Epicardial ventricular tachycardia

Epikardiyal ventrikül taşikardisi

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Summary– In ventricular tachycardia (VT) arising in the myocardial tissue, the site of origin may be the endocardium, mid-myocardium or epicardium. The incidence of epicardial origin varies with the underlying heart disease, and is probably not more than 20% in ischemic heart disease and higher in non-ischemic cardiomyopathies. Percutaneous subxiphoid access to the pericardial space has enabled a non-surgical approach to catheter mapping and ablation of epicardial VT. Several algorithms are available for electrocardiographic recognition of epicardial origin. Idiopathic epicardial VTs are rare but may be curable by catheter ablation. The electrophysiologic principles guiding the mapping and ablation of epicardial VTs are similar to those used for endocardial VTs, but the biophysics of energy delivery may be different. Complications of the epicardial approach are also different from those of endocardial ablation, and specific precautions have to be taken to protect the coronary arteries and phrenic nerves and to avoid pericardial tamponade.

Özet– Ventrikül taşikardilerinin (VT) odak noktası endokardta, epikardta, veya nadiren ventrikül duvarının içinde olabilir. Epikart dokusunda oluşan VT'lerin prevalansı altta yatan organik kalp hastalığının türüne göre değişir. İskemik kalp hastalığı ve VT'si olan hastalarda bu prevalansın %20'nin altında, iskemik olmayan kardiyomiyopatilerde ise daha yüksek olduğu saptanmıştır. Perkütan teknik ile perikardın içine girilerek yapılan elektroanatomik haritalama epikardiyal VT'lerin kateter ablasyon yöntemi ile tedavilerini olanaklı hale getirmiştir. Epikardiyal VT'lerin 12 derivasyonlu EKG ile tanısı için birden fazla kılavuz yayımlanmıştır. İdiyopatik olan epikardiyal VT'ler nadir olmalarına karşın ablasyon ile kesin tedavileri olanaklıdır. Epikardiyal VT'nin kateter ablasyonu için kullanılan teknik ve hedefler endokardiyal taşikardilerde uygulananlardan farklı değildir. Ancak epikardiyal kateter ablasyonlarının komplikasyon riski daha yüksektir ve özellikle koroner arterlerin ve frenik sinirin korunması ve perikart tamponadının önlenmesi için tedbirler alınması gerekir.

Ventricular tachycardias (VTs) are cardiac arrhythmias originating in the ventricular myocardium or the His Purkinje network. The His Purkinje fibers are anatomically located only in the subendocardium and endocardium. By contrast, the pathological substrate that gives rise to VTs originating in the ventricular myocardium may reside in the subendocardial layer, subepicardial layer, or even in a mid-myocardial site. The subendocardial substrate of VT has been a subject of intense study over the past 30 years, especially for VT of ischemic heart disease.[1-4] Even in the early days of electrophysiological map-guided surgical left ventricular (LV) aneurysmectomy, there were reports of VTs of LV epicardial origin.[5] There were also isolated case reports of VT termination by radiofrequency (RF) energy application in the coronary sinus, suggesting an epicardial site of origin.[6]

Epicardial VT became a major topic of electrophysiological investigation with the advent of percutaneous access of the pericardial space 17 years ago.[7] As with all VTs, epicardial VT may also be categorized as idiopathic VT occurring in the absence of any recognizable structural heart disease and VT of organic heart disease. Needless to say, an "idiopathic" VT mechanism may coexist with organic heart disease, and therefore these categories are not mutually exclusive. The following

Abbreviations:	
ARVC	Arrhythmogenic right ventricular cardiomyopathy
AV	Atrioventricular
CABG	Coronary artery bypass grafting
ECG	Electrocardiographic
ICD	Implantable cardioverter-defibrillator
LV	Left ventricular
LVAD	Left ventricular assist devices
MI	Myocardial infarction
RF	Radiofrequency
RV	Right ventricular
VF	Ventricular fibrillation
VTs	Ventricular tachycardias

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is an overview of the diagnosis, clinical importance, and treatment of epicardial VT.

Prevalence

The true prevalence of epicardial VT is not known exactly. A recently published multi-center European study estimated that 12% of the VTs, in patients with diverse clinical backgrounds, were of epicardial origin.[8] Another recently published series from three centers showed a 17% prevalence of epicardial site of origin, an estimate higher than the previous one.[9] It is important to underscore, however, that these estimates are derived from selected groups of cohorts referred to major centers for catheter ablation treatment, and therefore may not accurately reflect the true prevalence of epicardial VT. What is clear is that the ratio of endocardial to epicardial site of origin of VT is markedly influenced by the underlying heart disease. The estimated prevalence of epicardial origin in idiopathic VT is around 10%, and conversely, 22% of the catheter ablations are for idiopathic VT.[8,9] The majority of the VTs of chronic ischemic heart disease are subendocardial in origin. Sosa's initial study[10] reported that 7 of the 30 (23%) VTs in patients with chronic myocardial infarction (MI) had an epicardial site of origin, a figure that most probably overestimates the true prevalence. The European multi-center study estimated 16% as an upper bound for the prevalence of epicardial origin for VT in ischemic heart disease.[8] Soejima et al.[11] estimated the prevalence of epicardial VT as 25% in patients with dilated non-ischemic cardiomyopathy. Recent studies suggest a higher prevalence of epicardial origin, 35% in non-ischemic cardiomyopathy, and 41-53% in patients with arrhythmogenic right ventricular cardiomyopathy (ARVC).[9,12] The highest prevalence

of epicardial origin has been reported in patients with Chagas disease.[13]

Electrocardiographic recognition of epicardial VT

Several different algorithms based on electrocardiographic (ECG) QRS configurations have been proposed to distinguish epicardial VT from endocardial VT (Table 1). These algorithms have high sensitivity, high specificity and acceptable predictive accuracy, but none should be used canonically since exceptions always exist. When the initial activation starts at an epicardial site, the rapidly conducting His Purkinje system is not used immediately, and the intra-myocardial delay of conduction produces a slurred initial component of the QRS complex, manifesting a slow rate of rise in voltage before it reaches the intrinsicoid deflection. This is the ECG signature of epicardial VT, and pretty much the basis of all these proposed criteria.

The first systematic investigation of the ECG characteristics that may predict the epicardial origin of VT was undertaken by Berruezo et al.[14] in a very small group of patients with mixed ischemic and non-ischemic heart disease. This was a correlation study comparing the ECG characteristics of epicardial pace-mapping and VT. The authors used the term "pseudo-delta" wave to describe the slow rate of rise in QRS voltage during VT. A word of caution at this point: a "true" delta wave with positive concordance across the precordium is likely to be supraventricular tachycardia (SVT) with LV pre-excitation, especially in a young patient with no structural heart disease. On the other hand, the absence of atrioventricular (AV) dissociation does not rule out VT with a basal site of origin, since 25% of the

Table 1. Electrocardiographic parameters used to predict epicardial origin of ventricular tachycardia

Parameter	Favors epicardial site	Reference
Pseudo-Delta Wave	>34-75 ms	14, 18
Maximum Deflection Index (MDI)	>0.55	15
Intrinsicoid Deflection Time (IDT)	>85 ms	14
Shortest RS Complex Duration (SRS)	>121 ms	14
QRS Duration (QRSd)	Epicardial longer	14, 18
Q Waves in Lead I (QWLI)	+	18
Q Waves in II-III-AVF (QInf)	–	18
aVR/aVL Amplitude Ratio	Epicardial higher	17

VTs will not have evidence of AV dissociation on the ECG. Berruezo et al. found that a pseudo-delta wave >34 ms and an intrinsicoid deflection time >85 ms in lead V2 had >83% sensitivity and >90% specificity for predicting an epicardial site of origin. Two years later, Daniels et al.[15] published a report on idiopathic epicardial LV tachycardia in 12 patients. None had a site of origin in one of the sinuses of Valsalva. These authors claimed that an index called maximum deflection index (MDI), calculated by dividing the earliest time to maximum deflection in any precordial lead by the total QRS duration, had the highest sensitivity and specificity, and identified a LV epicardial site of origin with 100% sensitivity and 98% specificity. At the same time, Bazan et al.[16] published a report describing their findings in several patients with epicardial idiopathic RV outflow tract tachycardia. The authors noted that the previously published Berruezo criteria did not apply to the RV, and at the RV outflow tract, they could not identify any ECG features that reliably distinguished epicardial from endocardial VT origin. The same authors investigated the ECG features that might predict the epicardial origin of left VT in patients with non-ischemic heart disease.[17] More recently, Vallès et al.,[18] from the same institution, published a report on their updated criteria to identify epicardial VT in non-ischemic cardiomyopathy. They devised a four-step algorithm that includes the presence of q waves in inferior ECG leads favoring an endocardial site, and the presence of q waves in ECG lead I favoring an epicardial site of origin. Finally and most importantly, a recent study by Martinek et al.[19] evaluated a long list of ECG criteria, previously proposed to predict the epicardial site of origin in idiopathic and non-ischemic VT, for their ability to predict the epicardial site of origin in VT of ischemic heart disease. The study found that none of these criteria identified ischemic VTs that required ablation on the epicardium.

In summary, despite the high sensitivity and specificity of these algorithms, none is completely without pitfalls and there is no single ECG finding that can serve as the gold standard for epicardial VT applicable to all cardiac conditions. Therefore, the following proposed criteria should be taken as general guidelines rather than canonical rules. If VT is known to be idiopathic and the surface ECG suggests LV origin, then MDI (see above) is probably the best discriminator. If VT is idiopathic and originates in the RV, then absence of any r wave (only q or qs) in the ECG lead,
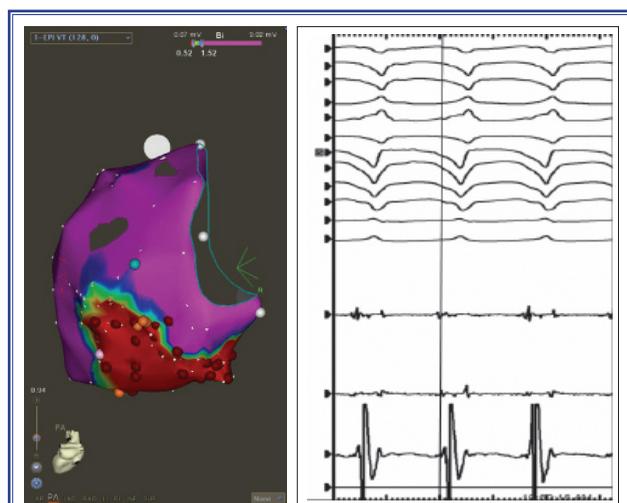
which best reflects the electrical activity at the site of origin, may be used. In non-ischemic cardiomyopathy, the absence of q waves in inferior leads, the presence of q waves in ECG lead I, and a pseudo-delta wave >75 ms may be used as the best discriminators. In patients with ischemic VT, especially in those with previous transmural MI, q wave criteria should not be used for obvious reasons. As to the other indices mentioned above, there is too much overlap between the ischemic epicardial and ischemic endocardial VTs to single out any one of them as the most reliable discriminator. Finally, the sensitivity and specificity of any ECG criteria will be affected in the presence of antiarrhythmic drug therapy.

Post-MI epicardial ventricular tachycardia

The first report of post-MI epicardial VT was in 1984 and described intraoperative data in four selected patients with ischemic heart disease undergoing surgical coronary revascularization and map-guided aneurysmectomy.[5] In a later report, Kaltenbrunner et al.[20] reviewed their intraoperative VT mapping experience in post-MI patients, and reported completely mapped epicardial circuits in 14% of the patients. The first report of percutaneous catheter mapping of post-MI epicardial VT appeared years later in 2000.[10] Seven of 30 VTs in this highly selected group of 14 patients had an epicardial site of origin. Of note, the authors described mid-diastolic potentials and fractionated low-amplitude local electrograms recorded from the epicardium, very similar to the local endocardial electrograms that had been observed for years in patients with chronic ischemic heart disease. Studies in patients who underwent both endocardial and epicardial catheter ablation for VT have shown that there are certain endocardial local electrogram characteristics that strongly suggest an epicardial site of origin.[21] Among these is a pattern of a far-field electrogram followed by a near-field electrogram showing earliest endocardial activation. The results from the multicenter safety study with a much larger cohort (n=722) showed 51 (16%) epicardial VTs in patients with ischemic cardiomyopathy, a figure that may represent a lower bound.[9] In a more recent article by Tung et al.,[22] of the 95 patients undergoing epicardial catheter mapping and ablation for VT, one-third had ischemic cardiomyopathy. Once again, these figures are reported from large referral centers and the estimations of incidence will always be subject to referral bias.

Epicardial ventricular tachycardia in non-ischemic cardiomyopathy

Ventricular tachycardias arising in non-ischemic dilated cardiomyopathy have a higher incidence of epicardial origin compared to the VT of ischemic heart disease. Conversely, the proportion of patients undergoing epicardial catheter mapping and ablation varies from 28% to 47%.[8,19] In 28 patients with non-ischemic dilated cardiomyopathy and VT, reported by Soejima et al.,[11] seven patients had an epicardial site of origin, and all epicardial VTs had reentrant mechanisms, except one VT that had a focal mechanism. Of note, an isthmus, similar to the subendocardial pathway described previously in ischemic cardiomyopathy, was identified by entrainment mapping in the majority of the VTs. A report by Cano et al.[23] further elaborated on the arrhythmogenic substrate of epicardial VT in non-ischemic cardiomyopathy by describing confluent areas of low-voltage, fractionated local electrograms, predominantly distributed over the basal lateral LV and peri-mitral region. Similar findings from a patient undergoing epicardial catheter ablation in our laboratory are shown in Fig. 1. In a more recent study, Hutchinson et al.[24] showed that endocardial unipolar voltage mapping was useful in indirectly identifying epicardial substrate for VT in patients with non-ischemic cardiomyopathy.

Epicardial ventricular tachycardia in ARVC

Arrhythmogenic right ventricular cardiomyopathy is a congenital cardiomyopathy with an autosomal dominant inheritance, except for the Naxos disease variant. The pathological substrate for VT in ARVC is the fibro-fatty infiltration of the myocardium, and these progressive histological changes result in marked electrophysiological abnormalities conducive to reentry. The incidence of VT ranges from 25% to 100% depending on the extent of the pathological changes. Garcia et al.[25] published their results from 13 patients with ARVC and VT in whom VT had recurred following a previous endocardial catheter ablation. Abnormal low-voltage, fractionated electrograms were far more extensive in the epicardium compared to the endocardium, and VT could be induced reproducibly by programmed cardiac stimulation. In a more recent report, Bai et al.[12] reported their catheter ablation results in 49 patients with ARVC and VT.[12] In 26 patients, extensive simultaneous endocardial and epicardial catheter mapping and ablation were carried out. In this group, the long-term freedom from recurrent VT and implantable cardioverter-defibrillator (ICD) therapies was significantly superior compared to the group that had endocardial ablation alone, suggesting an epicardial site of origin for many of these VTs.

Idiopathic epicardial VT

The ECG of the most common type of idiopathic LV tachycardia usually manifests a pattern of right bundle branch mimicry and left axis deviation and results either from reentry in the left- sided fascicles of the His Purkinje network or originates in the papillary muscles. These VTs are by definition endocardial or subendocardial in origin. If we also exclude VTs originating in the sinuses of Valsalva as non-epicardial, we are left with a very small group of idiopathic epicardial LV tachycardias. The sites of origin of these VTs have been described in recent publications. Many of these tachycardias have their earliest activation sites in the coronary sinus, the great cardiac vein and the middle cardiac vein, as reported by Daniels et al.[15] and Doppalapudi et al.[26] Thus, these VT sites cluster either at the area of LV summit or the crux of the

Figure 1. Epicardial ventricular tachycardia (VT) in one of our patients with non-ischemic cardiomyopathy. Note the "pseudo-delta" wave in several ECG leads during VT, and the early abnormal, fractionated, low-amplitude local electrograms. Also shown is the electroanatomical catheter voltage map of the left ventricular (LV) epicardium during sinus rhythm in the same patient, with a confluent region (color coded with red) over the posterolateral basal LV where abnormal local electrograms, with amplitude <0.5 mV, are recorded.

heart. There are, however, also reports of idiopathic epicardial LV tachycardias with origins remote from vascular structures, where the earliest site of activation cannot be accessed via the coronary sinus and its branches.[27] Compared to the high incidence of epicardial site of origin in ARVC, the epicardial origin of idiopathic RV tachycardias is rarer. Bazan et al.[16] found an epicardial site of origin of idiopathic RV outflow tract tachycardia in 13 patients undergoing a repeat procedure for a previous unsuccessful endocardial catheter ablation. These investigators could not identify any reliable surface ECG configurations reliably predicting an epicardial site of origin during VT or pace mapping.

Epicardial ventricular tachycardia in other diseases

The incidence of epicardial VT in sarcoidosis is not known. The presence of inducible VT in patients with sarcoidosis has been used for risk stratification, but detailed reports of catheter mapping and ablation are not available. Judging from cardiac magnetic resonance imaging (MRI) localization of inflammatory lesions, many patients with sarcoidosis and VT may well have an epicardial focus. Even less is known about the site of origin of VT in amyloidosis and other infiltrative diseases. Until recently, the VT in Brugada syndrome had been considered a "channelopathy"-related ventricular arrhythmia arising in the absence of organic heart disease, but recent data from Dr. Nademanee's[28] laboratory have shown that an electrophysiologically abnormal substrate can be detected in the epicardium in the RV outflow tract. These areas have been targeted in the catheter ablative therapy of VT in Brugada syndrome.

Treatment: catheter ablation

No study to date has shown that there is a difference between endocardial and epicardial VTs in their response to antiarrhythmic drug therapy in any type of heart disease. Theoretically, to the extent that there are differences in wall tension, in coronary perfusion, and more importantly in action potential characteristics between the endocardium and the epicardium, there may be a difference in the effectiveness of pharmacologic therapy. However, in the absence of convincing data, there is no reason to base the choice of an antiarrhythmic drug on the site of origin of the VT

if the intention is pharmacologic therapy; rather, the choice should be based on the presumed underlying mechanism, e.g. reentry, triggered activity, and most importantly, on safety concerns.

By contrast, the site of origin of VT is of paramount importance when catheter ablation is the preferred method of treatment. Many patients with organic heart disease, who have epicardial VT, also have VTs that arise in the endocardium or the subendocardium. Therefore, in patients with organic and especially progressive heart disease, recurrence of VT after an epicardial catheter ablation should not be equated with recurrence of epicardial VT. Rather, each epicardial procedure should incorporate simultaneous endocardial as well as epicardial catheter mapping. Idiopathic epicardial VT, on the other hand, is far more likely to have a single mechanism and a unique site of origin, and post-ablation recurrent VT is more likely to be recovery of the previously targeted mechanism rather than an entirely new one.

Methodology and safety concerns

The technical details of acquiring access to the pericardial space have been described in several previous publications.[7,13] We perform the epicardial ablation procedures under general anesthesia in our institution, although there are laboratories using only conscious sedation. Careful hemodynamic monitoring is critically important, especially if an open irrigation catheter system is used, to prevent excessive pericardial fluid accumulation. Pericardial fluid should be continuously or periodically aspirated and the input-output balance should be monitored to avoid pericardial tamponade. An anterior approach targeting an entry site in the anterior retrosternal pericardium or posterior approach targeting an entry site in the posterior or diaphragmatic pericardium may be used. The angle of entry is adjusted depending on whether an anterior or a posterior approach is preferred. The choice should be guided by the area of greatest interest based on ECG and previous endocardial catheter mapping data.

When epicardial catheter ablation is performed within the coronary sinus or its branches, there is a risk of injury to coronary arteries. Similarly, ablation within the pericardial space may injure epicardial coronary arteries resulting in thrombosis and MI. Therefore, coronary angiography is mandatory to determine the proximity of the ablation catheter to an epicardial

coronary artery branch. The phrenic nerves, especially the left one, also need protection. Pacing at high amplitude and watching for diaphragmatic capture should be performed before any RF energy delivery or cryoablation at sites anatomically close to the phrenic nerves.

In patients with ischemic heart disease, dilated non-ischemic cardiomyopathy, and ARVC, substrate mapping on the epicardial surface during sinus or paced rhythm may identify regions of abnormal local electrogram, which may be quite useful for identifying the site of origin of VT (Fig. 1b). During VT, such areas may display mid-diastolic potentials, and entrainment may be carried out to confirm their participation in the mechanism underlying the VT. Unlike endocardial mapping, however, the presence of epicardial fat may result in low amplitude local electrograms with low specificity. Capture threshold is high in such locations. The electrophysiological principles are the same as those that guide endocardial catheter mapping and ablation. If a hemodynamically tolerated VT is present, entrainment with concealed fusion during pacing within the reentry circuit, with a post-pacing interval matching the VT cycle length, is still the most reliable observation. If VT is not tolerated, surrogate methods targeting specific local electrograms, similar to those used during endocardial catheter mapping, may be used. In extreme cases, an intra-aortic balloon pump or an Impella device may be used for extra hemodynamic support during the procedure. In idiopathic VT, sizable epicardial areas with abnormal local electrograms do not exist. The best targets are the earliest pre-systolic local signals

during VT or during premature ventricular contractions manifesting the same QRS configuration as VT. The only surrogate technique that may be useful in idiopathic VT is pace-mapping at VT cycle length with exact reproduction of the VT QRS configuration in all 12 ECG leads.

Both RF energy and cryoablation have been used effectively for ablation of epicardial VT. In a multicenter safety study, 7% of the cases were performed by cryoablation, mainly due to the proximity of the ablation site to a coronary artery branch.[9] The biophysical features of epicardial RF energy application differ from the endocardial features since there is no blood pool acting as a heat exchanger. With the irrigated catheters, the power range varies from 20 to 40 W, and during energy delivery and the saline irrigation, the rate is set around 30 ml/min.

Complications

Complications of epicardial catheter ablation are rare when performed by an experienced team at large referral centers. They include the complications that occur during pericardial access, those that occur during mapping and ablation, and the post-procedural complications (Table 2). During subxiphoid transthoracic access, inadvertent puncture of an epicardial coronary artery or the RV may occur. In the absence of systemic anticoagulation, RV puncture may not be a serious complication provided that it is discovered early from the course of the guide wire, before the dilator and sheath are advanced. In the multicenter safety study, inadvertent RV puncture occurred in 17% of the patients.[9] The reported incidence of bleeding into

Table 2. Reported complications of epicardial catheter ablation

Complication	Reported incidence (%)
RV puncture without consequence	17
Hemopericardium	4.5-6.7
Pericardial tamponade	3.7-15
Pericardial effusion	34
Myocardial infarction	1
Coronary stenosis	0.6
Phrenic nerve palsy/paralysis	1
Pericarditis	0.8-2
Pulmonary embolism	1.3
Hepatic/intra-abdominal artery puncture	0.5-0.6

the pericardial space and pericardiac tamponade varies from 3.7% to 15%.[8,29] Procedure-related death has been reported.[29] Pneumopericardium may not be harmful, but may elevate the defibrillation threshold. Other structures that may be damaged during access include the left lobe of the liver, especially in patients with hepatomegaly, e.g. secondary to right heart failure, and an intra-abdominal artery, resulting in hemoperitoneum.[22,30]

The complications that occur during catheter mapping and ablation include pericardial effusion, coronary artery thrombosis, and injury to the phrenic nerves, esophagus, vagus nerve, and lungs. Pericardial effusion without hemodynamic consequences is quite common, and not considered a serious complication. The reported incidence of MI is 1%,[9] as is the incidence of phrenic nerve palsy.[9] The other complications mentioned above are quite rare.

Post-procedural pericardial pain is extremely common and usually self-limited. Delayed pericardial tamponade is a rare complication.[9] Severe pericarditis may also occur rarely, requiring prolonged treatment with anti-inflammatory drugs. Routine intrapericardial steroid infusion, as a preemptive measure to prevent pericarditis, is performed in many institutions, but its clinical benefit has not been proven.

Long-term efficacy of epicardial ablation

The long-term efficacy of epicardial catheter ablation for VT depends on the underlying heart disease, if any, and its rate of progression. This is no different than the earlier results observed with endocardial catheter ablation for VT. Many published studies have reported results obtained in select groups of patients, and the differences in long-term efficacy may reflect this selection bias. Grimard et al.[29] reported their long-term follow-up in 32 patients undergoing epicardial VT ablation. The procedure had been deemed immediately successful in 76% of the patients. During a mean follow-up period of 384 days, one-fourth of the patients had recurrent VT. One of the multicenter studies mentioned above reported a 71% freedom from VT during a mean follow-up period of 23 months.[9] In a European multicenter study, acute success was possible in 72% of the patients.[8] During a mean follow-up period of 17 months, VT recurred in 31%. There was no significant difference in the recurrence rate between the patients with ischemic car-

diomyopathy and those with idiopathic dilated cardiomyopathy. Of note, in this study, six patients died of "electrical storm" within 48 hours of the study. In a large series from a single center, Tung et al.[22] reported freedom from VT in 85% of the patients with ischemic cardiomyopathy who underwent hybrid endocardial-epicardial catheter mapping and ablation after a year, as compared to 56% observed in patients who had an endocardial procedure alone. Somewhat surprising was their finding that in patients with non-ischemic cardiomyopathy, there was no such difference in freedom from VT at 12 months between the group undergoing endocardial ablation alone and the group undergoing the hybrid procedure. Cano et al.[23] reported 78% freedom from recurrent VT at a mean follow-up of 18 months following epicardial catheter ablation in a small group of patients with non-ischemic cardiomyopathy. In patients with ARVC, clearly a progressive cardiomyopathy, Bai et al.[12] reported their long-term follow-up results in patients undergoing endocardial-epicardial hybrid ablation procedure. At three years, there was freedom from VT and ICD therapy in 85% of these patients, significantly better than the outcome in patients undergoing endocardial ablation alone. In summary, combined endocardial and epicardial catheter mapping and ablation may increase the VT-free follow-up period, but regardless of the site of origin, the underlying cardiac disease significantly influences the long-term outcome.

Special topics

Epicardial catheter ablation after cardiac surgery

The presence of intrapericardial adhesions may complicate percutaneous pericardial access as well as catheter maneuverability and mapping in patients who have undergone previous cardiac surgery. The posterior approach is preferable in patients with prior sternotomy. The first report of percutaneous subxiphoid access in five patients with prior cardiac surgery (3 of them coronary artery bypass grafting [CABG] surgery) appeared from Dr. Sosa's laboratory.[31] Pericardial access could be achieved in all five, and two underwent epicardial VT ablation. By contrast, Roberts-Thomson et al.[32] reported that pericardial access could be achieved in a minority of patients with prior cardiac surgery. More recently, Tschabrunn et al.[33] reported successful pericardial access and epicardial catheter ablation in eight patients with prior cardi-

ac surgery. Of note, none of these patients had had CABG surgery. After pericardial access, free movement of the catheter may also be limited due to pericardial adhesions, but some of these adhesions may be lysed during catheter manipulation. Intracardiac echocardiography may increase the safety of epicardial mapping and ablation of VT in patients with prior cardiac surgery. The experience with patients with prior CABG surgery is not extensive enough to estimate with certainty the safety of the epicardial ablation procedure in these patients.

The epicardial catheter mapping and ablation procedures may have similar limitations in patients with a history of pericarditis or previous epicardial ablation.[33]

Epicardial catheter ablation in patients with LVAD

Left ventricular assist devices (LVAD) are being used with increasing frequency in patients with very advanced heart failure, either as a bridge to cardiac transplantation, or as destination therapy. Sustained VT or ventricular fibrillation (VF) frequently complicates the course of LVAD therapy. Although these arrhythmias are well tolerated acutely, recurrent intractable VT or VF can result in frequent ICD shocks, which may carry significant morbidity, and these arrhythmias can also result in hemodynamic compromise despite LVAD support due mainly to worsening right heart failure.[34] Our own data have shown that a certain proportion of frequently recurrent or incessant VTs in patients with LVAD, especially in patients with advanced non-ischemic cardiomyopathy as the underlying diagnosis, are epicardial in origin. Catheter ablation should be undertaken with great caution, if at all, in patients with mechanical assist devices since there are no data regarding their safety and complications.

Conclusion

The incidence of epicardial VT varies from one patient population to another, and is influenced by the underlying heart disease, if any. Judging from the published reports, epicardial origin of VT is one of the commonest causes of unsuccessful ablation attempts limited to the endocardium, in diverse types of cardiac diseases. By approaching patients with both endocardial and epicardial catheter mapping and ablation, the overall success rate can be increased. The electrophysiologic principles are the same as those used in endocardial mapping and ablation procedures, even though the biophysical principles of energy delivery may differ. Additional safety precautions, not applicable to or required for endocardial catheter ablation, must be taken. In experienced centers, epicardial catheter ablation is a safe and effective treatment modality for recurrent VT.

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