Epicardial Ventricular Tachycardia Ablation: Clinical Practice and Recent Developments

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INTRODUCTION

Mapping and radiofrequency (RF) catheter ablation of ventricular tachycardia (VT) is a demanding procedure, with variable success rates (1). The presence of deep subendocardial or epicardial re-entry circuits is regarded as one of the reasons of failure of endocardial ablation, and these circuits have been acknowledged in ischemic and non-ischemic dilated cardiomyopathy (CMP), other types of CMP and especially in arrhythmogenic right ventricular cardiomyopathy (ARVC).

The significance of epicardial VT circuits was brought to light in Chagas’ disease, which characteristically results in epicardial involvement in approximately 70% of patients (2). A recent study found one third of VTs to be epicardial in origin among patients with nonischemic CMP, about double the incidence among those with ischemic heart disease (3). Mapping and ablation of these epicardial circuits is quite exigent. Although coronary veins can be used to perform epicardial mapping, the manipulation of the catheter is strictly limited to the anatomical distribution of these vessels. Thus, the subxiphoid percutaneous approach to the pericardial space is the only technique that allows extensive, unhampered mapping of the epicardial surface of both ventricles.

Selection criteria/parameters

It is ambiguous whether one should use this approach only after an endocardial VT ablation failure or when the ECG of clinical VT suggests an epicardial origin. Consequently, multiple published ECG criteria may be used to predict epicardial VT origin. A maximal deflection index (MDI, the longest precordial lead onset-peak time divided by total QRS duration) of 0.55 or more is reported to identify idiopathic epicardial VT with high sensitivity and specificity (4).

Three other criteria that have been reported to predict epicardial VT origin in a population with structural heart disease are: i) a pseudo–delta wave > 34 msec (earliest ventricular activation to the earliest fast deflection in any precordial lead), ii) an intrinsicoid deflection time > 85 msec (earliest ventricular activation to the peak of the R wave), and an RS complex duration > 121 msec (earliest ventricular activation to the nadir of the first S wave in any precordial lead) (5). QRS duration has been found to be considerably wider among patients with epicardial VT, though a specific cut-off value has not been determined. However, QRS duration >200 msec is highly suggestive of epicardial origin (4).

The epicardial approach could also be an option in patients who have a clinical VT of left ventricular origin and have thrombus in the left ventricular cavity or metallic prostheses in either the aortic or mitral valve. Magnetic resonance imaging may also be helpful in identifying an epicardial substrate in cardiomyopathies (6).

The epicardial ablation procedure

Accessing the pericardial space in the absence of pericardial fluid is a challenging task. A specific technique of percutaneous access to the pericardium has been described by Sosa et al. (7) and is adopted with some variation by electrophysiologists performing such procedures. With the patient under conscious sedation, the pericardium is accessed via a percutaneous subxiphoid puncture, using an epidural needle.

Once in the pericardial space, atrial and ventricular surface mapping can be performed. Atrial surface mapping can be limited by the normal pericardial reflections and by the irregular atrial anatomy (left and right atrial appendage). In contrast, ventricular surface mapping can be performed without any significant difficulty.
Different types of ablation catheters can be used in the pericardium (solid tip, internally and externally irrigated—tip catheters). Catheter type as well as RF ablation parameters used in the pericardium may vary, depending on centre preference. However, in patients with epicardial VT, the target for ablation is selected with exactly the same method and criteria as the endocardial target is selected in endocardial VT ablation. Electroanatomical mapping is performed, and the VT substrate and circuit(s) are defined using voltage, activation, entrainment and pace-mapping.

The percutaneous approach to the pericardial space can be difficult in patients who have undergone cardiac surgery in the past or after myocarditis, since adhesions may prevent access or limit mapping. In post-surgical patients, adhesions are mostly located at the anterior portion of the heart; therefore, the puncture should be directed towards the diaphragmatic area.

Coronary angiography analysis prior to energy delivery is reasonable and preferable, so as to select a safer area for RF application, since there is a significant risk for coronary artery lesions in case of RF application to epicardial coronary vasculature. It is considered that the base, the anterior and posterior septum areas are the more dangerous zones, while coronary vessels are usually absent from the RV free wall.

**Complications**

Epicardial mapping and ablation is associated with major and minor complications, acute or delayed; death, RV puncture, cardiogenic shock, intrapericardial bleeding and tamponade, hepatic bleeding, coronary artery stenosis, phrenic nerve paralysis, inadvertent entry of the guidewire into the pleural space, major pericardial reaction. Chest pain is present after almost all procedures (related to pericardial inflammation), usually requiring treatment with a non-steroidal anti-inflammatory drug, particularly in order to avoid a major pericardial reaction, which could rarely lead to constrictive pericarditis[^3][^8].

**Studies on epicardial VT ablation**

The largest study, to date, on epicardial VT catheter mapping and ablation is a multicenter study, consisted of a referral patient population with ischemic and non-ischemic dilated CMP; ARVC and other CMPs (valvular, sarcoidosis, noncompaction of the left ventricle, etc) who underwent VT ablation at 3 tertiary care electrophysiology centres between 2001 and 2007[^10]. This retrospective study included 913 VT ablations; 156 procedures (17%) in 134 patients involved epicardial mapping and/or ablation.

The majority of the patients (86%) had prior failed endocardial VT ablation attempts. Percutaneous subxiphoid epicardial access was achieved in 136 procedures (about 90%). Failure of a percutaneous approach (n=15) was mostly associated with a history of prior cardiac surgery (n=11) or pericarditis.

A surgical subxiphoid or lateral thoracotomy approach was performed under general anesthesia in the remaining patients. The highest prevalence of epicardial VT was observed in patients with the diagnoses of ARVC (41%) and non-ischemic dilated CMP (35%), followed by patients with ischemic heart disease (16%). A total of 14 procedure-related major complications (9%) were observed acutely or before discharge. Complications related to the epicardial approach (5%) included 7 episodes of epicardial bleeding as well as 1 coronary stenosis.

Overall, the findings of this study are consistent with prior observations. Soejima et al.[^9] found epicardial ablation necessary in 28% of patients with dilated CMP, and Sosa et al.[^10] found epicardial VTs in 23% of patients with VT late after myocardial infarction. It appears that epicardial involvement may be present to various degrees in a significant minority of patients with VT in different types of CMP, especially when a prior endocardial ablation approach has failed. Garcia et al.[^11] discovered that the necessity of epicardial approach occurred in 13 of 33 patients (39%) with ARVC who underwent endocardial ablation. Schmidt et al.[^12] demonstrated that an epicardial VT substrate is present in three-quarters of patients with a previously failed endocardial VT ablation, and epicardial ablation is required for successful VT abolition in more than half of patients.

In non-ischemic dilated CMP, detailed epicardial mapping has highlighted more extensive confluent low-voltage areas compared with the endocardial surface, often situated over the basal lateral left ventricle near the valve annulus[^11][^12]. The low-amplitude electrograms recorded in these areas are typically >80 msec wide, split, and/or late, which facilitate the differentiation of scar from epicardial fat[^12][^13].

Da Silva et al.[^14] investigated the importance of the simultaneous epicardial and endocardial mapping of sustained VT in patients with nonischemic cardiomyopathy, mainly chronic chagasic cardiomyopathy. This strategy allowed a more accurate mapping and helped in the identification of extensive areas with fractionated electrograms and with pre-systolic activity, arrhythmogenic substrate for VT. Henz et al.[^15] observed the same in chagasic patients.

Unlike those with non-ischemic cardiomyopathy, patients with ischemic heart disease are inclined to have a larger endocardial than epicardial scar, usually restricted to a specific coronary vascular territory. Although there is a preference for a subendocardial location of the VT substrate, the frequency of epicardial circuits may be high, especially in patients with old inferior infarctions[^10].

In patients with ARVC, the presence of sizeable epicardial low-voltage areas, often with fractionated and late electrographic recordings, has been recognised. The epicardial scar is considerable and consistently larger than that on the endocardial surface. The epicardial focal points targeted for successful catheter ablation are also commonly positioned outside the endocardial defined scar[^11]. In a very recent study, Bai et al.[^16] suggest that a dual, endo-epicardial based ablation strategy achieves higher long-term freedom from recurrent ventricular arrhythmias off anti-arrhythmic therapy in patients with ARVC when compared to endocardial-alone ablation.

Catheter ablation has been shown to be an efficient therapy for patients with idiopathic VT. However, sporadic patients have been reported in whom such an arrhythmia could not be ablated from the ventricular endocardium or from the aortic cusps. Often under-recognised, the prevalence of an epicardial origin in idiopathic VT may be as high as 9%[^4]. Catecholamine sensitivity and triggered mechanism appear to be common features among these VTs, frequently arising from areas adjacent to epicardial coronary vessels[^4][^17].
In conclusion, further studies are necessary to define the role of epicardial ablation as patient populations differentiate and technologies continue to evolve. Furthermore, the development of specific, sophisticated equipment for percutaneous pericardial access, as well as epicardial mapping and ablation, is essential. Other advances in technologies and refinement in imaging and ablation techniques might allow us to better illustrate the ventricular substrate and diminish the risks of the procedure.

**In Brugada syndrome, the underlying electrophysiological mechanism that causes the abnormal ECG pattern and ventricular tachycardia and/or ventricular fibrillation (VF) remains obscure. Nademanee et al (18) studied patients with typical type 1 Brugada ECG pattern and inducible VT/VF. They found to have abnormal low voltage, prolonged duration, and fractionated late potentials clustering exclusively in the anterior aspect of the RVOT epicardium. Ablation at these sites eradicated VT/VF inducibility and rendered normalisation of the Brugada ECG pattern in 89% of patients, both during electrophysiological studies as well as spontaneous recurrent VT/VF episodes.**

Combined epicardial and endocardial mapping and ablation have also been evaluated recently in hypertrophic cardiomyopathy (HCM) patients. Dukkipati et al. (19) generated epicardial and endocardial ventricular 3D voltage maps in 10 patients with monomorphic HCM-related VT, and identified ablation sites using a combination of entrainment, activation, late/fractionated potential, and pace mapping. Electrophysiologically-identified epicardial scar was present in 8 (80%), while endocardial scar was present in 6 (60%) patients. During a median follow-up of 37 months, the freedom from recurrent implantable cardioverter-defibrillator shocks was 78% in those who underwent ablation, rendering combined epicardial and endocardial mapping and ablation a feasible and efficacious option for monomorphic VT in highly selected patients with HCM.

**Indications for epicardial mapping and ablation**

Based on the aforementioned data and our experience with patients with non-ischemic cardiomyopathy (Figure 1), patients should be selected for epicardial mapping and ablation taking into account the following key points:

a. The substrate of the cardiomyopathy. The highest prevalence of epicardial VT is observed in patients with ARVC and non-ischemic dilated CMP.

b. The ECG of clinical VT. An ECG suggestive of an epicardial origin of VT could lead to epicardial mapping.

c. Failure of previous endocardial ablation could reflect the presence of an epicardial origin.

d. Epicardial access is successfully achieved in the majority of patients via a percutaneous subxiphoid approach. The majority of failures of percutaneous subxiphoid punctures are observed in patients with previous cardiac surgery.

e. High-output pacing and coronary angiography should be performed during epicardial mapping and prior to energy delivery, in order to define the course of the phrenic nerve and to confirm the absence of coronary arteries near the ablation site.

f. Simultaneous epicardial and endocardial substrate mapping could provide additional information than each mapping alone and accomplish higher radiofrequency catheter ablation success rates.


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