Outflow tract PVCs : Left or Right?

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A 40-year-old man with drug refractory palpitations was referred for catheter ablation. A transthoracic echocardiogram and cardiac magnetic resonance imaging revealed no structural heart disease. Electrocardiogram revealed premature ventricular contractions (PVCs) with two different morphologies and coupling intervals (PVC1 630ms, PVC 2 540ms, Figure 1 A). Although both PVCs had left bundle branch with inferior axis morphology, PVC1 was narrow (QRS duration =140ms, V2 transition) compared to PVC2 (QRS duration = 190ms, V4 transition) and had a later precordial transition.

The electroanatomical mapping (EAM) and ablation was performed using a three dimensional EAM system (Carto3; Biosense Webster, Diamond Bar, CA) with 7.5F irrigated-F curve catheter (Navistar Themocool; Biosense Webster, Diamond Bar, CA). Activation and pace mapping was initially performed in the right ventricular outflow tract (RVOT) and pulmonary cusps (Figure 1 B-C). With regards to PVC2, the earliest activation site was in the posterior septum, where local ventricular activation preceded QRS onset by 20ms. The unipolar electrogram at this site showed a QS pattern. Pace mapping at this site produced a QRS morphology with a pace-map score of 19/24 for PVC2. The pacing stimulus-QRS (S-QRS) interval of 8 ms. A radiofrequency (RF) application using a power of 30 W up to 43^{O}C failed to suppress PVCs.

What is the possible reasons why radiofrequency ablation in the RVOT failed to suppress the PVCs?

Where should further mapping of these PVCs be carried out?

Discussion

At this point, the options in this case include using a higher energy in the RVOT, mapping of the coronary venous system or the aortic cusp. As there not any suppression of PVCs using a power of 30 W up to 43^{O}C, a higher energy was not attempted. Mapping of distal CS and anterior interventricular vein junction was performed but the activation during PVC2 was not early. Hence, further mapping of the coronary venous system was not performed. We proceeded with activation and pace mapping of the aortic cusp. (Figure 2 A-D)

Activation mapping in the right and left coronary cusp (RCC/LCC) junction revealed that local ventricular activation preceded QRS onset by 30 and 28 ms for PVC1 and PVC2, respectively. The unipolar electrograms at this site for both PVCs showed a QS pattern. Pacing with an output of 6mA (fixed pulse width of 2ms) at the RCC/LCC junction produced QRS morphologies only similar to PVC1 with a pace map score of 20/24. Pacing with an output of 9mA at the same location produced QRS morphologies similar to both PVC 1 and PVC2 with pace map scores of 20/24 and 19/24, respectively. Interestingly, pacing with an output of 15mA at the same site produced QRS morphologies similar to only PVC 2 with a pace map score of 21/24 and a S-QRS interval of 38ms. Subsequent pacing at the same output showed a decremental conduction (38,50,56 ms). Ablation at this site in the RCC/LCC junction using a power of 30 W up to 43^{O}C suppressed both PVCs within 5 seconds of starting energy. The lesion was further consolidated for total of 60 seconds. The

ectopy did not recur during a waiting time of 30 minutes including an isoporterenol challenge. At 18 months of follow up, the patient remains asymptomatic without any recurrence of PVCs. PVC originating from single site had 2 exits with 2 different morphology .

In this case, a PVC originating from the aortic cusp had preferential conduction to two exits in the outflow tract and exhibited two different morphologies of PVCs. Outflow tract anatomy and electrophysiological properties of the surrounding myocardium may explain this observation. Parts of the right and left coronary aortic leaflets are related to the ventricular septum and left ventricular free wall, respectively.[1] In these areas, ventricular myocardium extends beyond the semilunar valves, enclosing muscle at the cusps of the aortic sinuses. These extensions can vary in course (oblique or longitudinal), location (endocardial or epicardial), or continuity with underlying ventricular musculature. In addition, myocardial hypertrophy, fibrosis, and interposed adipose tissue have been described within these myocardial extensions.[2] The complex anatomy of these extensions may contribute to variable exits across the circumference of the aortic cusps.

Studies have suggested that specialized myocardial fibers can contribute to preferential conduction from the aortic sinus cusp to the RVOT.[3-5] As hypothesized by Yamada et al, preferential conduction via myocardial fibers in this case is supported by the significantly longer stim-QRS interval pacing from the aortic cusp compared to the RVOT.[6] In addition, this case demonstrated two novel properties of these myocardial fibers. First, pacing at a higher output from within the aortic cusp yielded a closer match to the QRS morphology of PVC2 than pacing from within the RVOT. Pacing at lower outputs from the same location diminished the preferential conduction of PVC 2 from the aortic cusp to the RVOT. These findings suggest that an insulated myocardial fiber travelling from the origin in the aortic cusp to the breakout site in the RVOT might exist. (Figure 3) Such a myocardial fiber may only be selectively captured with a higher pacing output. Second, pacing at a higher output in the aortic cusp revealed decremental conduction with longer S-QRS intervals. This may support the presence of slow conduction within these myocardial fibers. Preferential conduction of arrhythmias originating from the aortic cusp may be explained by a combination of structural and functional properties unique to myocardial fibers in this location.

References:

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Figure Legends

Figure 1: 12 Lead ECG with Activation and Pace Mapping of PVC 2 in the RVOT

A : Twelve lead electrocardiogram recorded during the procedure showing sinus rhythm (SR) and premature ventricular contractions (PVC) 1 and 2. B: Intracardiac electrograms recorded during activation mapping

of PVC2 at the RVOT posterior septum. The black arrow indicates the local ventricular potential preceding the QRS onset by 20 ms. The unipolar electrogram showed a QS pattern. C: Pace mapping at the RVOT posterior septum revealed a pace map score of 18/24 for PVC 2. The pacing stimulus to QRS interval was 8ms (arrowhead). MAPD, MAPP (the distal and proximal electrode pairs of the mapping catheter); UNI (the distal unipolar electrode of the mapping catheter).

Figure 2: Activation and Pace Mapping of PVC 1 and PVC 2 in the Aortic Cusp

A: Intracardiac electrograms recorded during activation mapping of PVC 1 and PVC 2 at the junction of the left coronary and right coronary cusp (LCC/RCC junction). The black arrow indicates the local ventricular potential preceding the QRS onset by 30 and 28 ms for PVC 1 and PVC 2, respectively. B: Pacing at a 6mA output at the LCC/RCC junction revealed a 20/24 pace-map for PVC 1. C: Pacing at 9mA at the same site revealed QRS complexes with morphology similar to PVC 1 (5^{th}QRS complex, pace map 20/24) and PVC 2 (4^{th} QRS complex, pace map 19/24). The first three paced complexes represent fusion complexes. The pacing stimulus to QRS (S-QRS, black arrowhead) for PVC2 was 28ms. D: Pacing at 15mA at the same site revealed QRS morphology similar to only PVC 2. Pace-map score was 21/24 with a S-QRS interval of 38, 50 and 56 ms, suggestive of decremental conduction. Refer to Figure 1 for other abbreviations.

Figure 3

Diagram showing the origin and presumed preferential conduction paths of PVC 1 and PVC 2. With regards to PVC2, we hypothesized that preferential conduction occurred via an insulated myocardial fiber from the origin in the aortic cusp and the exit in the RVOT septum.