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How To Identify & Treat Epicardial Origin Of Outflow Tract Tachycardias

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Abstract

The right ventricle outflow tract (RVOT) is the most common site of origin of idiopathic ventricular arrhythmias. The typical outflow tract arrhythmias pattern on ECG is an inferior axis deviation and left bundle branch block when originated on the RVOT and right bundle branch block morphology when originated on the left ventricular outflow tract (LVOT). There are several ECG tricks for different locations of origin. An increased Maximum Deflection Index (MDI) suggests epicardial origin of arrhythmia. In general the result of ablation is very good, but sometimes there are difficult and unsuccessful procedures. The origin in the aortic cusps and epicardium are the reason for failure in some cases. When they are epicardial, the arrhythmias can be accessed by the venous system or by subxiphoid epicardial mapping.

Introduction

Idiopathic ventricular arrhythmias are characterized by the absence of structural heart disease based on resting ECG, echocardiogram and cardiac resonance with gadolinium and late enhancement. The right ventricular outflow tract (RVOT) is the most common source of origin, typically expressed by premature ectopic beats (PVCs), non-sustained ventricular tachycardia (NSVT) and rarely, by sustained VT. Their mechanism seems to be related to triggered activity due to catecholamine-mediated delayed after-depolarizations. There is an increase in intracellular calcium from the sarcoplasmic reticulum, resulting in delayed after-depolarizations and triggered activity.[1]

The typical outflow tract arrhythmias pattern on ECG is an inferior axis deviation, characterized by positive QRS complexes in D2, D3 and aVF. Left bundle branch block morphology often suggests an RVOT origin and right bundle branch block morphology typically suggests a left ventricular outflow tract (LVOT) origin. However, there are many ECG tricks for different locations.[2-6]

The anatomy of the outflow tract is very complex (Figure 1), since the RVOT is located leftward and anterior to the left ventricular outflow tract (LVOT). The pulmonary valve is located superiorly and the superior aspect of the tricuspid valve is located inferiorly.[7] Coronary venous and arterial systems have close relations with those regions.

Key Words:
Idiopathic Ventricular Arrhythmias, Right Ventricular Outflow Tract, Left Ventricular Outflow Tract, Ablation, Epicardial Mapping, Subxiphoid.

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None.

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Catheter ablation of RVOT and LVOT has been standardized and, in general, their success rate is very high. However, 15% to 20% of patients have difficult and sometimes unsuccessful procedures. One of the reasons for failure is the epicardial origin of the PVC, related or not to the vascular system. Sometimes, it is non-accessible from the conventional endocardial approach. So, it is fundamental to determine their precise location to perform an adequate ablation.[8-10]

How To Identify Epicardial Origin Of Outflow Tract Arrhythmias?

Since there is no scar on typical patients with idiopathic outflow tract arrhythmias, the main tool prior to the procedure is to attempt epicardial origin location on the electrocardiogram.

Several ECG characteristics have been suggested to recognize an epicardial origin of idiopathic outflow tract arrhythmias. Bazan and colleagues[3] reported that QRS duration was shorter in endocardial septal compared to endocardial and epicardium free wall during epicardial and endocardial pacing. However, there was no difference in QRS duration for endocardial and epicardial free wall sites. For RV anterior segments, a Q wave in lead I suggests epicardial origin. Additionally, a QS complex in lead V2 was observed more frequently at epicardial sites compared to endocardial.

Daniels et al associated the initial time of the QRS complex length to an epicardial origin of idiopathic tachycardia.[11] They created an index, named Maximum Deflection Index (MDI) measuring the time to maximum deflection and dividing it by the QRS duration (Figure 2). This index was longer for epicardial left ventricular arrhythmias compared to endocardial left ventricular, endocardial

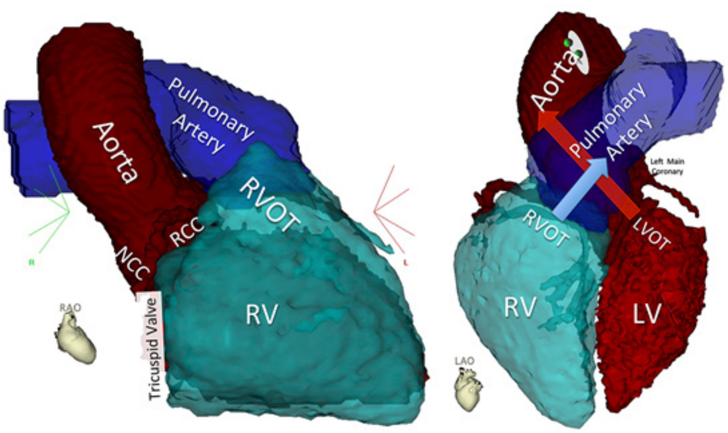

Figure 1: Anatomy of the outflow tract. CT scan 3D reconstruction showing the ventricles, outflow tract and the aorta and pulmonary artery. On the left panel, RAO view, and on the right panel, LAO. The pulmonary artery and the RVOT lie anterior to the LVOT and the aorta. The RV is located to the right, but when it advances into the RVOT and pulmonary artery, they are located on the leftward to the LV, LVOT and aorta. In the opposite, the LVOT and aorta are located rightward to the middle axis of the heart. There is an intimate relationship between the right coronary cusp and the septal aspect of the RVOT. The left main and coronary sinus (not shown in this model) locate to the left of the RVOT and LVOT and anterior to the LV. CT: Computerized tomography; RAO: right anterior oblique; LAO: left anterior oblique; RVOT: right ventricular outflow tract; LVOT: left ventricular outflow tract; RV: right ventricle; LV: left ventricle; NCC: right coronary cusp; NCC: non-coronary cusp

right ventricular and aortic sinus of Valsalva. They identified a cut off value of 0.55, suggesting epicardial arrhythmias. All epicardial arrhythmias were mapped from the epicardial venous system.

One of the reasons for failure of RVOT arrhythmias ablation is their localization on the aortic cusps. The leading ECG characteristic suggesting this localization is an earlier transition V1/V2 instead of V3/V4 typical for RVOT. Additionally, R wave duration index, calculated as a percentage by dividing the QRS complex duration by the longer R wave duration in lead V1 or V2, and R/S wave amplitude ratio in leads V1 and V2 were longer for aortic sinus of Valsalva premature ventricular complexes.[12]

Tada et al showed that LV epicardial arrhythmias had a greater R amplitude wave in inferior leads. Lead I had an S wave and an rS(s) or QS pattern. The Q wave amplitude in aVR and aVL was greater in the LV-epi group than RV and LV endocardial and the Q wave amplitude in aVL was deeper than in aVR in the LV-Epi group (Figure 3). The precordial R wave transition occurred from V2 to V4. In the LV-Epi group, there was a distinct R(r) wave in V1, and its amplitude was significantly greater than in the RV-endo group. There was a distinct S wave in V1 and V2 in the LV-Epi group, and the S wave amplitude in V2 was significantly greater than in the LV-Endo group.[13]

When the R/S transition is V3, the PVC can be ablated in the RVOT in most of the cases (Figure 4), but sometimes the LVOT, aortic sinus of Valsalva, pulmonary artery and epicardial ablation by coronary sinus or via pericardial puncture mapping are necessary for a successful ablation. Tanner and colleagues[5] proposed a stepwise approach for PVC with V3 transition. They suggest starting on the most common site that is the RVOT, followed by the pulmonary artery. If the mapping indicates a focus outside the RVOT and the pulmonary artery, mapping of the coronary sinus may add useful information of the left side and epicardial origin of PVC.

All criteria for epicardial origin of RVOT identification have limitations; one important one is that small changes in ECG electrode placement markedly alter QRS morphology of outflow tract arrhythmias. Superior displacement of V1 and V2 reduced R wave amplitude and led to a decreased R/S ratio, while inferior displacement of leads V1 and V2 resulted in an increased R wave and R/S ratio. Additionally, anterior displacement of the arm leads from shoulders to chest resulted in the reduction in the R wave amplitude in lead I.[14]

How To Ablate Epicardial Origin Of Outflow Tract Tachycardias

The approach we use in our institution for ablation of RVOT tachycardias starts with placing a decapolar catheter advanced in the coronary sinus as close as possible to the Interventricular Anterior Vein, especially in cases where there is large R waves in leads V1 and V2. If there is very early activation on the coronary sinus then we do a quick activation mapping on the right outflow tract to identify if there is early activation on the right side. If there is an inadequate signal on the right side then we perform activation mapping on the aortic cusps and on the left ventricular outflow tract. Additionally, we perform activation mapping on the coronary sinus. Once all maps

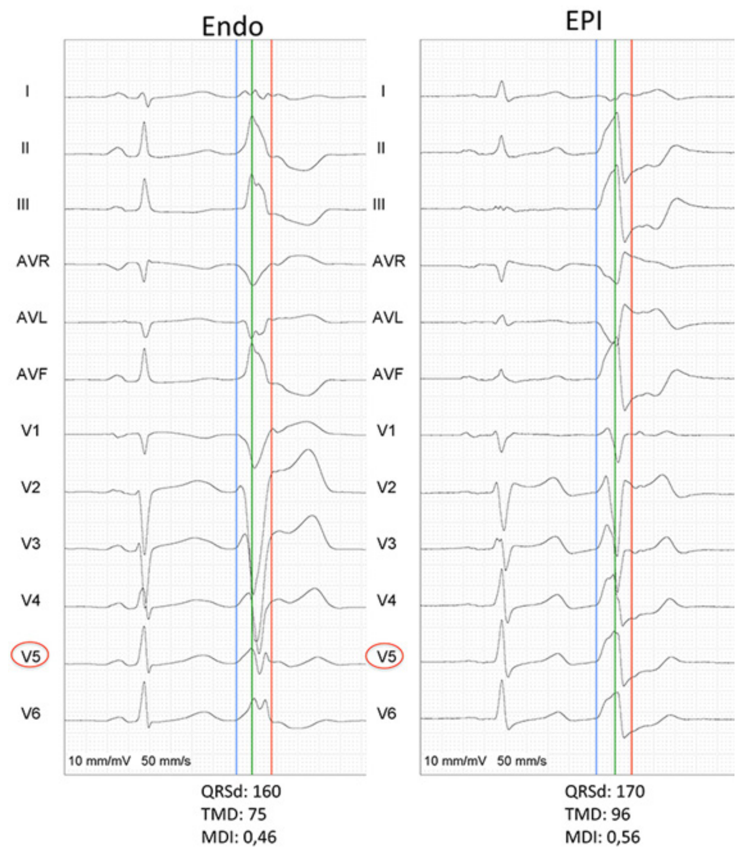

Figure 2: The measurement of the MDI (maximum deflection index) (11) on the left panel, endocardial PVC. The time to the maximum deflection is 75ms measured on V5 and the duration of the QRS 160ms. The MDI is 0,46, suggesting non-epicardial PVC. This PVC was succesfully ablated on the septal RVOT. On the right panel is epicardial PVC. The time to maximum deflection is 96ms measured on V5 and the duration of the QRS is 170ms. The MDI in this patient is 0,56 suggesting epicardial PVC that was ablated succesfully on the coronary sinus

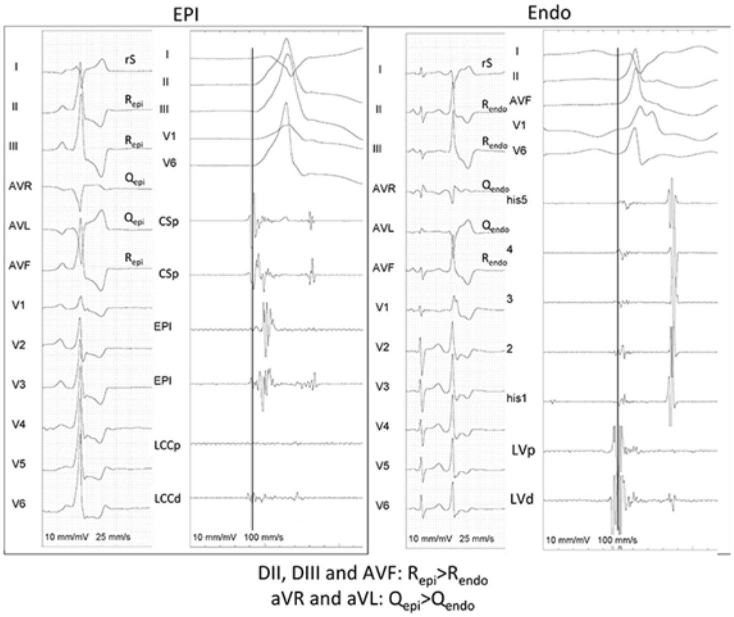

Figure 3: Analysis of PVC morphology to identify epicardial or endocardial origin. On the left panel, the PVC has rS on DI, large R wave on inferior leads and deep Q wave in aVR and aVL suggesting epicardial origin. This PVC was successfully ablated on the epicardium through the Left coronary cusp. Epicardial subxyphoid mapping showed also an early activation. On the right panel, endocardial PVC. There is rS, with R wave larger than the epi. Additionally, R waves on inferior leads have less amplitude than the R on the epicardial example as well as Q wave in aVR and aVL. This PVC was ablated on the infra-valvar lateral region

are constructed we compare the activation between all the maps. If there is earlier (at least 20ms from the QRS) or isochronic activation on the right side, than we perform RF ablation on that surface. For non-irrigated catheter we use 50W 60C settings and for irrigated 30W and 43C. If there is no early activation or ablation failed, we position the ablation catheter on the best site on the coronary sinus and then perform coronary angiography. Most of the time, the close relation of the coronary artery with coronary sinus is a limitation for ablation (Figure 5). If there is a safe distance from the artery we perform ablation using an irrigated tip catheter with the power of 20W, temperature limited to 43C and the impedance cut off is turned off. Cryoablation could be an alternative when the target on coronary sinus is very close to the artery.[15] If there is close relation of the coronary sinus spot and the coronary artery we search for adequate early signals on the sinus of Valsalva or just infravalvar on the LVOT. Is it of great importance when ablating above the aortic valve to be aware of the coronary artery ostium distance from the ablation catheter.

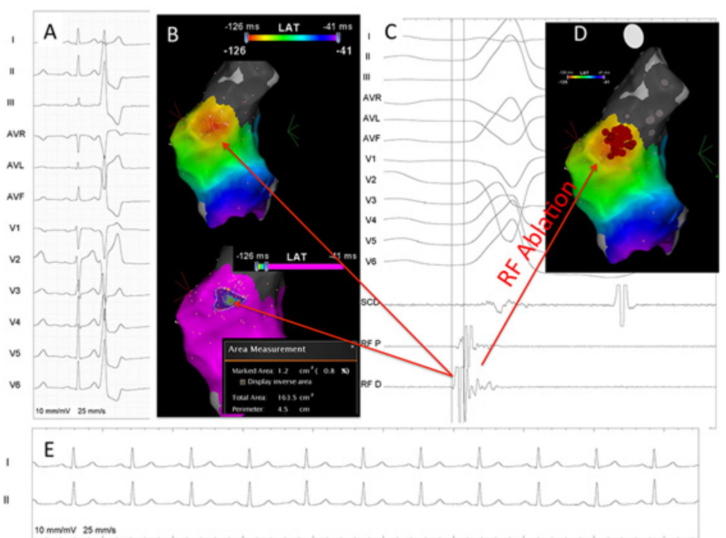

Figure 4: Outflow PVC with V3 transition successfully ablated on the RVOT. Panel A shows the inferior axis and morphology of left bundle brunch block and V3 transition. Electroanatomic (Panel B) and electrophysiological mapping (Panel C) showed earliest activation on the postero-septal aspect of the RVOT. The area of the earliest 10ms on the electroanatomical mapping is 1.2cm2. RF applications (Panel D) were performed on the area of earliest activation with complete elimination of the PVC (Panel E)

Asirvatham et al presented a series of 3 patients with the earliest activation site at coronary artery ostium, one at left coronary artery and two at the right coronary artery. They ablated these arrhythmias with the guidance of intracardiac echocardiogram targeting the aortic root (1 patient) or the right coronary cusp (2 patients) isolating the focus of origin.[16]

Our group reported many years ago on a patient where epicardial LVOT PVC was successfully ablated from the left atrial appendage.[17] In a second patient ablation was attempted but the patient presented LAA perforation necessitating open-chest surgery, so we abandoned such approach.

Reddy et al presented a series of 4 patients with previous ablation failure that was successfully ablated with bipolar RF ablation.[18] The catheters were positioned on the septal aspect of the RVOT and on the right or left coronary cusp. Ablation was successfully in two patients, in one there was a transient suppression of PVC and in other only rare PVC persisted after ablation.

Sometimes direct epicardial mapping using subxiphoid puncture is necessary (Figure 6). Schweikert and cols presented a series of 48 patients with subxiphoid access mapping for refractory arrhythmias, in which 20 patients had symptomatic PVC or sustained VT with normal hearts. Seventeen were located on the LV and only three on the RV. In 9 patients the arrhythmia was successfully ablated from the epicardial surface and in another 6 the earliest activation was

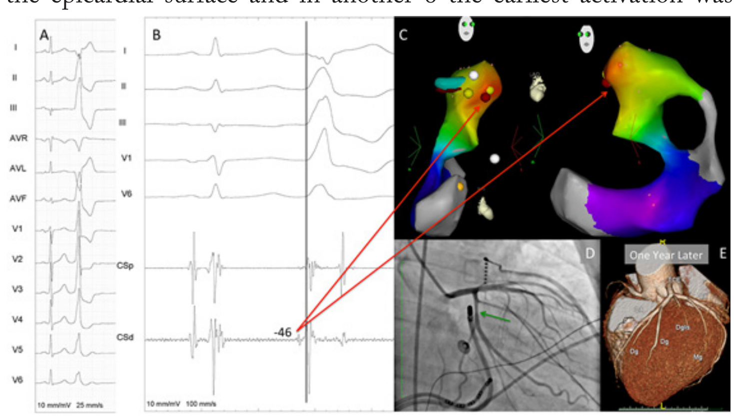

Figure 5: Premature ventricular complex that was successfully ablated on the coronary sinus. A panel shows the PVC morphology, with R waves from V1 to V6 and QS morphology on DI. Panel B and Panel C show earliest ventricular activation on anterior aspect of the coronary sinus. Early after RF ablation we performed a coronary angiogram showed on Panel D presents a narrowing of the left circumflex artery (green arrow). A coronary CT scan performed one month (not shown) and one year after ablation (Panel E) showed no lesion on left circumflex artery

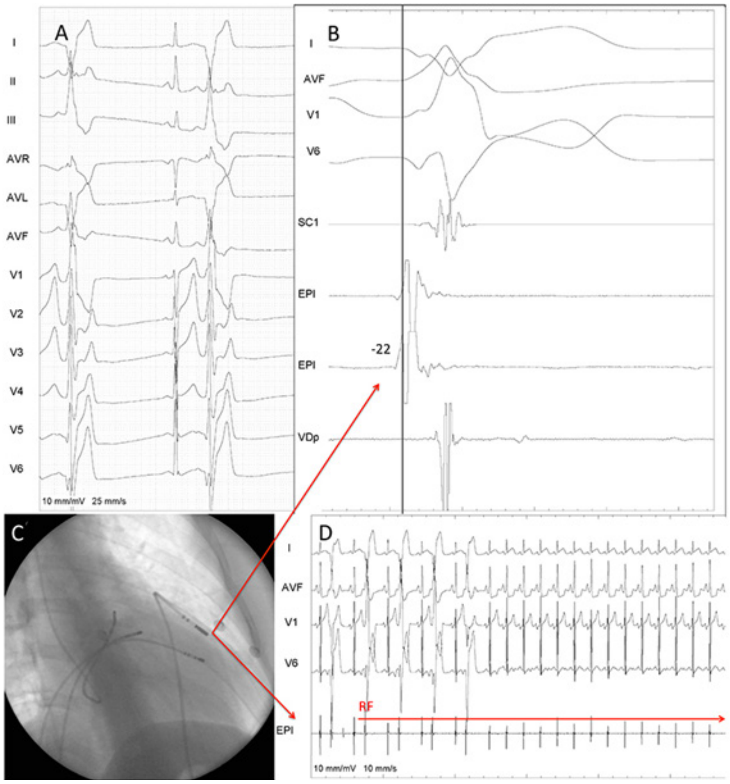

Figure 6: A 21-year-old male patient with very frequent PVC (31%) and mild LV dysfunction (EF: 45%) with no scar on MRI. There were 3 previous ablation failures. Panel A shows PVC morphology, with QS on DI, large R wave from V1 to V4, but with Q wave on V6. Epicardial subxiphoid mapping (Panel B) showed an early activation on the anterior LVOT (but not close to the mitral annulus) (Panel C). RF ablation performed on the earliest ventricular activation (Panel D) on the epicardial surface quickly suppressed PVC with no recurrence. There was a transient ST segment elevation on lead D1, but coronary angiogram was normal

on the epicardium, but ablation failed epicardially or was successful on the endocardial or the coronary cusp.[19] Yamada et al, presented a case report of a patient successfully ablated from the epicardium, where the best early activation time was 24ms on the endocardium and 48ms on the epicardium.[20]

One major limitation for subxiphoid epicardial ablation is the presence of the coronary arteries and the epicardial fat. Most of the idiopathic ventricular arrhythmias have a perivascular distribution.[11] In the epicardial surface close to the epicardial vessels there is a thick epicardial fat layer covering it, this fat limits mapping and adequate tissue lesion creation by RF. Another issue related to the coronary arteries is that the myocardium of PVC origin can be located underneath the arteries and could remain intact after ablation. Additionally, direct RF ablation on the artery could lead to artery lesion or even occlusion.[21] When ablating epicardial arrhythmias, phrenic nerves must be mapped by high voltage pacing. If there is phrenic nerve capture then the placement of an intrapericardial balloon close to the phrenic nerve during ablation could avoid damage to this structure.[22]

We do not recommend an epicardial approach on the first procedure for idiopathic outflow tract VT arrhythmias.[23] In our institution we performed subxiphoid epicardial mapping for 8 patients with idiopathic outflow tract arrhythmias that had failed to a median of 1 (range: 0 to 3) procedure prior to ablation. The median age was 32.5 years old (range 1 to 46) and 7 patients were male. Epicardial subxyphoid ablation was successful in two patients, one of these patients is presented on Figure 6; in 4 an additional endocardial mapping was successful (1 RVOT, 1 LCC, 1 RCC and 1 LV infra-valvar). One patient that epicardial subxyphoid ablation was successful had arrhythmia recurrence. There were 2 complications; one was transient ST segment elevation during ablation, with subsequent coronariography with no lesions and other patient that had hemopericardial with the need for open chest surgery, with the patient presenting complete recovery.

Conclusion

Idiopathic outflow tract arrhythmias are frequently ablated successfully from the right outflow tract, but sometimes their origin is on the left ventricular outflow tract, on aortic cusps or on the epicardial surface of the ventricles. When the arrhythmias are not located on the RVOT, the success of ablation decrease. Early transition of precordial leads to QS complexes and slow initial activation of the QRS suggests non-RVOT localization of the arrhythmias. Therefore, successful ablation may require aortic cusps, LVOT and great cardiac vein explorations in some patients. Epicardial arrhythmias can be ablated from the coronary venous system, but sometimes epicardial subxyphoid mapping can be necessary.

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