

Role of Ablation Therapy in Ventricular Arrhythmias

Mithilesh K. Das, MD, MRCP, FACC*, Gopi Dandamudi, MD,
Hillel Steiner, MD

Krannert Institute of Cardiology, 1800 North Capitol Avenue, Indianapolis, IN 46202, USA

Sustained ventricular tachycardia (VT) and ventricular fibrillation (VF) are associated with a poor prognosis because of an increased risk for sudden cardiac death (SCD), particularly in patients who have structural heart disease (SHD) [1]. In addition, frequent nonsustained VT (NSVT), premature ventricular complexes (PVCs), or ventricular couplets may cause tachycardia-induced cardiomyopathy, a rare consequence of these arrhythmias. In the present era, the implantable cardioverter defibrillator (ICD) is the mainstay therapy for primary and secondary prevention of SCD. Recurrent VT develops in 20% and 40% to 60% in patients who receive an ICD for primary and secondary prophylaxis for SCD, respectively [2]. ICDs terminate most ventricular arrhythmia (VA) episodes. ICDs do not prevent recurrence of VAs or change the underlying substrate of VA, however. In fact, there is evidence that ICDs may increase the incidence of VA. Repeated ICD shocks reduce quality of life and increase mortality. Recurrent VAs in these patients often are treated with antiarrhythmic agents with only moderate success. Furthermore, these drugs are associated with an increased risk of proarrhythmia, systemic toxicity, and increased defibrillation threshold (especially amiodarone). Catheter ablation is the treatment of choice to cure or reduce the recurrences of VA in patients who have an ICD [3]. Catheter ablation can be life-saving for electrical storms (ES), defined as three separate episodes of VT or VF within a 24-hour period, each separated by 5 minutes.

ES is an independent predictor of short-term mortality and occurs in 3.5% and 20% of patients who have an ICD implanted for primary and secondary prophylaxis, respectively. Catheter ablation is also the treatment of choice for symptomatic idiopathic VT or PVCs. Polymorphic VT or VF initiated by a single monomorphic PVC also can be treated with catheter ablation. A recent randomized trial showed that ablation therapy in patients who have an ICD implanted for secondary prophylaxis reduces the risk for ICD therapy by 65% during a 2-year follow up as compared with the patients who do not receive ablation therapy.

Catheter ablation of VT or VF in the electrophysiology (EP) laboratory remains a challenging procedure. Patients who have SHD often have poor hemodynamic tolerance to the VA induced in the EP laboratory. Catheter ablation of VA requires a precise understanding of cardiac EP, the VA mechanism, and mapping techniques. Most VAs can be ablated endocardially. Epicardial ablation is needed for VTs with an epicardial circuit or focal source. The purpose of this article is to describe current mapping techniques and indications and to discuss the present status of catheter ablation for VA.

Mechanisms of ventricular arrhythmia

VA mechanism, like any arrhythmia, has either a focal source or a reentrant circuit. Sustained monomorphic VT (SMMVT) occurs predominantly because of reentry in patients who have SHD, whereas a focal VT or a PVC occurs because of enhanced automaticity or triggered activity in patients who have normal hearts and rarely, in patients who have SHD (Box 1, Table 1).

Dr. Steiner is a recipient of a Fellowship Grant from The American Physicians Fellowship for Medicine in Israel.

* Corresponding author.

E-mail address: midas@iupui.edu (M.K. Das).

Box 1. Classification of ventricular arrhythmia

Monomorphic VT

1. SHD
 - a. CAD (Post-MI):
 - Scar related reentry
 - Focal VT
 - His-Purkinje related VT
 - b. Cardiomyopathies and myocarditis:
 - DCM
 - Others
 - Myocarditis
 - Hypertrophic cardiomyopathy
 - ARVD/C
 - Infiltrative (Sarcoidosis)
 - Chagas disease
 - c. Incisional (after cardiac surgery):
 - VSD or TOF repair
 - Aortic and mitral valve repair
 - LVAD
 - d. Bundle branch reentry;
 - DCM
 - CAD
 - Myotonic dystrophy
 - Mitral and aortic valve-surgery
2. Structurally Normal Heart (Idiopathic VT):
 - a. Idiopathic RVOT VT and its variants
 - b. Verapamil-sensitive left ventricular fascicular VT
 - c. Catecholamiergic monomorphic VT

Polymorphic VT/VF

1. SHD:
 - a. CAD
 - Late post-MI
 - During acute myocardial ischemia
 - b. DCM and other cardiomyopathies
2. Structurally Normal Heart (Idiopathic VT):
 - a. Idiopathic RVOT VT (malignant variety)
 - b. Idiopathic VF
3. Inherited arrhythmia syndromes
 - a. Long QT syndrome
 - b. Short QT syndrome
 - c. Brugada syndrome
 - d. Exercise induced catecholaminergic polymorphic VT
 - e. Overlap syndrome (SCN5A mutation)

Abbreviations: ARVD/C, arrhythmogenic right ventricular dysplasia cardiomyopathy; CAD, coronary artery disease; DCM, dilated cardiomyopathy; LVAD, left ventricular assist device; RVOT, right ventricular outflow tract; SHD, structural heart disease; TOF, tetralogy of Fallot; VSD, ventricular septal defect; VT, ventricular tachycardia.

Ventricular arrhythmia in structural heart disease

The mechanism of VA in patients who have SHD is mostly scar-related reentry, but rarely can be focal in origin [4,5]. Reentry occurs at the border zone of myocardial infarction (MI) scar, around areas of scar in nonischemic dilated cardiomyopathy (DCM), and around surgical scars in patients who have congenital heart disease, valvular heart disease, or implanted hardware such as a left ventricular assist device (LVAD). SMMVT in patients who have an MI scar usually is caused by myocardial reentry in the infarct border zone. The reentrant circuit of scar-related VT can be modeled as having an isthmus or corridor bounded by two scars, or a scar and a line of anatomic barrier or physiologic conduction block. Most of these VT circuits have surviving strands of myocardium interlaced with interstitial fibrosis and diminished cell-to-cell coupling with slow conduction. Wave front conduction through these areas creates an excitation gap and promotes reentry. The conduction of wave fronts through these corridors is mapped as mid-diastolic or presystolic electrograms (Egms). These voltages, however, are too low to contribute to the surface ECG. The QRS complex is inscribed when the impulse exits the isthmus and spreads rapidly across the relatively healthier ventricular myocardium. The VT cycle length depends mainly upon the length of isthmus and conduction velocity of the impulse while passing through the isthmus (Fig. 1). During a slow VT, the isthmus can be mapped for a few centimeters with relatively slow conduction across the isthmus. Of note, a single area of the scar border zone may be a substrate for multiple channels giving rise to multiple VT morphologies of varying cycle lengths. Unlike coronary artery disease (CAD), DCM is associated with diffuse myocardial scar, which occurs mostly in the basal left ventricle, primarily in midmyocardial and epicardial layers [6]. In addition, His-Purkinje system-related reentrant arrhythmia such as bundle branch reentry and focal SMMVT preceded by Purkinje potentials are occasionally encountered in patients who have SHD [7,8].

Idiopathic and inherited ventricular arrhythmia

Focal SMMVT originating from right ventricular (or less commonly left ventricular [LV]) outflow tract (RVOT) is the most common type (approximately 90%) of VT in patients who have

Table 1
Comparisons between the electrophysiological characteristics of the focal and reentrant ventricular tachycardia

Characteristics	Focal ventricular tachycardia (VT)	Reentrant VT
Initiation	Spontaneous or during isoproterenol infusion	Spontaneous or programmed stimulation
Number of VT morphologies present	Single	Single or multiple
Electrogram voltage around site of successful ablation	Normal voltage in normal hearts or low voltage around the focal source in structural heart disease (SHD)	Low voltage area at the site and surrounding area usually has a protected corridor.
Electrogram (Egm) during VT	1. Shorter Egm to QRS interval 2. A lower ratio of Egm to QRS duration and diastolic interval	1. Isolated mid to early diastolic potential 2. Relatively higher ratio of Egm to QRS duration and diastolic interval
Pace map	Identical to nearly identical pace-map, short stimulus-QRS interval (approximates Egm-QRS interval in VT)	Identical to nearly identical pace map, long stimulus-QRS interval (approximates Egm-QRS interval in VT)
Entrainment	Cannot be entrained	Entrainment in most stable VT is possible.
Activation mapping (isochronal electroanatomical map)	Focal source with a centrifugal spread of activation	Reentrant excitation with a protected diastolic corridor

structurally normal hearts. The mechanism of these VTs is postulated to be cAMP-mediated delayed afterdepolarizations and triggered activity caused by an acquired somatic cell mutation in the inhibitory G protein. Idiopathic LV fascicular VT (ILVT), the second most common SMMVT, has a reentrant mechanism with a relatively small circuit in close proximity to the left posterior fascicle (10% in close proximity to the left anterior fascicle). Both these arrhythmias have a benign prognosis. Rarely, repetitive idiopathic PVCs from RVOT or originating from other sites in the ventricles can initiate polymorphic VT or VF (idiopathic VF), which is associated with a high risk for SCD. The mechanism of VA varies in inherited arrhythmia syndromes: early after depolarization-induced triggered activity (long QT syndrome), delayed afterdepolarization-induced triggered activity (exercise-induced catecholaminergic polymorphic VT), and phase 2 reentry (Brugada syndrome).

Indications for catheter ablation

Catheter ablation is indicated as a first-line therapy for symptomatic VT in patients who have normal hearts, and recurrent VT despite antiarrhythmic therapy in patients who have SHD (Table 2). Consideration of risks and benefits should be individualized. Procedural risks are likely to be increased in the elderly and in patients

who have severe underlying heart disease. In many patients, VT recurrences are reduced acceptably by antiarrhythmic drug therapy, and ablation is not required. Recurrent PVCs or VT episodes can result in deterioration of LV systolic function (tachycardia-induced cardiomyopathy) and increase the risk for heart failure and SCD. Catheter ablation is indicated in these patients. Additionally, symptomatic frequent PVCs and monomorphic PVCs that repeatedly initiate polymorphic VT or VF (idiopathic VF and in rare cases of Brugada syndrome and long QT syndrome) can be treated with catheter ablation.

Preprocedural evaluation

Patients should be risk stratified according to their clinical status and the type of VA. Patients who have poor LV function or advanced heart failure may not tolerate VT induction necessary for precise mapping and ablation of VA. Therefore, active coronary ischemia and fluid as well as electrolyte status should be evaluated and treated appropriately before the procedure. Patients who present with polymorphic VT or VF and ischemic symptoms should be evaluated for active myocardial ischemia, whereas patients who have an SMMVT often have stable CAD and usually do not need ischemia evaluation. Therefore, evaluation for active coronary artery disease usually is not needed before catheter ablation of SMMVT.

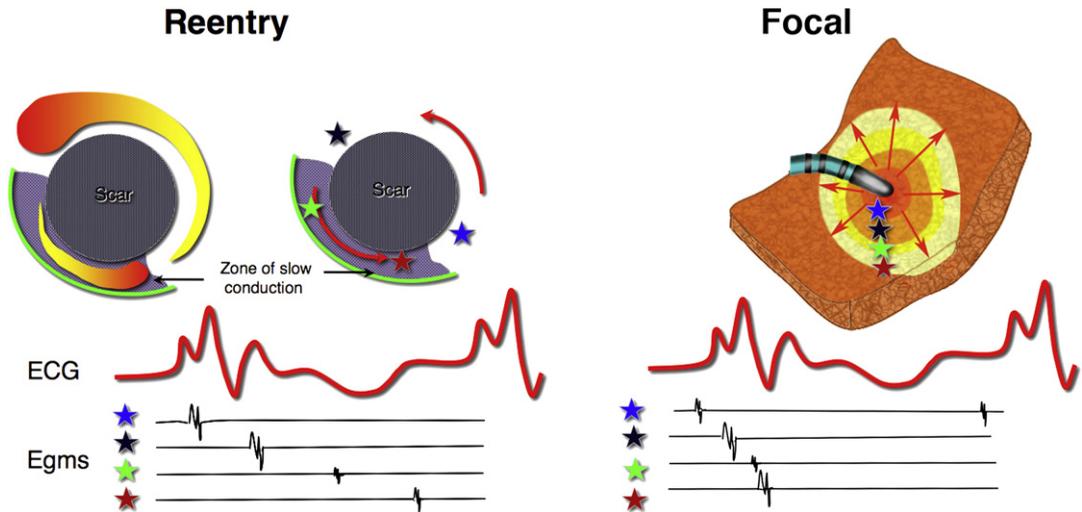


Fig. 1. Reentrant versus focal ventricular tachycardia (VT). The figure shows the timing of intracardiac recordings in relation to the surface ECG during mapping of a reentrant VT and a focal VT. Focal VT has a centrifugal activation.

Cardiac imaging before catheter ablation

Echocardiographic evaluation is required in patients who have SHD and poor systolic LV function to rule out any LV clot. It also may provide information regarding the location and extent of scar in the left ventricle. Traditional fluoroscopic mapping during EP study is a two-dimensional mapping system. Therefore, fine anatomic details such as papillary muscles and LV aneurysm are not visible. Recently, various mapping systems have been developed that create a three-dimensional map of the ventricles or the area of interest. These mapping systems recreate the geometry of the ventricles from point-by-point sampling while providing continuous display of the catheter position. Electrophysiological data such as the activation time, Egm amplitude, and impulse propagation are color-coded for display, which helps in localizing a focal source or a critical isthmus. This is possible only during a hemodynamically stable VT and allows mapping of multiple points during the VT. During a hemodynamically unstable VT, however, circuits cannot be localized well and therefore, ablation is performed in the scar border zones using substrate and pace mapping techniques. Scars in the left ventricle can be defined better by importing preacquired CT or cardiac magnetic resonance (CMR) images on mapping systems and using them as anatomic shells. These three-dimensional images can be incorporated into the three-dimensional map acquired during the EP study,

which allows for a better understanding of the substrate and possible channels of VAs to be ablated.

Ventricular tachycardia morphology: an important guide to ablation

A careful analysis of ECGs of the clinical VT (mainly QRS morphology, QRS axis, and R wave transition in precordial leads) is prudent, because it is a major clue to the area of interest for mapping and ablation [9]. These serve only as a general guideline, however, because the QRS morphology in a reentrant VT also depends on several other factors such as the amount of scar, use of antiarrhythmic drugs, and orientation of the heart in the thorax (horizontal versus vertical) or on other pathology in the thoracic cavity (eg, pneumonectomy), causing alteration of the QRS vector.

Ventricular morphology in structural heart disease

As a general rule, in scar-based VT, all left bundle branch block (LBBB) morphology VTs arise from the LV septum (rarely from the right ventricle), whereas right bundle branch block (RBBB) morphology VTs can arise on the LV septum or the LV free wall. The QRS axis is the next major clue regarding the site of exit of a VT. A superior axis suggests an inferior wall exit site, whereas an inferior axis suggests an anterior wall exit site. The R wave amplitude in precordial leads is also a very important indication of the exit site

Table 2

Indications for catheter ablation of ventricular tachycardia and ventricular fibrillation according to current American College of Cardiology/American Heart Association guidelines [26]

	Indications	Level of evidence	Common scenario
Class 1	Patients who are otherwise at low risk for sudden cardiac death (SCD) and have sustained predominantly monomorphic ventricular tachycardia (VT) that is drug resistant, who are drug intolerant, or who do not wish long-term drug therapy	C	Idiopathic VT
	Patients with bundle-branch reentrant VT	C	Usually require an implantable cardioverter defibrillator (ICD) implant because of high recurrence of scar-related VT
	As adjunctive therapy in patients with an ICD who are receiving multiple shocks as a result of sustained VT that is not manageable by reprogramming or changing drug therapy or who do not wish long-term drug therapy	C	Patient with recurrent VT shocks or VT storm refractory to antiarrhythmic therapy
	Patients with WPW syndrome resuscitated from sudden cardiac arrest caused by atrial fibrillation and rapid conduction over the accessory pathway causing VF	B	High-risk patients with WPW syndrome
Class 2a	Patients who are otherwise at low risk for SCD and have symptomatic nonsustained monomorphic VT or monomorphic premature ventricular complex (PVC) that is drug-resistant, who are drug intolerant or who do not wish long-term drug therapy.	C	Idiopathic VT, tachycardia-induced cardiomyopathy
Class 2b	Ablation of Purkinje fiber potentials may be considered in patients who have ventricular arrhythmia storm consistently provoked by PVC of similar morphology	C	Idiopathic VF or VF in patients with structural heart disease (SHD) repeatedly initiated by a single monomorphic PVC
	Ablation of asymptomatic PVC may be considered when the PVCs are very frequent to avoid or treat tachycardia-induced cardiomyopathy.	C	Tachycardia-induced cardiomyopathy in patients with normal hearts and SHD
Class 3	Ablation of asymptomatic relatively infrequent PVC is not indicated	C	Asymptomatic PVC in patients with normal hearts or SHD

of a VT. Dominant R waves in precordial leads (V_1 to V_5) suggest a basal exit, and dominant R waves in the midprecordial leads (V_3 and V_4) suggest an exit site between the base and apex. Apical VAs generate dominant S waves in all the precordial leads (similar to that encountered during right ventricular apical pacing).

Idiopathic ventricular tachycardia

Various algorithms have been described to localize idiopathic VTs also. VT originating from RVOT has an LBBB, inferior axis configuration

with precordial transition to more positive QRS by V_4 , whereas the LV outflow tract (LVOT) VTs have RBBB inferior axis configuration [10]. A free wall origin is suggested by QRS duration greater than 140 milliseconds and notches in the inferior leads (II, III, AVF). Deeper S waves in aVL than in aVR suggest a leftward superior focus, and this ratio decreases with sites located more rightward and inferior in the RVOT. VT originating from the pulmonary artery and the aorta typically has large R waves in the inferior leads and greater R/S ratio in lead V2. The QRS axis depends on the location on the annulus.

Epicardial ventricular tachycardia

VTs that originate in the subepicardium generally have a slurred upstroke (pseudodelta wave). A pseudodelta wave of greater than or equal to 34 milliseconds (sensitivity: 83%, specificity: 95%), an intrinsicoid deflection time of greater than or equal to 85 milliseconds (sensitivity: 87%, specificity: 90%), and an RS complex duration of greater than or equal to 121 milliseconds (sensitivity: 76%, specificity: 85%) suggest an epicardial origin of a VT [11].

Ablation catheters and mapping techniques

Various mapping techniques are used to locate the circuit of a reentrant VT and the site of origin of a focal VT. VT that cannot be induced during EP study or be mapped because of hemodynamic compromise has a lower ablation success rate than the VTs in which the critical isthmus or the focal site can be localized. Several mapping techniques are used during catheter ablation of VT, including activation mapping, pace mapping, entrainment mapping, and substrate mapping (Table 3) [2,12]. Most commonly, a 4 mm or 5 mm tip mapping and ablation catheter is used for endocardial radiofrequency ablation (RFA). A 3.5 mm irrigated tip catheter is also used for VT ablation, especially for inflicting deeper tissue damage. Alternatively, an 8 mm catheter can be used for inducing a larger area of tissue damage; however, mapping is less precise because of a relatively larger antenna of these catheters. An irrigated tip catheter is preferred over the regular 4 mm tip RFA catheter for epicardial ablation. Cryoablation (4 mm to 6 mm tip) is another modality of catheter ablation, which inflicts tissue damage by decreasing the local temperature to -70° (usually applied for approximately 4 minutes at a single site). It has been used for VT ablation in the epicardium and the coronary sinus.

Activation mapping

Activation mapping is a very useful technique in patients who have hemodynamically stable VT. In focal VTs such as idiopathic RVOT VT, the earliest site of activation usually has a local intracardiac Egm 10 to 60 milliseconds earlier than the QRS onset. In reentrant VT in patients who have SHD, single or multiple presystolic or mid-diastolic Egm can be recorded in the isthmus or blind loop of the circuit (see Fig. 1).

Pace mapping

Pace mapping of VT and PVCs is performed by pacing from putative sites in the ventricles to entirely replicate the 12-lead QRS morphology of the VT induced in the EP laboratory or recorded on a standard ECG during the clinical arrhythmia, thereby to indicate the origin of a focal VT and critical isthmus of a reentrant VT. The QRS morphology obtained during pace mapping depends, not only on the location of the catheter in relation to the VT circuit or exit site, but also on several other factors. These include location of scar, catheter contact to the myocardial tissue, pacing output, unipolar versus bipolar pacing, and interelectrode distance during bipolar pacing. Pace mapping should be performed at a rate similar to the target VT using the minimum possible pacing output to ensure capture. Use of high pacing output may result in a relatively larger area of myocardial capture in the vicinity of the isthmus and may give rise to an erroneous QRS morphology, even when pacing is performed in the true isthmus. The 12-lead ECG during pace mapping should be matched carefully with the 12-lead ECG of the clinical VT or the target PVCs. An ideal pace map is considered perfect or exact if the QRS complexes in all 12 leads during pacing are identical to those of the targeted VT (ie, superimposable) (Fig. 2). A pace map is considered good if the QRS complexes during pacing and VT are identical in 10 or 11 of the 12 leads. An exact pace map (match) can be obtained in 49% to 81% of VTs [13]. Pace mapping at the site of fractionated Egm in the region of the suspected isthmus results in progressive prolongation of the S-QRS interval as the pacing site moves along the length of the isthmus, consistent with pacing progressively further from the exit site. Pace mapping is helpful in localizing the critical isthmus in up to 85% of VTs.

Entrainment mapping

Entrainment mapping is a very useful technique for mapping an SMMVT to determine the reentrant mechanism of the VT, and it can be performed from different sites in the ventricles. Usually, entrainment initially is performed from a site remote from the presumed isthmus by pacing at a cycle length marginally faster than that of the induced VT to demonstrate QRS fusion (manifest entrainment). Pacing also can be performed at progressively faster rates from the

Table 3
Comparison of mapping techniques and success rates of focal and reentrant ventricular tachycardia ablation

Hemodynamic status	Mechanism of VA	VT morphology	VA in structural heart disease (SHD)	Idiopathic VA	Mapping				Success rate
					Activation	Pace	Entrainment	Substrate	
Stable	Reentrant VT	SMMVT	Scar related	ILVT	+	±	+	+	High
	Focal	SMMVT or PVC	His-Purkinje disease	RVOT VT/ PVC	+	+	-	-	High
Unstable	Reentrant VT	Sustained monomorphic VT	Scar-related reentry		-	±	-	+	Moderate to low
		Polymorphic VT/VF	Scar related reentry		-		-	-	Moderate to low
	Focal	Polymorphic	Monomorphic PVC-induced VT/VF	Idiopathic VT/VF, BS, LQTS	-	+	-	-	High

Abbreviations: BS, Brugada syndrome; ILVT, idiopathic left ventricular tachycardia; PVCs, premature ventricular complexes; SMMVT, sustained monomorphic ventricular tachycardia; VA, ventricular arrhythmia; VF, ventricular fibrillation; VT, ventricular tachycardia; +, useful; -, not useful; ±, sometimes useful.

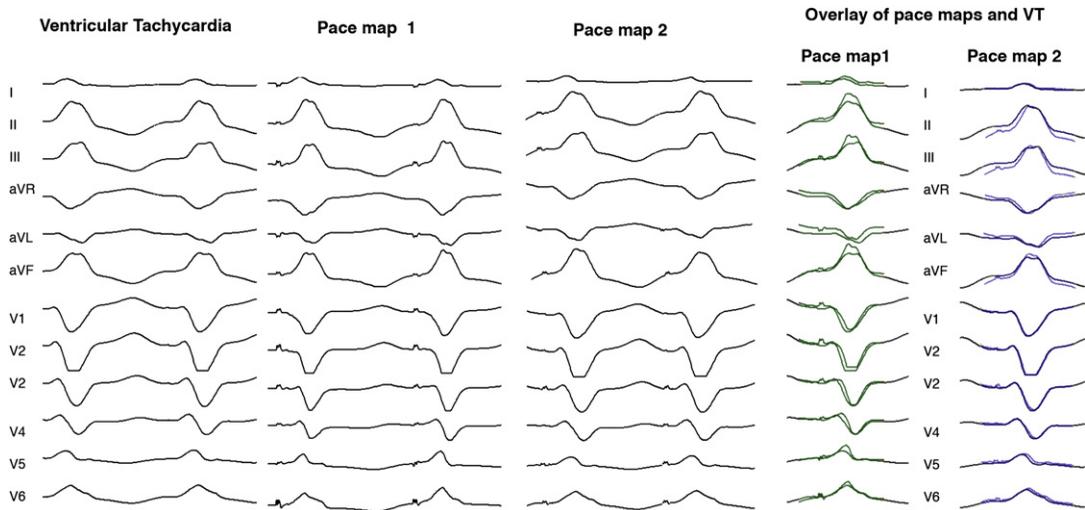


Fig. 2. Pace mapping of ventricular tachycardia (VT). Pace maps 1 and 2 of the VT are overlaid on the left. Pace map 1 is an example of a “not so good” pace map (9/12) of reentrant VT. At first look, the match appears to be close for QRS morphology in most of the leads but is obviously different in inferior leads (II, III and aVF), whereas pace map 2 is an excellent match (12/12) with the VT.

same site (or other sites remote from the presumed VT circuit) to demonstrate the progressive fusion of VT morphology on the 12-lead ECG, confirming its reentrant mechanism (entrainment with progressive fusion). Entrainment mapping then can be performed from additional sites, comparing the difference between the interval from the stimulus (S) artifact to the Egm at the pacing site on the first return complex of the VT (the postpacing interval, PPI) and the VT cycle length as a indicator of proximity of the pacing site to a critical isthmus. Finally, when entrainment is performed by pacing from the site of presumed critical isthmus, the 12-lead QRS morphology is identical to the VT, and the PPI at that site is equal or minimally longer (less than 30 milliseconds) than the VT cycle length (entrainment with concealed fusion) (see Fig. 2; Fig. 3).

Substrate mapping

Activation and entrainment mapping of rapid SMMVT, polymorphic VT, or VF is difficult because of a changing wavefront propagation, rapid heart rate, and hemodynamic instability. Careful analysis of clinical VT morphology helps to guide the regionalization of exit sites of VT. Because most of the VT circuits in CAD are located in the peri-infarct region (border zone),

pace mapping, along with substrate mapping, is helpful in localizing these areas. Single or multiple lines of ablation are performed perpendicular to the area of scar, which is extended from the scar tissue to the normal myocardium. Recently, a T-shaped line of ablation at the border zone has been used with reasonably good success [14]. Overall success rates of these types of ablation are less, because the precise circuit cannot be localized.

Therefore, these mapping techniques complement each other. All of these tools can be used to localize the critical isthmus of a hemodynamically stable sustained MMVT, whereas activation mapping and pace mapping are the only useful techniques available for localizing the exit sites of a focal VT or frequent symptomatic PVCs [15,16]. Mapping techniques for hemodynamically unstable MMVTs are limited to pace mapping and substrate mapping. Pace mapping complements the findings of activation and entrainment mapping [15]. Entrainment mapping is not helpful for focal VT mapping.

Ablation techniques

Most of the VT circuits are mapped and ablated endocardially. Epicardial ablation is needed for a minority of VTs associated with

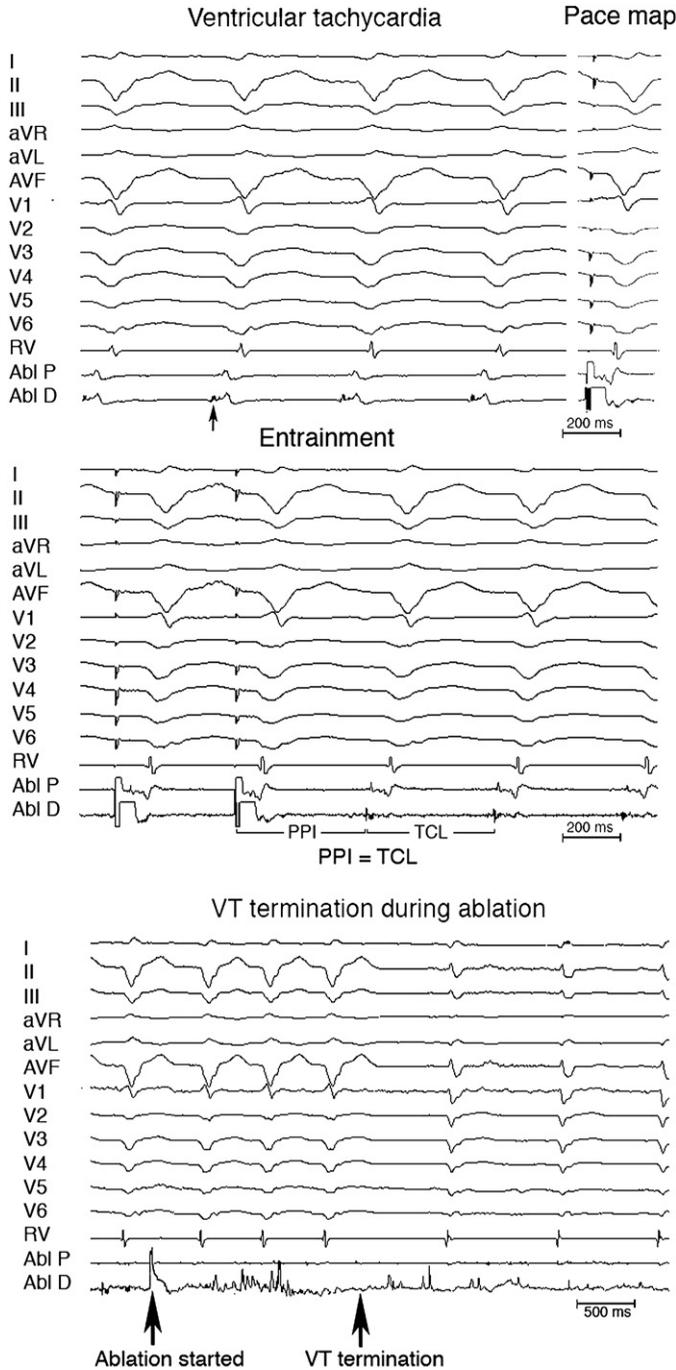


Fig. 3. Pace and entrainment mapping of a reentrant ventricular tachycardia (VT). A good pace map of VT (11/12) is displayed by pacing from the site where a presystolic potential (*small arrow*) was recorded by the ablation catheter (*upper panel*). Pacing during VT from the same site revealed concealed entrainment with a postpacing interval (PPI) equal to the tachycardia cycle length (TCL) (*middle panel*). Lower panel shows tachycardia termination during radiofrequency ablation. *Abbreviations*: Abl D: ablation distal, Abl P: ablation proximal, RV: right ventricle.

CAD, 30% to 40% of VTs associated with DCM, and a minority of idiopathic focal VTs [17]. In Chagas disease, 70% of VTs are epicardial in origin. Rarely, VT may arise from the pulmonary artery, the aortic sinus of Valsalva, the muscle bands in relation with the coronary sinus and the middle cardiac vein. Most idiopathic RVOT VTs and VTs associated with ARVD, congenital heart disease, or sarcoidosis arise from the right ventricle.

Endocardial ablation

LV mapping is performed retrogradely across the aortic valve or anterogradely across the mitral valve by means of a transseptal approach. The transseptal approach is the only method used for patients who have a mechanical aortic valve or severe peripheral vascular disease. LV mapping needs continuous anticoagulation during the procedure. The right ventricle is accessed by means of the femoral veins. The major risks of ablation include cardiac tamponade, thromboembolism including cerebral embolism (0% to 2.7%), coronary artery damage, incessant VT requiring multiple DC shocks, damage to the aortic valve, conduction system, or a coronary artery ostia, and rarely death. Significant vascular access complications, including bleeding, arterial dissection, and femoral arteriovenous fistulas can occur in about 2% of patients.

Epicardial ablation

Epicardial ablation is needed for VTs that originate in subepicardial or deep myocardial layers and cannot be ablated via the endocardium. The pericardial space is entered using an epidural needle under fluoroscopic guidance and contrast injection, followed by placement of a guidewire and introducer sheath for the mapping catheter [18]. Epicardial ablation is associated with a risk for epicardial coronary artery damage. Therefore, coronary angiography is recommended before the application of radiofrequency energy in all cases but infarct-related VT. Other risks include hepatic hemorrhage, phrenic nerve damage, pericardial effusion, and pericarditis. Pericarditis usually resolves in a few weeks, but patients may need aspirin or other anti-inflammatory drugs for that period.

Ablation strategies for sustained monomorphic ventricular tachycardia

Reentrant ventricular tachycardia

Mapping and ablation of a hemodynamically stable reentrant VT is very rewarding because of the high success rate of ablation. If a VT is inducible, then the QRS morphology guides toward the area of interest for mapping and ablation. Fig. 4 shows a reentrant circuit of an SMMVT with representation of different possible channels: critical isthmus, inner loop, outer loop, blind loop, and a bystander pathway. Careful manipulation of the mapping catheter locates the earliest mid-diastolic to late diastolic Egm during activation mapping (sites 1 and 3). Entrainment from these sites usually differentiates a blind loop (site 7) from the true isthmus (PPI-tachycardia cycle length is usually greater than 30 milliseconds at the blind loop). Additionally, pace mapping is performed within the isthmus. The stimulated wave front can proceed along certain paths, only which occur in at least two directions: the orthodromic and antidromic directions of propagation during the VT. Similar to the reentrant VT, the wave front during pace mapping only is detected on the surface ECG when it leaves this protected isthmus. Pacing from the exit site should give rise to a similar QRS morphology and a S-QRS interval that is relatively short and similar to the Egm to QRS (Egm-QRS) interval (sites 4 and 5). Pacing proximal to the exit sites should give similar results, except that the S-QRS and Egm-QRS intervals will be longer because of slow conduction of the impulse from those sites to the exit site [19]. Pace mapping from site 3 of the reentrant VT in Fig. 4 shows a long Egm-QRS (arrow) and similar S-QRS intervals with pacing, and a complete replication of each feature of each ECG lead. When the isthmus is short, or the catheter is positioned more proximally close to an entrance site of the critical isthmus (site 1), the stimulated antidromic wave front leaves the protected isthmus at the entrance and propagates to the surrounding myocardium producing a different QRS morphology. If the orthodromic wave front reaches the exit, a fused QRS complex is produced that includes depolarizations from both antidromic and orthodromic wave fronts. Therefore, the resultant QRS morphology depends upon the precise location of the pacing site relative to the reentrant circuit. As the mapping catheter is moved toward the

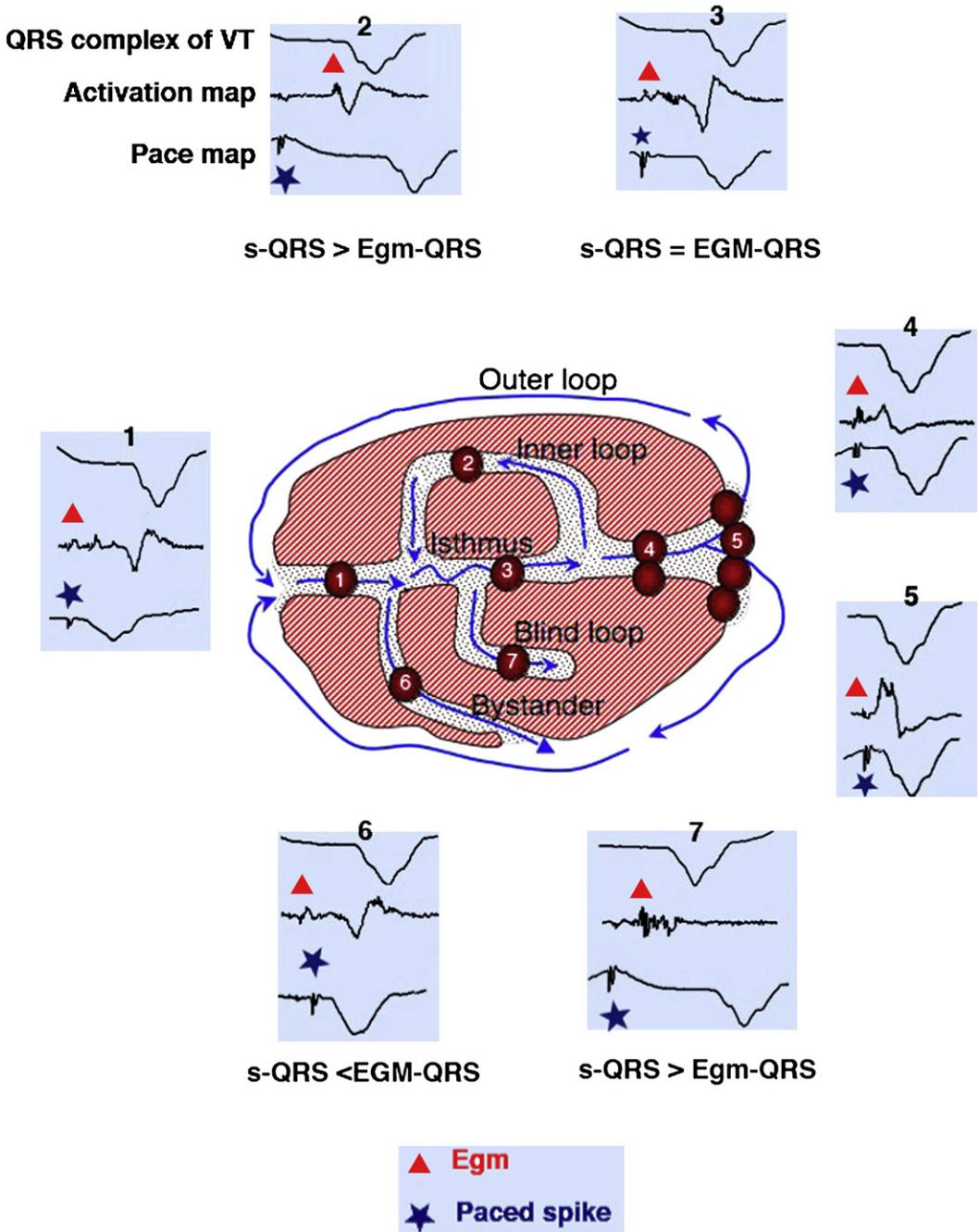


Fig. 4. Activation and pace mapping of a ventricular tachycardia (VT) from the presumed sites of VT circuits in a patient with structural heart disease. There is a long electrogram (Egm)-QRS interval (arrow) and similar stimulus (s)-QRS with pacing, and complete replication of each feature of each ECG lead when paced from the critical isthmus (site 3). Pacing during VT near entrance (proximal to slow conduction zone, (site 1) resulting in long S-QRS and QRS complexes identical to those of VT. Pacing at the same site, but during NSR, shows a completely different QRS, because the wave front can take a shorter path to normal myocardium than going through the diastolic corridor. Pacing near exit (sites 4 and 5) yields QRS match with a shorter S-QRS.

exit site (sites 4 and 5), pacing results in a QRS match of the VT but have a relatively shorter S-QRS interval. At the hypothetical bystander site (site 6), the activation map shows a relatively long Egm-QRS interval and a short S-QRS duration, whereas pace mapping shows a QRS morphology different from the clinical VT. In contrast to the entrance site of the critical isthmus (site 1), ablation at this site (site 6) will be unsuccessful. In the inner loop, the wavefront travels in the same direction as the outer loop. Therefore, the impulse reaches late during diastole at site 2, resulting in a relatively short Egm-QRS interval, whereas pacing from this site will show a relatively longer S-QRS interval, and ablation at this site also will be unsuccessful.

Fig. 5 shows various scar-related macroreentrant circuits encountered during mapping. Presystolic or mid- to late diastolic potentials (or Egms) are identified in approximately two thirds of patients during activation mapping of these VTs. A complete reentry circuit is defined in less than 20% of VTs with catheter mapping, whereas multielectrode and noncontact mapping systems identify endocardial exit regions of presystolic electric activity in greater than 90% of VTs. It is

not imperative to do this, however, because locating and ablating at the ideal site in the isthmus interrupts the particular circuit and terminate the VT. The participation of a particular isthmus with diastolic potential recorded during VT is confirmed by entrainment and pace mapping.

Focal ventricular arrhythmia

Focal MMVT (or PVC) generally is encountered in patients who have idiopathic VT, but it rarely may occur in patients who have MI scar. The bipolar Egm is used universally for this purpose, seeking a timing of 10 to 60 milliseconds before the QRS onset. The timing and morphology of unipolar recording from the catheter tip are also very helpful. At the suspected site of origin, this should have a sharp QS deflection that times with the bipolar recording and precedes the QRS onset by a similar amount.

As mentioned earlier, VTs in the setting of SHD have a reentrant mechanism in most patients, but a focal source of sustained MMVT has been reported also. A focal mechanism of VT has been found in 5% to 8% of VTs in patients who have CAD (Fig. 6). Similarly, a focal automatic

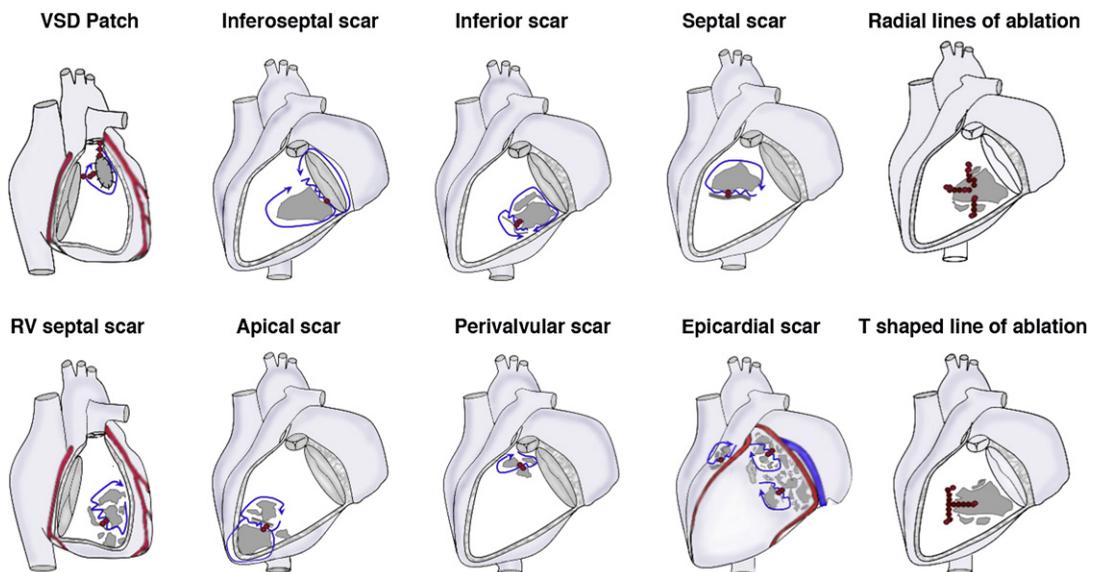


Fig. 5. Reentrant ventricular tachycardia (VT) circuits around myocardial scar in structural heart disease. The figure shows different endocardial and epicardial reentrant circuits with single loop or figure-of-eight wave fronts of VT, which can be ablated at critical isthmus sites. Reentry around a ventricular septal defect patch or right ventricular outflow tract (RVOT) patch can be ablated by connecting the ablation line from the patch to the pulmonary or the tricuspid valve. A single T-shaped line or multiple radial lines of ablation across the boarder zones often are drawn in patients in hemodynamically unstable VT. Blue lines denote potential circuits, red dots represent ablation points needed to interrupt the reentrant circuit.

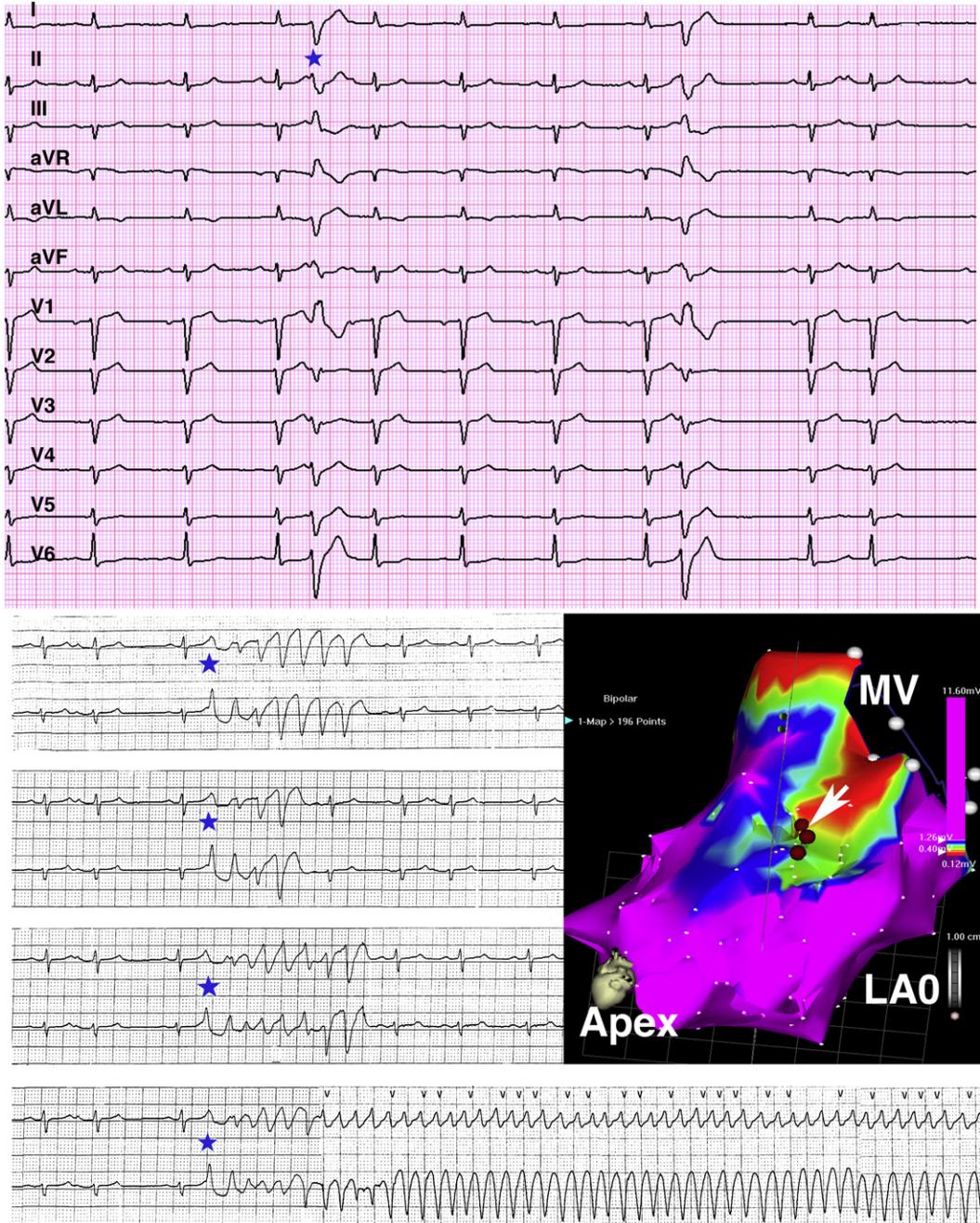


Fig. 6. Focal monomorphic premature ventricular complexes (PVCs) initiating nonsustained polymorphic ventricular tachycardia (VT) and ventricular fibrillation (VF). The telemetry recordings of a patient with history of myocardial infarction who presented with recurrent VF revealed frequent monomorphic PVCs and nonsustained VTs. Ablation of the PVC focus in the lateral left ventricle eliminated the ventricular arrhythmias.

mechanism was found to be responsible for VT in 27% of patients with DCM who underwent radiofrequency ablation for VT in one study [20].

Purkinje system ventricular tachycardia

His-Purkinje system-related SMMVT has been reported in patients who have SHD (8%) and in patients who have normal hearts [21].

Bundle branch reentrant ventricular tachycardia

Bundle branch reentrant VT occurs in 15% to 40% of patients who have DCM. It also can occur in ischemic cardiomyopathy (6%), after mitral or aortic valve surgery, myotonic dystrophy and rarely, in conduction system disease [22]. Typically, the VT has a LBBB pattern because of the wave fronts propagating retrogradely up the left bundle and anterogradely down the right bundle branch. Less frequently, the circuit revolves in the opposite direction, producing a RBBB configuration. Therefore, interruption of either of the bundles results in termination of the VT. Right bundle branch ablation, however, is preferred to avoid the hemodynamic consequences of chronic left bundle branch block (LV dyssynchrony). Usually, complete atrioventricular block does not occur. An ICD often is warranted, because of high risk of scar-related reentrant VT in these patients.

Ventricular tachycardias with participation of the left-sided His-Purkinje system

Interfascicular and fascicular VT occurs rarely in patients who have SHD. It can be cured by ablating the left posterior or left anterior fascicle depending upon the site of origin of the VT. It is associated with a considerable risk of complete heart block caused by preexisting conduction abnormalities. Similar VTs with a narrow QRS complex have been reported after acute or remote MI. During the VT, Purkinje potentials are captured orthodromically with decremental conduction properties, whereas presystolic Purkinje potentials are captured antidromically and appear between the His and QRS complex. In one study, catheter ablation at the site exhibiting a Purkinje-QRS interval of 58 plus or minus 26 milliseconds successfully eliminated VTs without provoking any conduction disturbances.

Idiopathic left ventricular tachycardia

ILVT involves the left posterior fascicle (less commonly, the anterior fascicle) and the posterior Purkinje network. The VT has a relatively narrow QRS (RBBB, superior axis), probably because of the conduction system being an integral part of

the circuit. An abnormal diastolic potential within the posterior Purkinje network during sinus rhythm and VT can be used to guide successful catheter ablation of ILVT.

Ablation strategies for polymorphic ventricular tachycardia and ventricular fibrillation

Structural heart disease

For single or multiple unstable MMVTs and polymorphic VTs or VF, voltage maps are created from three-dimensional anatomic plots of low-voltage regions (less than 1.55 mV in bipolar recordings) to identify areas of scar and perinfarct zones. Pace mapping at the scar border zone usually is required for a SMMVT or PVC that triggers polymorphic VT/VF. Brief entrainment is tried and is often possible, even for unstable VTs, to confirm the location of a reentry circuit. Low-amplitude isolated potentials and late potentials inscribed after the end of the QRS complex during sinus rhythm also suggest potential isthmus sites. Connecting the two scar zones or scar and a line of conduction block/anatomic structure also is performed empirically (Fig. 7). A radial line or T-shaped line of ablation also is performed often at the scar border zones (see Fig. 5).

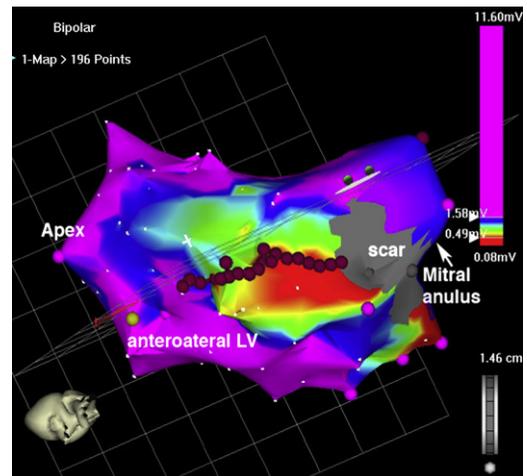


Fig. 7. Endocardial substrate mapping for ventricular tachycardia (VT). Endocardial substrate and pace mapping was performed for hemodynamically unstable reentrant VT with an exit site in the scar border zone of the anterolateral mid-left ventricle in a patient with history of myocardial infarction. Note a line of ablation was drawn across the border zone where good pace maps were achieved.

Polymorphic ventricular tachycardia and idiopathic ventricular fibrillation in normal hearts

Rare forms of polymorphic VT originate from the RVOT in structurally normal hearts and in the left ventricle in patients who have a history of MI. These VAs are triggered by monomorphic PVCs, which usually are preceded by a Purkinje-like potential. In idiopathic VF, activation and pace mapping of PVCs that initiate VF can be identified. Catheter ablation at these sites eliminates these trigger PVCs and prevents the recurrence of malignant polymorphic VT or VF episodes with a high success rate. Although this variety of life-threatening VA is initiated by repetitive monomorphic PVCs, the QRS morphology changes in the subsequent beats, and rapid pace mapping from the earliest site of activation also results in a change of QRS morphology similar to the clinical polymorphic VT. It is presumed that a shift in exit site during rapid pacing is responsible for the changing QRS morphology. Thus, PVCs that trigger polymorphic VT or VF in patients who have normal hearts, or SHD can be treated successfully with catheter ablation.

Ventricular tachycardia ablation in specific subpopulations

Coronary artery disease

Patients who have multiple VTs generally are tried on one or more antiarrhythmic drugs (most frequently, amiodarone) that have a limited response (19% to 50%). Most VTs can be ablated endocardially with a reasonably good long-term success rate of 77% to 95% for the clinical VT [2]. The epicardial approach for ablation is used for epicardial circuits or in presence of a LV thrombus (see Fig. 5).

Nonischemic dilated cardiomyopathy

SMMVT is uncommon in DCM. It occurs mostly (80%) because of scar-related reentry [20]. SMMVTs caused by reentry related to low-voltage regions (scars) often are located adjacent to a valve annulus that forms a border for the reentrant circuit. When a VT circuit cannot be defined during endocardial mapping, and significant low-voltage regions are not present on the endocardium, it often is present in the subepicardium (see Fig. 5). This likely contributes to the lower reported success rate of endocardial ablation. Some physicians start with epicardial ablation if

VT morphology suggests epicardial circuits. In Chagas disease, approximately 70% of VTs are epicardial in origin. Less commonly, it occurs because of bundle branch reentry [20]. Rarely, focal VTs are encountered. Of a series of 22 patients who had VT caused by reentry associated with DCM, endocardial ablation failed in 10 patients. Seven of these patients underwent epicardial mapping, with successful ablation in six patients. Fig. 8 demonstrates the endocardial and epicardial map of a patient with DCM in whom perivalvular and RV epicardial scar is demonstrated, whereas endocardial scar is minimal. His clinical VT was terminated by catheter ablation in the epicardial right ventricle medial to the proximal left anterior descending artery.

Other cardiomyopathies

Sustained VA is one of the major causes of SCD in cardiomyopathies such as hypertrophic cardiomyopathy, arrhythmogenic right ventricular dysplasia/cardiomyopathy (ARVD/C), various infiltrative cardiomyopathies such as sarcoidosis, and Chagas disease. ICD is indicated for most of these patients for secondary prophylaxis and for primary prophylaxis in high-risk patients.

Cardiac sarcoidosis

Cardiac manifestations of sarcoidosis include cardiomyopathy, AV block, and VA. Steroid treatment improves the heart block in 62% of patients, while the VT is unaffected. Many cases of VT related to sarcoidosis are misdiagnosed as idiopathic cardiomyopathy or ARVD. In a study of 98 patients who had nonischemic cardiomyopathy and VT, sarcoidosis was the cause in eight patients, and most were scar-related. Ablation abolished one or more VTs in six (75%) of eight patients, but other VTs remained inducible in all but one patient. After ablation, some form of sustained VT recurred in six of eight patients within 6 months. During a longer follow-up (range 6 months to 7 years), however, four of eight patients were free of VT with antiarrhythmic drugs and immunosuppression. Cardiac transplantation eventually was required in five of eight patients because of either recurrent VT ($n = 4$) or heart failure ($n = 1$). In another study, 68% of VTs were determined to be reentrant VTs.

Arrhythmogenic right ventricular dysplasia/cardiomyopathy

SMMVT occurs mostly because of reentry circuit in the vicinity of the tricuspid and

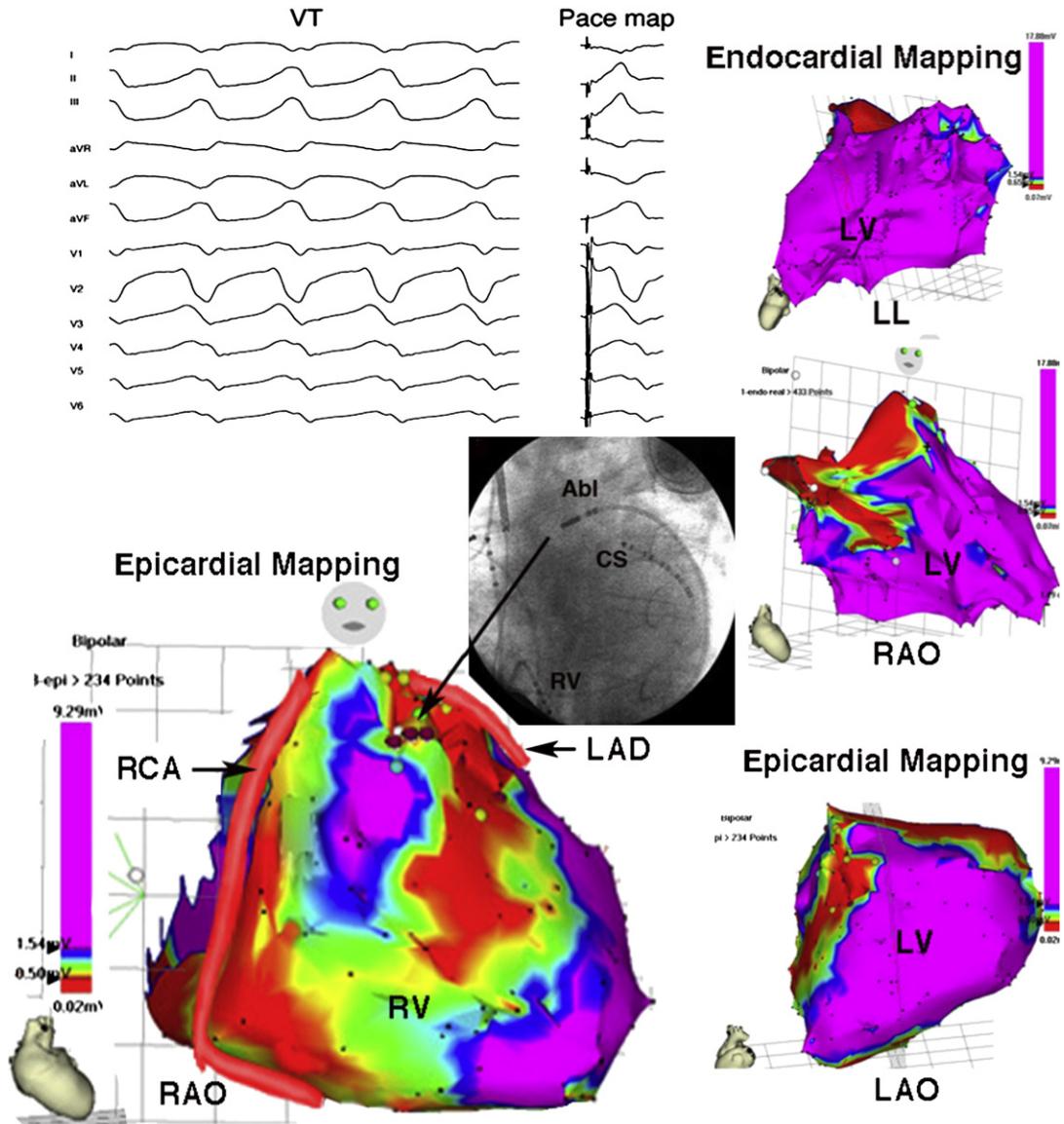


Fig. 8. Endocardial and epicardial bipolar voltage mapping to guide ablation. Endocardial and epicardial bipolar voltage mapping using an electroanatomical mapping system in a patient with dilated cardiomyopathy with sustained monomorphic ventricular tachycardia (VT). The endocardial mapping recorded minimal basal scar regions in the left ventricle (LV), whereas the epicardial mapping revealed extensive LV basal scars and right ventricular (RV) scar. The tachycardia was hemodynamically unstable. Pace mapping revealed an exit site medial to the proximal left anterior descending artery (LAD), which was ablated successfully. LAD and right coronary artery are drawn by mapping the coronary artery locations during coronary angiography. The fluoroscopic position of the catheter is shown in the left anterior oblique (LAO) view. *Abbreviations:* LL: left lateral view; RAO: right anterior oblique view.

pulmonary valve and the lateral right ventricle near the apex. The RV becomes paper-thin in scar regions; therefore, catheter ablation is associated with a 74% to 77% acute success rate. It also carries a higher risk for right ventricular perforation, and

a high recurrence rate (11%–75%) due to the progressive nature of the disease [2].

Chagas disease. Cardiomyopathy caused by Chagas disease is associated with segmental scars,

similar to ischemic cardiomyopathy. LV infero-lateral scar is often the source of VT (80%) and can be ablated successfully.

Congenital heart disease

Right ventriculotomy scar becomes a substrate for reentry in patients who have congenital heart disease such as repair of tetralogy of Fallot and ventricular septal defect. After repair of tetralogy of Fallot, the incidence of VT is 11.9%, with an 8.3% risk of SCD by 35 years of follow-up. Substrate mapping to evaluate congenital, surgical, and electrophysiological anatomy identifies the isthmus for successful VT ablation. Naturally occurring anatomic barriers in the right ventricle are limited, and the placement of a single ventriculotomy, outflow tract patch, or transannular patch, along with closure of the ventricular septal defect, would be expected to create only a limited number of possible tachycardia circuits (see Fig. 5). Unexcitable tissues from patch material or myocardial scar along with the tricuspid and pulmonary valve annuli form the channel. Common anatomic boundaries are isthmuses between the tricuspid or pulmonary annulus and septal scar or patch. These channels can be mapped with three-dimensional substrate mapping and connected by ablation lines during sinus rhythm. In one study, the acute success rate was 100%, and the long-term success rate was 91% (mean follow-up: 30.4 + 29.3 months).

Postvalve surgery

VTs after aortic or mitral valve surgery (without CAD) account for 4% of VTs. They have a bimodal presentation and occur either early after surgery (3 to 10 days) or years later (5 to 15 years) [23]. Most VTs are scar-related (70%). Nearly two thirds (64%) of patients have perianular scar in which an identifiable endocardial circuit isthmus (71%) and bundle branch reentry (10%) are present (see Fig. 5). The acute success rate of VT ablation is reported to be 98% [23].

Idiopathic monomorphic ventricular tachycardia in patients with normal hearts

Left ventricular outflow tract ventricular tachycardia and its variant

RVOT VT most commonly arises from RVOT (90%), and less commonly, it may originate from LV outflow tract, mitral annulus, pulmonary artery, aortic sinus of Valsalva, coronary sinus, and coronary veins. In fact, it may originate from

any part of right ventricular or LV myocardium. These patients have frequent PVCs, nonsustained VT in salvos, or sustained VT. In a few patients, it is related to exercise [24]. These VTs or PVCs often are induced with burst pacing and with isoproterenol, adrenaline or aminophylline infusion, and terminated with intravenous injection of adenosine, β -blockers or calcium channel blockers. Success rates of ablation are higher in RVOT VTs and lower in septal VTs. The risks of ablation include RV perforation during RVOT or free wall ablation and complete heart block during septal ablation. The potential for acute occlusion of the left main or right coronary arteries is a major risk consideration, especially with VTs originating from the aortic sinuses of Valsalva. Coronary angiography and intracardiac ultrasound imaging have been used to define the proximity of the coronary ostia to the ablation site. Radiofrequency ablation has been performed safely at sites more than 8 mm away from the coronary artery ostia with careful continuous monitoring of catheter position during RF application. Standard RF ablation with tip temperature maintained at less than 55°C has been suggested to prevent aortic valve damage observed in animal studies. Epicardial origin of VT in close relation to coronary sinus can be ablated by means of the coronary sinus (Fig. 9).

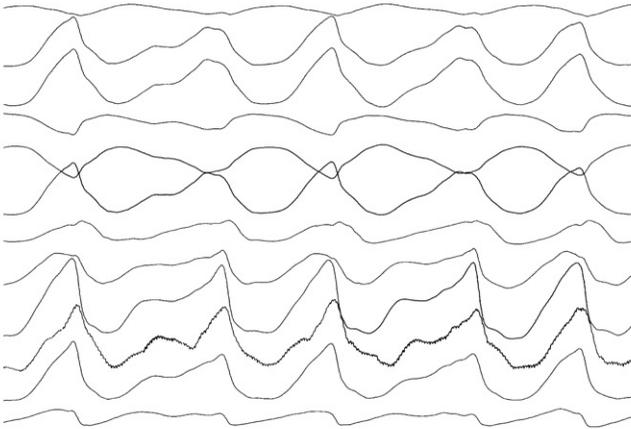
Polymorphic ventricular tachycardia and ventricular fibrillation

The malignant variety of RVOT VT and idiopathic VF that is initiated by a single or two monomorphic PVCs can be ablated successfully with good long-term results (Fig. 10).

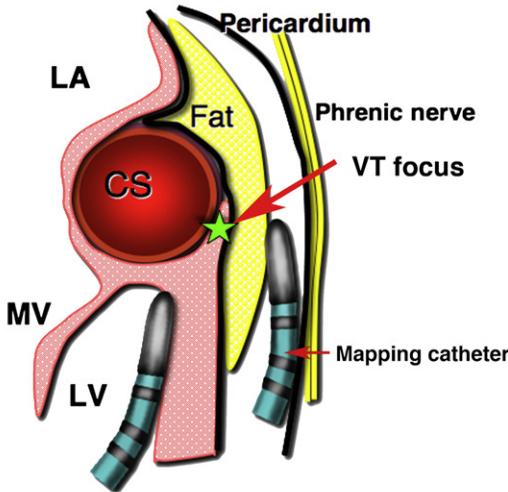
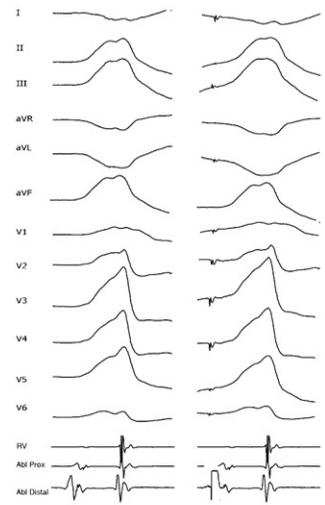
Inherited ventricular arrhythmia syndromes

Symptomatic and high-risk patients who have inherited arrhythmia syndromes are treated with an ICD. VT storm has been shown to have triggers (unifocal PVCs) from the Purkinje arborization or the RVOT. They play a crucial role in initiating VF in patients with long QT and Brugada syndromes. VT storm in Brugada syndrome is managed medically by infusion of isoproterenol. In one study, three patients who had long QT syndrome and four patients who had Brugada syndrome, episodes of polymorphic VT and VF, were associated with frequent isolated or repetitive PVCs, which could be ablated successfully without any recurrence during a follow-up of 17 + 17 months.

Sustained Ventricular Tachycardia



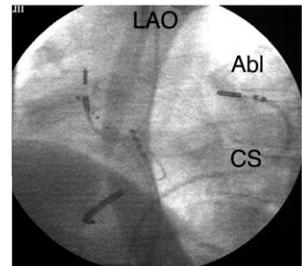
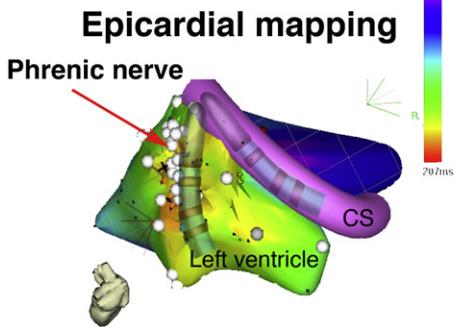
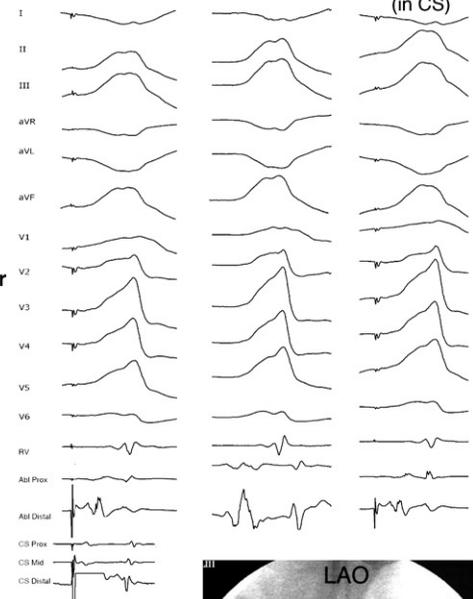
Pace mapping from epicardium



Distal CS pacing

VT

Pacing from ablation catheter (in CS)



Electrical storms or ventricular tachycardia storm

Electrical storm (ES) is an important, independent marker for subsequent death among ICD recipients, particularly in the first 3 months after its occurrence. The development of VT/VF unrelated to ES, however, does not seem to be associated with an increased risk of subsequent death. Short- and long-term efficacy of catheter ablation for ES was studied in patients who had SHD (CAD, DCM and ARVD/C) [25]. After one to three procedures, induction of any clinical VT(s) by programmed electrical stimulation was prevented in 89% patients. VT storm was suppressed acutely in all patients; a minimum period of 7 days with stable rhythm was required before hospital discharge, and 92% patients were free of VT storm and 66% patients free of VT recurrence during a median follow-up of 22 months (range, 1 to 43 months). Eight of 10 patients who had persistent inducibility of clinical VT(s) had ES recurrence; four of them died suddenly despite ICD therapy.

Inducible ventricular tachycardias and ablation end points

Acute success

During EP study, in patients who have VT in the setting of SHD, an average of three different monomorphic VTs, including the clinical VT (VT requiring ablation) and nonclinical VTs, are induced. Of note, in many patients VT cycle length may be the only clue regarding the clinical VT, because the ICD promptly terminates VT. Ablation of incessant VTs and the inducible clinical VT is often an acceptable end point for success. Ablation of nonclinical VTs also is attempted because these VTs may recur subsequently. In one study, at least one VT was no longer inducible after ablation in 73% to 100% of patients, whereas all inducible VTs were abolished in 38% to 95% of patients [2].

Long-term results

VTs that are noninducible after ablation generally have low recurrence rates (less than 3% to

27%), whereas when the targeted VT remains inducible after ablation, the recurrence rate is greater than 60%. However, the frequency of episodes often is reduced in these patients. In a multicenter trial of 146 patients, the immediate effect on inducible VT did not predict outcomes; VT recurred in 44% of patients who had no inducible VT and 46% of those who had inducible VT. The frequency of spontaneous VT during short-term follow-up was reduced by more than 75% in most patients. Recurrence rate is higher after VT ablation in DCM [17].

The annual mortality after VT ablation ranges from 5% to greater than 20%, with death from progressive heart failure being the most common cause. The substantial mortality is consistent with the severity of heart disease and association of spontaneous VT with mortality and heart failure even when VT is treated effectively by an ICD. Older age, greater LV size, and LV dysfunction increase mortality. The potential for ablation to adversely affect LV function is a cause for concern, although assessment of LV ejection fraction after ablation has not shown any deterioration. Confining ablation lesions to regions of low-voltage scar and attention to appropriate medical therapy beneficial to patients who have LV dysfunction are prudent.

Future directions

VT ablation remains a relatively high-risk procedure with a success rate far from the desired because of several factors, including poor LV systolic function (in most patients who have SHD), inability to map the VT circuits with presently available mapping tools, hemodynamic instability, and changing activation patterns. Failure of ablation may be caused by organized thrombus preventing optimum energy delivery to the deeper myocardium, progression of disease, and multiple epicardial VT circuits that are not amenable to successful ablation due to epicardial fat and risk of coronary vessel and phrenic nerve injury. A high risk of recurrence after VT ablation occurs because of continued disease process,

←
 Fig. 9. Mapping and ablation of epicardial ventricular tachycardia (VT). Initial mapping of the monomorphic premature ventricular complexes (PVCs) and VT with alternating QRS morphologies revealed a focal source originating from the epicardial surface of anterolateral mitral annulus. Because of the close proximity to the phrenic nerve (*white dots*), however, PVCs could not be ablated. Later, mapping of the same area was attempted by means of the coronary sinus, which revealed an early presystolic electrogram and an excellent pace map. The PVC was ablated successfully from this site. *Abbreviations:* CS: coronary sinus, LA: left atrium; LV: left ventricle; MV: mitral valve.

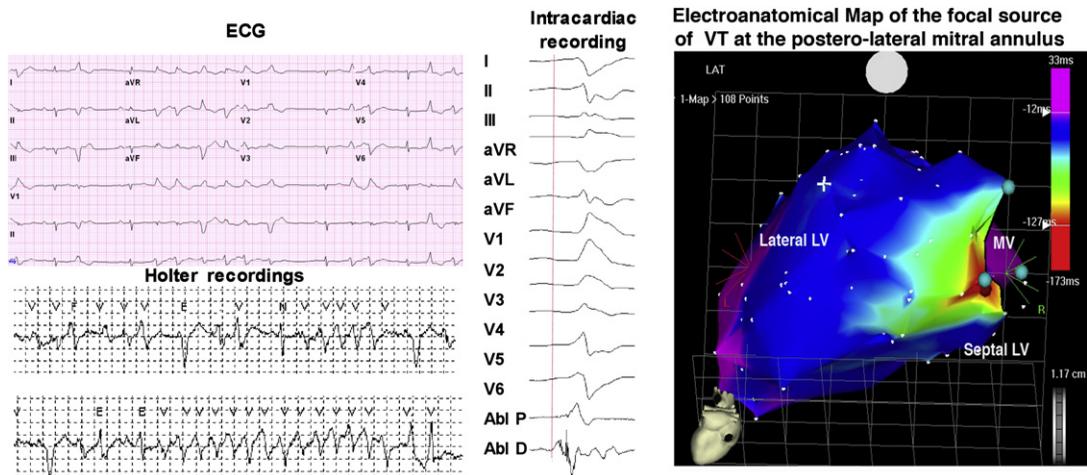


Fig. 10. Focal polymorphic ventricular tachycardia (VT) in a patient with normal heart. The 12-lead ECG shows a single monomorphic premature ventricular complex (PVC) that initiates frequent nonsustained polymorphic VT. The Holter recordings revealed multiple runs of symptomatic rapid nonsustained polymorphic VT. The intracardiac recording revealed a focal source originating from the lateral mitral annulus with a presystolic electrogram preceding the QRS during the premature ventricular complex by 30 milliseconds. Electroanatomical mapping shows a centrifugal activation pattern during the initiating premature complex that was ablated successfully (*red spot*). Follow-up Holter monitor did not show any polymorphic VT. Abl D and Abl P denote bipolar electrogram recordings from distal and proximal poles of ablation catheters, respectively.

electrolyte imbalance, ICD-induced proarrhythmia, and drug noncompliance. Development of better mapping tools (noninvasive and/or invasive modalities) with the capability to incorporate information regarding the substrate and pathophysiology of VT foci or reentrant circuits will reduce the procedure time and improve efficacy. The quest for alternative energy sources such as high intensity focused ultrasound, laser, and microwave are underway. VTs originating from deep within the ventricular myocardium may be treated by needle electrodes. Transcoronary ethanol ablation as an adjunctive therapy has been tried with limited success. Hemodynamic support with a percutaneous LV assist device may be used for ablation in patients with poor LV function. A recently published trial has opened the door for catheter ablation as a primary therapy in CAD patients who have VT.

Summary

Catheter ablation of VA may yield very satisfying palliation in most patients who have SHD and can be curative in those who have normal hearts. Catheter ablation, however, remains a challenging procedure for patients who have hemodynamically unstable VA. The success

rate is less than desirable in patients who have unmappable VT and VT storm. Newer imaging modalities, mapping systems, and ablation techniques may improve the procedural success rate in the future.

References

- [1] Zipes DP. Epidemiology and mechanisms of sudden cardiac death. *Can J Cardiol* 2005;21(Suppl A):37–40.
- [2] Stevenson WG, Soejima K. Catheter ablation for ventricular tachycardia. *Circulation* 2007;115:2750–60.
- [3] Haissaguerre M, Shoda M, Jais P, et al. Mapping and ablation of idiopathic ventricular fibrillation. *Circulation* 2002;106:962–7.
- [4] de Bakker JM, van Capelle FJ, Janse MJ, et al. Reentry as a cause of ventricular tachycardia in patients with chronic ischemic heart disease: electrophysiologic and anatomic correlation. *Circulation* 1988;77:589–606.
- [5] Pogwizd SM, Hoyt RH, Saffitz JE, et al. Reentrant and focal mechanisms underlying ventricular tachycardia in the human heart. *Circulation* 1992;86:1872–87.
- [6] Gottlieb I, Macedo R, Bluemke DA, et al. Magnetic resonance imaging in the evaluation of nonischemic cardiomyopathies: current applications and future perspectives. *Heart Fail Rev* 2006;11:313–23.
- [7] Blanck Z, Dhala A, Deshpande S, et al. Bundle branch reentrant ventricular tachycardia: cumulative

- experience in 48 patients. *J Cardiovasc Electrophysiol* 1993;4:253–62.
- [8] Lopera G, Stevenson WG, Soejima K, et al. Identification and ablation of three types of ventricular tachycardia involving the his-Purkinje system in patients with heart disease. *J Cardiovasc Electrophysiol* 2004;15:52–8.
- [9] Miller JM, Marchlinski FE, Buxton AE, et al. Relationship between the 12-lead electrocardiogram during ventricular tachycardia and endocardial site of origin in patients with coronary artery disease. *Circulation* 1988;77:759–66.
- [10] Badhwar N, Scheinman MM. Idiopathic ventricular tachycardia: diagnosis and management. *Curr Probl Cardiol* 2007;32:7–43.
- [11] Berruezo A, Mont L, Nava S, et al. Electrocardiographic recognition of the epicardial origin of ventricular tachycardias. *Circulation* 2004;109:1842–7.
- [12] Rothman SA, Hsia HH, Cossu SF, et al. Radiofrequency catheter ablation of postinfarction ventricular tachycardia: long-term success and the significance of inducible nonclinical arrhythmias. *Circulation* 1997;96:3499–508.
- [13] Bogun F, Good E, Reich S, et al. Isolated potentials during sinus rhythm and pace-mapping within scars as guides for ablation of postinfarction ventricular tachycardia. *J Am Coll Cardiol* 2006;47:2013–9.
- [14] Reddy VY, Reynolds MR, Neuzil P, et al. Prophylactic catheter ablation for the prevention of defibrillator therapy. *N Engl J Med* 2007;357(26):2657–65.
- [15] Josephson ME, Waxman HL, Cain ME, et al. Ventricular activation during ventricular endocardial pacing. II. Role of pace mapping to localize origin of ventricular tachycardia. *Am J Cardiol* 1982;50:11–22.
- [16] Josephson ME, Horowitz LN, Spielman SR, et al. Role of catheter mapping in the preoperative evaluation of ventricular tachycardia. *Am J Cardiol* 1982;49:207–20.
- [17] Soejima K, Stevenson WG, Sapp JL, et al. Endocardial and epicardial radiofrequency ablation of ventricular tachycardia associated with dilated cardiomyopathy: the importance of low-voltage scars. *J Am Coll Cardiol* 2004;43:1834–42.
- [18] Sosa E, Scanavacca M, d'Avila A, et al. Nonsurgical transthoracic epicardial catheter ablation to treat recurrent ventricular tachycardia occurring late after myocardial infarction. *J Am Coll Cardiol* 2000;35:1442–9.
- [19] Harada T, Tomita Y, Nakagawa T, et al. Pace mapping conduction delay at reentry circuit sites of ventricular tachycardia after myocardial infarction. *Heart Vessels* 1997;(Suppl 12):232–4.
- [20] Delacretaz E, Stevenson WG, Ellison KE, et al. Mapping and radiofrequency catheter ablation of the three types of sustained monomorphic ventricular tachycardia in nonischemic heart disease. *J Cardiovasc Electrophysiol* 2000;11:11–7.
- [21] Reithmann C, Hahnefeld A, Remp T, et al. Ventricular tachycardia with participation of the left bundle-Purkinje system in patients with structural heart disease: identification of slow conduction during sinus rhythm. *J Cardiovasc Electrophysiol* 2007;18:808–17.
- [22] Mazur A, Kusniec J, Strasberg B. Bundle branch reentrant ventricular tachycardia. *Indian Pacing Electrophysiol J* 2005;5:86–95.
- [23] Eckart RE, Hruczkowski TW, Tedrow UB, et al. Sustained ventricular tachycardia associated with corrective valve surgery. *Circulation* 2007;116:2005–11.
- [24] Morin DP, Lerman BB. Management of ventricular tachycardia in the absence of structural heart disease. *Curr Treat Options Cardiovasc Med* 2007;9:356–63.
- [25] Carbucicchio C, Santamaria M, Trevisi N, et al. Catheter ablation for the treatment of electrical storm in patients with implantable cardioverter defibrillators: short- and long-term outcomes in a prospective single-center study. *Circulation* 2008;117:462–9.
- [26] Zipes DP, Camm AJ, Borggrefe M, et al. ACC/AHA/ESC 2006 guidelines for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: a report of the American College of Cardiology/American Heart Association Task Force and the European Society of Cardiology Committee for Practice Guidelines. *Circulation* 2006;114:e385–484.