Utility of Multi-spline Multi-electrode 3D Mapping Catheters in Idiopathic Outflow Tract Ventricular Tachycardia.

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Abstract

We describe two cases of idiopathic VT arising from the postero-septal RVOT in both cases. Each case demonstrates an important learning point and advantage of multi-spline multi-electrode mapping catheters in idiopathic outflow VT. In the first case two dominant PVC morphologies can be clearly seen which we believe originate from a single site of origin with multiple exit sites caused by focal fibrosis. In the second case, focal fibrosis in this region leads to anisotropic conduction of the RVOT in areas of low voltage. We surmise that multi-spline multi-electrode catheters are essential in such cases as extensive point-by-point mapping could significantly prolong procedure time and fail to identify the earliest activation site in the case of anisotropic tissue conduction due to variations in activation latency.

Introduction

The mechanism of premature ventricular complexes (PVCs) and ventricular tachycardia (VT) arising from the ventricular outflow tracts is thought to be enhanced automaticity by cAMP-mediated triggered activity
(1). Imaging studies involving cardiac magnetic resonance (CMR) have not demonstrated any structural abnormalities to date (2, 3). However, there are multiple studies using intracardiac mapping that have demonstrated areas of low voltage within the outflow tracts of patients with idiopathic VT (1, 2). Fibrosis is associated with late local activation and areas of slow conduction and slower wavefront propagation speed, known as deceleration zones (DZ) which facilitate re-entry VT (4). Previous studies have observed that wavefront propagation of PVCs from a right ventricular outflow tract (RVOT) origin is slower than those from a left ventricular outflow tract (LVOT) origin which the authors attributed to muscle fiber orientation (5). Brugada et al. (1991) showed the significance of tissue anisotropy in the initiation of re-entrant VT which they also attributed to muscle fiber orientation (6). However, this finding could be attributed to areas of low voltage and activation delay which has been found in patients with arrhythmogenic cardiomyopathy and Brugada syndrome (7, 8). Here we describe two cases of posterior RVOT PVC/VT ablation showing the utility of multi-spline multi-electrode mapping catheters to elucidate the site of origin particularly in the context of low voltage areas and anisotropic tissue activation.

Case 1

A 52-year-old lady presented with a 5-year history of palpitations with occasional presyncope. She underwent a 24-hour Holter monitor which showed a 60% burden of unifocal PVCs and non-sustained monomorphic VT. Her 12 lead ECG was in keeping with a posterior RVOT origin. Her CMR showed a structurally normal heart with no evidence of late gadolinium enhancement in keeping with idiopathic VT. She had no significant background medical history. Her only medication was bisoprolol 5mg prescribed for her PVCs/VT. Her family history was significant with an older brother who died at 49 years due to sudden cardiac death although the details of the autopsy were not available to us.

She underwent a VT ablation under general anaesthetic. An 8 French femoral sheath was placed in the right femoral vein and right femoral artery under ultrasound guidance. Her baseline ECG showed two dominant morphologies both consistent with an outflow tract origin with the largest differentiation seen in V3 (Figure 1.). The RVOT was initially mapped with a five-spline multi-electrode mapping catheter with a broad area of the posterior RVOT noted to be early. The aortic root was then mapped and the left sinus of Valsalva was found to be early however the PVCs and VT terminated upon introduction of the catheter into the left sinus of Valsalva. An isoprenaline infusion was commenced at 8 mcg/min for 20 min and then stopped. VT returned but terminated once again in response to introduction of the multielectrode catheter. VT returned and radiofrequency (RF) applications at 30W at the earliest sites in the aortic root did not terminate or reduce frequency of PVCs/VT. The RVOT was then remapped with the multielectrode catheter and subsequently with the RF ablation catheter. The earliest posterior wall site was 38ms pre the surface ECG. An application of 30W RF at this site terminated all PVCs and VT. The initial map was created using a pattern match filter of 97% for point collection. It was then discovered that PVC 1 had a match of 99% to the original template and PVC 2 had a match of 97%, with the biggest discrepancy in lead V3. From the 7000 points taken in the original map, these were then separated into two maps; one map consisting of 99% match points and one map consisting of 97% match points (Figure 1).

Case Two

A 43-year-old lady presented with a history of palpitations and a 42% burden of outflow tract PVCs on a 24-hour Holter monitor. Her 12 lead ECG was in keeping with a posterior RVOT origin. She had no significant past medical history and was taking Flecaainide 100mg twice daily and Diltiazem XL 120mg once daily for symptomatic PVCs. She had no significant family history of note.

She underwent a PVC/VT ablation under general anaesthetic. An 8 French sheath was inserted into her right femoral vein under ultrasound guidance. Her baseline ECG showed a dominant morphology consistent with an outflow tract origin. The RVOT was mapped with an eight-spline multi-electrode catheter with an area of the posterior RVOT noted to be early. A propagation map showed anisotropic activation of the RVOT from a focus in the high septal RVOT (Figure 2) which arose from the junction of the low voltage annular section and the normal voltage lower RVOT section (Figure 3). The RF ablation catheter was inserted and
the earliest site was 32ms pre the surface ECG in the high postero-septal RVOT at the transition of normal to low annular voltage. An application of 30W RF at this first site terminated all PVCs and VT.

Discussion

Multi-spline multi-electrode mapping catheters provide higher mapping density, better substrate definition and higher detection of local abnormal ventricular activity (9). This has proved particularly advantageous in scar-related VT for identifying arrhythmogenic surviving myocardial bundles (9). Here we demonstrate their utility in idiopathic outflow tract VT through two cases showing: (1) activation mapping of two subtly different PVC morphologies suggesting different exit sites from a single site of origin; and (2) potentially anisotropic activation of the RVOT in association with low voltage areas.

Activation mapping is the current gold standard for localizing an RVOT-VT focus (10). In case 1, two dominant PVC morphologies were observed with the most significant differentiation being seen in V3. Figure 1 shows activation maps of these two dominant PVC morphologies found to be earliest in the posterior RVOT. The earliest site was found in the postero-septal RVOT in low voltage areas and RF ablation at this site terminated all PVCs and VT. We hypothesise that the different PVC morphology is caused by multiple exit sites in areas of focal fibrosis from a single site of origin. Given the earliest site in the postero-septal RVOT was found in low voltage areas we speculate this represents areas of impaired and anisotropic conduction secondary to focal fibrosis. The variation in conduction velocities accounts for preferential conduction to multiple exit sites. With multiple different PVC morphologies, multi-spline multi-electrode mapping catheters can rapidly identify the areas of earliest activation which would be challenging with point-by-point single RF catheter mapping.

Anisotropic activation can be most clearly seen in case 2 as shown by the propagation map in Figure 2. This shows a centrifugal conduction velocity that is non-uniform from the postero-septal RVOT. Two potential mechanisms may account for this phenomenon including (1) focal fibrosis and (2) muscle fiber orientation in the RVOT. Case 2 demonstrates areas of tissue anisotropy occurred in association with low voltage areas at the junction of the upper and lower RVOT segments as shown in Figure 3. Previous studies have observed the presence of low voltage areas in the RVOT of patients undergoing RF ablation despite normal CMRs (1, 2). Despite normal CMR imaging, subtle areas of interstitial fibrosis cannot be excluded. Focal fibrosis would lead to conduction delay and deceleration zones which is associated with re-entry rather than triggered activity which is typically associated with idiopathic outflow tract VT. However, it is possible that idiopathic outflow tract VTs do not all share the same mechanism. It has previously been shown that the site of origin is located in low voltage areas in 3% of patients, transitional voltage zones in 89% of patients and in high voltage areas in 8% of patients (11). In case 2, the successful ablation site was at a transition of normal to low annular voltage in the RVOT. The site of earliest activation was between 32ms and 38ms pre the surface ECG in both cases suggestive of significant delayed activation.

In terms of muscle fiber orientation, the RVOT has circumferential muscle fibers parallel to the atrioventricular groove in the subepicardial region but longitudinally aligned in the endocardial surface (12). Therefore, the shape of the isochronal maps in the RVOT may differ depending on the site of origin because the propagation velocity depends on the fiber orientation with the velocity of the impulse propagation being higher in the longitudinal direction (13). A previous study of 23 patients, found that PVCs originating from the RVOT exhibited a slower wavefront propagation speed than PVCs from adjacent sites (14). Previous work by Herczku et al. (2012) showed that PVCs originating from the RVOT can be differentiated from the left ventricular outflow tract by a smaller 10ms isochronal map area and higher longitudinal/perpendicular axis ratio (5). In both studies their findings were attributed to muscle fiber orientation in the RVOT creating different propagation speeds with a site in the septal RVOT exhibited an isochronal map with a rounded shape. In contrast, we believe the change in conduction speed is related to the presence of substrate in the RVOT in the cases presented above. Both cases demonstrate earliest sites of activation in areas of low voltage in association with centrifugal conduction velocity that is non-uniform. In support of this hypothesis previous studies have shown the presence of low voltage areas in the RVOT of patients undergoing PVC/VT ablation despite normal CMRs (1, 2, 15).
Whether caused by focal fibrosis, muscle fiber orientation or a combination of both this would make mapping with a single catheter very challenging as the anisotropic activation would result in significant variations in activation latency at points in close anatomical proximity.

Overall, the use of multi-spline multi-electrode mapping catheters to identify areas of substrate and sites of earliest activation proved clinically very useful in these two cases. Extensive point-by-point mapping could significantly prolong procedure time and fail to identify the earliest activation site in the case of anisotropic tissue conduction due to variations in activation latency.

Conclusions

The use of multi-spline multi-electrode mapping catheters is highly advantageous, effective and arguably essential for determining the optimal ablation site for idiopathic VT originating from the RVOT, especially in patients with anisotropic activation patterns which we attribute to areas of low voltage.

**Figure 1.** Case 1. 3D Activation maps of the two dominant ventricular ectopic morphologies found to be earliest in the posterior right ventricular outflow tract.

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**Figure 2.** Case 2. Propagation map showing anisotropic activation of the right ventricular outflow tract.
Figure 3. Case 2. 3D Activation and Voltage maps of dominant ventricular ectopic morphology in the right ventricular outflow tract in association with areas of low voltage.

References


