrhythmias of left bundle branch block–inferior QRS morphology. Detailed activation and pace mapping were performed above the pulmonary valve within the sinuses of Valsalva and below the valve in the RVOT, coupled with right ventriculography and/or pulmonary angiography. After excluding tricuspid annular (n = 4) and left ventricular outflow tract arrhythmias (n = 5), the earliest ectopic activations were mapped to the pulmonary valve sinuses and ablation within the sinuses effectively eliminated arrhythmias in all remaining 81 patients, without any additional ablation in the RVOT.

This study is the first prospective analysis of idiopathic RVOT arrhythmias of pulmonary valve sinus origin and represents a new concept regarding our understanding of the anatomy and mechanism of RVOT arrhythmias. One of the limitations of the study is the lack of intracardiac echocardiography imaging to corroborate the angiographic findings. Although RVOT arrhythmias can frequently be ablated from the "RVOT," most of the ablation sites may actually be supravalvular as the location of pulmonary valve is poorly defined. Nonetheless, it is remarkable that nearly all unselected patients with a diagnosis of RVOT arrhythmias had successful ablation in the pulmonary valve sinuses.

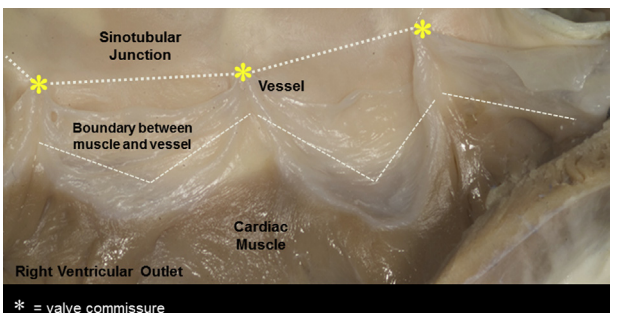

Figure 1 Close-up of the complex junction between the infundibulum and the pulmonary artery root. The attachments of the valve leaflets repeatedly cross the planar boundary between the infundibular cardiac muscle and the arterial wall. Thus, each sinus of Valsalva has a wall of cardiac muscle proximally (dark tissue in the depths of the sinus) and a wall of vascular tissue distally. Excluding the sinuses, the same biphasic walls are seen between the leaflet attachments. Permission obtained from Dr Philip C. Ursell, Department of Pathology, University of California, San Francisco, CA.

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Based on anatomical findings, mapping results, and imaging, ubiquitous RV myocardial extensions beyond the pulmonary valves form the basis of the current observations, with myocardial fibers within the pulmonary valve sinuses as the true origin of RVOT ventricular arrhythmias. Since muscle fibers seldom invade the valve leaflets, successful ablation in the pulmonary valve sinuses involves ablation of the contiguous RV infundibular muscle, lining the walls and floor of the sinus of Valsalva.

This paradigm shift may have a significant impact on our ablation strategy and outcomes for this common arrhythmia. In this study, the authors suggested that mapping and ablation in the pulmonary valve sinus using the "reversed U curve" supported by a long sheath should be considered as the preferential approach for idiopathic RVOT arrhythmias. However, this technique for mapping the pulmonary valve sinuses may be difficult and is associated with a learning curve.[10] One important consideration for such supravalvular ablation is the close proximity (<5 mm) of septal superior RVOT and pulmonary valve sinuses to the major coronary arteries with the risk of coronary artery injury.[12] Other potential procedure-related complications include damage to the pulmonary valves/sinuses, resulting in valvular insufficiency/stenosis.[13] Prospective comparison of the safety and efficacy of primary pulmonary sinus cusp ablation to the conventional "RVOT" ablation is needed with real-time intracardiac echocardiography imaging, before routinely adopting such supravalvular ablation as a first-line approach in all patients with idiopathic RVOT ventricular arrhythmias.

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