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Catheter-based Ablation for Ventricular Arrhythmias

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Abstract Catheter ablation for patients with recurrent ventricular arrhythmias has emerged as an important and effective treatment option. The approach to ablation, and the risks and likely efficacy are determined by the nature of the severity and type of underlying heart disease. Although implantable defibrillators remain the corner stone for prevention of sudden cardiac death, ablation successfully reduces tachycardia recurrences and storms of ventricular arrhythmias triggering defibrillator shocks in patients with structural heart disease. Our understanding of idiopathic ventricular tachycardia (VT) has grown substantially with several new sites of VT origin recognized in recent years. Ablation is often curative for idiopathic VT. This review discusses common mechanisms and clues to diagnosis of the various VTs, and current advances in ablation options. In particular, endocardial ablation techniques have been complemented by newer approaches such as percutaneous epicardial ablation. In rare cases, transcoronary alcohol ablation can be effective for life-threatening arrhythmia.

Keywords Ventricular tachycardia · Idiopathic ventricular tachycardia · Catheter ablation · Radiofrequency ablation · Epicardial ablation · Transcoronary ethanol ablation · Ventricular Arrhythmia

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Introduction

Ventricular arrhythmias are typically a manifestation of significant structural heart disease; they are associated with a high risk of sudden cardiac death. Implantable cardioverter-defibrillators (ICDs) remain the mainstay of therapy for these arrhythmias. However, the ICD although effective in aborting sudden cardiac death, has no role in the prevention of ventricular tachycardia (VT) or ventricular fibrillation (VF). Recurrent VT or VF can result in frequent shocks that are painful and reduce quality of life. In addition, there is evidence to suggest that recurrent ICD shocks are a marker for progressive heart failure and increased mortality [1]. Antiarrhythmic drugs and ablation are frequently necessary for control in such situations.

Less frequently, VT or repetitive monomorphic ectopic activity occurs in the absence of structural heart disease. They are termed idiopathic VT and generally carry a benign prognosis. In addition to symptoms associated with the arrhythmias, frequent and repetitive premature ventricular contractions (PVCs) have come to be recognized as a cause of progressive ventricular dysfunction. Suppression of such ectopic activity can potentially reverse cardiomyopathy in some patients [2].

Ablation techniques for VT are largely dependent on the presence or absence of structural heart disease and the type of VT. Most VTs relating to myocardial disease result from reentry involving channels within or around scars or islands of myocardium rendered abnormal by delayed conduction. Such structural changes can result from infarction, inflammation, infiltrative disorders, and familial cardiomyopathies and, following cardiac surgery as in repaired tetralogy of Fallot. Associated VTs are usually monomorphic although, occasionally, degeneration to polymorphic VT can occur

due to break up of a propagating wavefront and spiral wave reentry. Ablation techniques rely primarily on locating scar areas and defining narrow channels where ablation can interrupt reentrant circuits. In contrast, idiopathic VTs most frequently emanate from localized areas of abnormal automaticity in the ventricular myocardium, or due to interfascicular reentry. Frequent PVCs or repetitive ectopic activity tend to be of focal origin; mapping to guide ablation of focal VT relies primarily on detection of early activation and/or identification of sites where pacing generates an electrocardiogram (ECG) that mirrors the VT morphology (pace-mapping). Current indications for VT ablation are shown in Table 1 [3•].

Mapping and Ablation Technologies and Procedural Considerations

Due to the relative safety and simplicity of radiofrequency (RF) current, it remains the most commonly used energy source for ablation and is delivered through electrodes mounted on steerable catheters. Lesion size is limited by coagulum formation on the electrode when temperature exceeds 70°C. Although 4- or 5-mm electrodes maybe sufficient for idiopathic VT ablation, ablation of the thicker substrate of scar-related VTs is facilitated with the use of irrigated electrodes to cool the catheter tip, or 8-mm tip electrodes to effect deeper and larger lesions. Mapping systems enable the creation of a three-dimensional shell of the chamber of interest, and allow for catheter manipulation with limited fluoroscopy. Point-by-point charting of electrograms can create visual maps of voltage (voltage map) or impulse propagation (activation map) on the shell. Often, concomitant use of intracardiac echocardiography aids in the creation of the anatomic shell, visualization of valve structures and papillary muscles, and helps monitor for related complications, such as deteriorating ventricular function and pericardial effusion.

For endocardial ablations, access to the left ventricle is usually obtained retrogradely via the aortic valve. In the event of significant peripheral vascular disease or a mechanical aortic valve, a transeptal approach provides access to the left ventricle by traversing the mitral valve. Deep intramural VTs or those originating from the subepicardium can be approached via the pericardial space. In the absence of prior cardiac surgery or pericarditis, the pericardial space can be accessed via the subxiphoid approach by introducing a needle under fluoroscopy and injection of small amounts of contrast to identify the pericardial space [4]. Once the pericardial space is entered, a guide wire is advanced followed by a sheath for the mapping and ablation catheter. In the absence of pericardial adhesions, catheters can be moved freely on the epicardial surface for mapping. Prior to any ablation, proximity to the coronary arteries is usually assessed by coronary angiography. Ablation close to a coronary artery (within 4 mm) poses a high risk of acute coronary occlusion and should be avoided [5]. Left phrenic nerve injury is another concern and high-output pacing is performed prior to ablation along the anatomical course of the nerve to assess proximity of the nerve [6].

Once catheters are in place, it is common practice to induce the ventricular arrhythmia to confirm diagnosis, define the number of inducible VTs, and assess for hemodynamic stability. Many patients have VTs that are hemodynamically unstable and will need immediate cardioversion. In such cases, the substrate for the VT can be defined during stable sinus or paced rhythm and

Table 1 Indications for catheter-based ablation for VT

Patients with structural heart disease:
1. Symptomatic sustained monomorphic VT that recurs despite antiarrhythmic drug therapy or when drugs are not tolerated or desired
2. Incessant VT or VT storm that is not due to a reversible cause
3. Frequent PVCs, nonsustained VT, or VT that is presumed to cause ventricular dysfunction
4. For bundle branch reentry or interfascicular reentrant VTs
5. Polymorphic VT or VF refractory to antiarrhythmic drugs when there is a suspected trigger that can be targeted for ablation
Patients without structural heart disease:
1. Symptomatic monomorphic VT with severe symptoms
2. Monomorphic VT when antiarrhythmic drugs are not effective, not tolerated, or not desired
3. Sustained polymorphic VT or VF that is refractory to antiarrhythmic drugs when there is a suspected trigger that can be targeted for ablation
Catheter ablation for VT is contraindicated:
1. Mobile ventricular thrombus (epicardial ablation or alcohol ablation can be considered)
2. For asymptomatic PVCs or nonsustained VT that is not causing or contributing to ventricular dysfunction
3. VT due to transient reversible causes or torsade de pointes VT related to prolonged QT

PVC premature ventricular contraction; *VF* ventricular fibrillation; *VT* ventricular tachycardia.

ablation performed to modify the substrate. Occasionally left ventricular (LV) hemodynamic support is used to allow mapping.

Mapping and Ablation Techniques for VT Associated with Structural Heart Disease

Substrate for VT in Structural Heart Disease

Surviving islands of muscle fibers within areas of fibrous tissue form the basis for reentry. Loss of gap junction proteins and cell-to-cell uncoupling within these muscle strands contribute to slow conduction. When interspersed within fixed anatomical barriers or surrounded by tissue that is functionally refractory, slow conducting channels are formed [7]. Models of these circuits propose a central isthmus of slow conduction tissue within islands of conduction block with inner and outer loops (Fig. 1). Some circuits are large, extending over several centimeters. The substrates often support multiple circuits creating VTs of multiple ECG morphologies and cycle lengths. Not infrequently, scars supporting VT border a valve annulus; a typical example is VT utilizing a channel between an inferior wall infarct scar and the mitral annulus.

Electrocardiographic Data

The surface ECG of VT can offer multiple clues to the VT mechanisms and potential site of origin. Sustained monomorphic VT in the presence of ventricular scar, for example, often suggests stable reentrant mechanisms. Polymorphic VT or VF, conversely, can be a manifestation of active ischemia, electrolyte disturbances, or abnormalities of repolarization. During monomorphic VTs, QRS morphology can direct one to the exit site where impulses emerge from a reentrant circuit. A left bundle branch block (LBBB) configuration in lead V1 (dominant S wave) suggests an exit site in the right ventricle or the interventricular septum. A dominant R wave or right bundle branch block (RBBB) pattern in V1 indicates a LV exit site. The QRS axis defines VT origins in the coronal plane; an inferiorly directed QRS axis suggests a superior or anterior wall exit, whereas a superiorly directed axis indicates an inferior wall exit. The precordial leads are more indicative of directionality in the sagittal plane. Deep S waves in the apical leads (V3–V6) indicate earliest activation in the LV apex, whereas prominent R waves in these leads point to a basal origin of activation. It should be borne in mind that areas of scar, conduction block, and abnormal ventricular anatomy can render these rules misleading. Pacing from the mapping catheter during sinus rhythm in an attempt to reproduce the QRS morphology during VT is a better way of determining the anatomic exit location in any particular individual. This method of mapping is termed pace-mapping. Subepicardial origin of VT is suggested by wider QRS complexes and delayed initial upstrokes in the precordial leads [8].

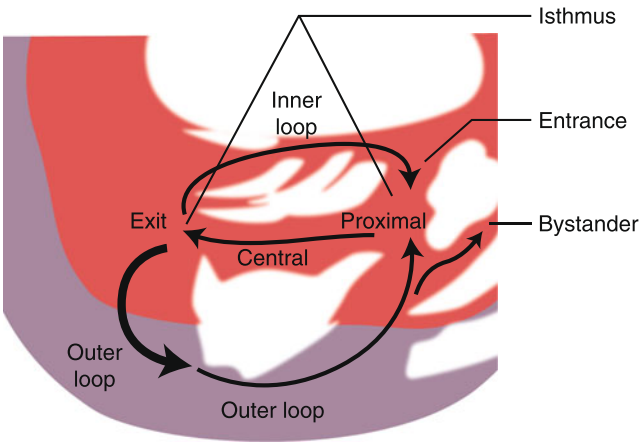

Fig. 1 Shows a reentry loop model for ventricular tachycardia in a segment of ventricle. The *red area* represents an area of infarct zone and the *white areas* denote dense scars forming anatomic boundaries. The reentry circuit has an isthmus with a region of entrance of the impulse and an exit region from which wavefronts propagate to the ventricle to produce a QRS complex. The wavefront returns to the isthmus by conduction through an inner loop within the infarct zone or an outer loop in the border of the infarct zone with normal muscle (*purple area*). Bystander areas may be attached to the reentrant loop

Mapping During Stable VT

A stable clinical VT offers the advantage of mapping during the arrhythmia to identify an isthmus where ablation can terminate VT and render it noninducible. Exit sites of a VT circuit can be identified by electrograms that precede surface QRS complexes. Such presystolic activity can be documented on endocardial sites with multipolar catheters in greater than 85% of cases [9]. Diastolic electrograms during VT indicate sites of impulse propagation in regions of slow conduction activated between QRS complexes. Electrogram timing alone is not entirely reliable as a guide to successful ablation sites due to multiple conduction channels, some of which are bystanders (Fig. 1). Thus, additional information is sought with pacing maneuvers. Pacing with capture from the tip electrode in contact with the tissue of interest indicates electrically excitable tissue. The stimulus to QRS (S-QRS) interval is indicative of the conduction time in the channel between the pacing site and the point of exit of the stimulated impulse. A short S-QRS would suggest a stimulus closer to the exit, whereas a long S-QRS indicates an entrance to the channel. The ability to entrain the tachycardia using criteria set out by Waldo [10] confirms reentry as a mechanism. During VT, pacing at a

rate slightly faster than the VT rate will result in continuous resetting (entrainment) of the VT. If the pacing site is within the inner loop of a circuit, the interval from the last paced complex to the first return cycle (post-pacing interval) will approximate the tachycardia cycle length. Ablation is more likely to terminate tachycardia if the post-pacing interval is within 30 msec of tachycardia cycle length [11]. In addition, the degree of fusion between paced and VT complexes is helpful in locating an isthmus. QRS fusion occurs if the stimulated wavefront alters activation over a large area to change the ECG. If pacing is performed from the isthmus, the stimulated wavefront emerges from the circuit replicating ventricular activation during VT, a process termed entrainment with concealed fusion (ECF) because the fusion between antidromic and orthodromic wavefronts in the circuit is concealed. During ECF, the S-QRS indicates the conduction time between the pacing site and the reentry circuit exit and matches the local electrogram to QRS during VT.

Substrate Mapping and Ablation

The majority of patients (70% to 80%) with scar VT will have hemodynamically instability precluding mapping during prolonged periods of sustained arrhythmia [12•]. Additionally, VTs of multiple morphologies may be present or change from one to another spontaneously or during pacing maneuvers such that mapping during any one stable VT becomes difficult. Finally, a clinical VT may not be inducible at the time of the electrophysiology study. Thus, techniques to target the substrate for VT have been developed.

In substrate mapping, a three-dimensional electroanatomic mapping system is used to define areas of scar by charting electrogram amplitude in a voltage map. In the ventricular endocardium, peak-to-peak bipolar electrogram voltage less than 1.5 V is characterized as areas of scar [3•]. These low-voltage areas contain the reentrant scar but are often too large to cover by catheter-based ablation. Thus, additional markers such as exit points from the circuit or evidence for slow channels are sought. Pace-mapping in the exit sites will replicate the QRS morphology of the VT. An isthmus serving as a channel for reentry is suggested by low amplitude–isolated potentials and late potentials inscribed after the end of the QRS. A stimulated wavefront that emerges from the exit with a long S-QRS and complexes that are similar to the morphology of the VT is strongly suggestive of an isthmus involved in the reentrant VT. Further, areas of dense scar that form borders for circuits can be identified by electrical unexcitability [13]. By marking them on the voltage map, one gets a visual impression of potential channels for directing ablation energy.

Purkinje System VT

A VT is due to a damaged Purkinje system in approximately 8% of patients with structural heart disease and recurrent arrhythmias [14]. VT may be due to catecholamine-sensitive automaticity or reentry involving the bundle branches. Recently, monomorphic PVCs, usually from the Purkinje system, have been shown to initiate VF in patients with no recognized structural heart disease and in patients with ischemic cardiomyopathy [15, 16]. Suppression of recurrent VF can be achieved with catheter ablation of the local Purkinje network origin of the triggering PVCs.

Bundle branch reentry causes sustained monomorphic VT [17]. Most commonly, the circulating wavefront propagates up the left bundle and antegrade via the right bundle resulting in VT with typical LBBB pattern. This VT can be entrained from the RV apex with post-pacing intervals approximating the tachycardia cycle length. A constant relationship of the His deflection preceding the QRS is seen, usually with any oscillation in the H-H interval preceding V-V cycle length alteration. Less frequently, the circuit revolves up the right bundle and down the left producing a typical RBBB-type VT. Ablation of either of the bundle branches terminates tachycardia; ablation of the right bundle is easier and preferable to avoid induction of ventricular dyssynchrony from LBBB. Not infrequently, there is additional conduction system disease and one third of patients will develop heart block requiring pacing support.

Epicardial Ablation

In approximately 10% to 15% of patients, VT circuits are intramural or subepicardial and not accessible with an endocardial catheter [18•]. An epicardial approach for ablation is often needed for VTs due to nonischemic cardiomyopathy or arrhythmogenic right ventricular (RV) cardiomyopathy; it is less frequently required in patients with coronary disease. Once epicardial access is obtained as described above, methods of mapping and ablation are identical to those on the endocardium. Voltage criteria for defining scar on the epicardium are similar to the endocardial electrograms but epicardial fat may mimic low-voltage scars due to insulation of the underlying myocardium [19]. Overall, epicardial scars tend to border a valve annulus especially in patients with nonischemic cardiomyopathy. In patients with prior cardiac surgery, creation of a subxiphoid surgical window can allow access to the pericardial space in some patients. In the presence of dense adhesions, even such access may be limited to the inferior surface of the ventricles and a full thoracotomy may be necessary [20].

Transcoronary Ethanol Ablation

When catheter-based ablation on the endocardial and epicardial surface fails to control VT or is precluded due to access issues, controlled infarction of the VT circuit may be possible by ethanol injection into the branch of an epicardial coronary vessel that supplies the region of interest [21]. Once a suitable branch is identified, the relationship between the branch and a critical portion of the VT circuit is proven by observing that VT terminates with injection of iced saline or by occlusion of the branch by balloon inflation. If VT termination is not achieved, another branch is tested. Once a clear relationship is established, 1 mL of sterile absolute alcohol is injected after balloon occlusion to prevent reflux into other branches. In a series from our center, acute clinical success was obtained in 56% of patients [22]. The need for this approach is infrequent (1% to 2%) and complications include heart block and extension of infarction with further deterioration of LV function. However, in patients with intractable VT unresponsive to traditional ablation techniques, this approach can be lifesaving.

Mapping and Ablation for VT in the Absence of Structural Heart Disease

Idiopathic VT represents approximately 10% of all ventricular arrhythmias encountered by arrhythmia specialists. Most commonly, they occur in young healthy individuals and are precipitated by exercise or emotion. They often present as nonsustained VT in repetitive monomorphic salvos. Less commonly, they can present as paroxysmal sustained tachycardia or with frequent monomorphic PVCs that comprise the majority of QRS complexes. These VTs have specific VT morphologies indicating the likely region of origin. The vast majority (70% to 80%) demonstrate an LBBB inferior axis morphology, indicating origin in the outflow tract most commonly of the right ventricle. Those from the mitral annulus have an RBBB pattern. VT originating from the left posterior fascicle typically displays an RBBB, left superior axis pattern.

Substrate for Idiopathic VT and PVC

The term idiopathic VT includes a collection of different VTs with varying mechanisms. They include outflow tract VTs from the right ventricle or left ventricle, reentrant fascicular VT, mitral annular VT, and papillary muscle VT. The exact mechanisms are not fully understood but response to certain drugs and pacing maneuvers has helped surmise potential mechanisms. The outflow tract VTs cannot be entrained, and tend to respond to adenosine, verapamil, and β blockers—features consistent with cyclic AMP-mediated–triggered automaticity. Induction of the arrhythmia is more likely with burst pacing than programmed extrastimulation and a critical range of paced cycle lengths is often necessary. Isoproterenol infusion is often needed. The fascicular VTs, conversely, are due to localized reentry involving the fascicles, often initiated by programmed stimulation, occasionally during isoproterenol infusion. VT originating from the LV papillary muscle can simulate fascicular VT but have a focal mechanism [23].

Idiopathic VT is a diagnosis of exclusion. Evidence for structural heart disease should be sought and excluded by appropriate diagnostic testing. In particular, arrhythmogenic RV dysplasia, sarcoidosis, and nonischemic cardiomyopathies can manifest as outflow tract VT before other clinical features of the disease become apparent [24]. The presence of any resting ECG abnormality suggests structural heart disease. In addition, patients with structural heart disease may have different VT morphologies during different episodes, whereas patients with idiopathic VT rarely have more than one QRS morphology of VT. The occurrence of superior axis LBBB VT morphologies tends to be associated with structural heart disease [25]. MRI demonstrating delayed enhancement with gadolinium, in keeping with fibrosis, generally indicates a scar-related VT.

Outflow Tract VT

The outflow tract VTs usually emanate from a discrete focus and their origin is suggested by QRS morphology and axis. Approximately 80% of outflow tract VTs arise from the RV outflow tract, the majority from an area 1 to 2 cm caudal to the pulmonary valve. VT from the RV outflow tract has an LBBB pattern with transition in lead V3 or V4. A free wall (rightward) focus is suggested by wider QRS complexes (> 140 msec) and notching in the inferior leads. A more leftward focus produces deeper S waves in leads 1 and aVL, whereas a lower focus in the RV closer to the His bundle is indicated by positive QRS in aVL. Precise mapping for ablation requires inducible VT or PVCs to allow activation mapping. Earliest activation at successful ablation sites precedes surface QRS by 15 to 45 msec. Bipolar electrograms often show sharp rapid deflections and unipolar recordings demonstrate a QS pattern. Pace-mapping to reproduce a 12/12 QRS match while pacing from a suspected site at a rate similar to the VT can be effective in localization when the arrhythmia is not adequately provoked for activation mapping; but it should be recognized that exact pace-maps can sometimes be generated from sites within 4 to 5 mm of earliest activation [26]. RF application often induces acceleration of VT before complete suppression and noninducibility of the VT or PVC.

Less frequently, outflow tract VTs are localized to the left ventricle, valve cusps, or the origin of the great arteries. LV outflow tract VTs can originate from the base of the septum, the free wall just beneath the aortic valve, aortomitral continuity, or LV epicardium. QRS complexes demonstrate an inferior axis but show prominent R waves in V1 or V2. LV epicardial VTs often have QRS complexes that are slurred with delayed early QRS activation in the precordial leads [27]. These VTs require an approach via the great cardiac vein or anterior interventricular vein and by epicardial access as described above.

Aortic cusp VTs emanate from extensions of the ventricular myocardium above the aortic annulus. These foci are approached from the left or right sinus of Valsalva and defined by early activation rather than pace-mapping [28, 29]. Typically, a double-component electrogram is seen in sinus rhythm due to late activation of this muscle bundle. During PVCs or VT, the second component becomes early preceding QRS by 30 to 40 msec. Ablation of these targets requires that the origin of the coronary arteries be clearly defined and a safe distance established. VT can also arise from sleeves of the myocardium extending above the pulmonary valve into the pulmonary artery. Ablation is required within the pulmonary artery for suppression of these rare VTs.

In a recent report of 278 patients undergoing ablation for idiopathic PVCs or VT, 29 (10%) had VT from the lower RV body with half of them arising from regions within 2 cm of the tricuspid valve annulus [30]. All had LBBB morphology. Ablation was effective in preventing long-term recurrence of PVCs or VT in 80% of patients. It is important to exclude arrhythmogenic RV cardiomyopathy in such patients; voltage maps of the right ventricle can be helpful in excluding areas of RV scar.

Mitral Annulus VT

Focal VTs from the region of the mitral annulus accounted for 5% of idiopathic VTs in a Japanese series [31]. Areas of the valve annulus included the anteroseptal region in 58%, and posterior or posteroseptal in the remaining. Similar to aortic cusp VT, a delayed potential is observed during sinus rhythm at the valve annulus. During VT this potential preceded the QRS by as early as 70 msec. The possibility of a remnant of the atrioventricular ring has been suggested as the potential mechanism for these annular VTs. Endocardial ablation is usually successful in suppressing VT; occasionally, an approach via the coronary sinus may be necessary.

Fascicular VT

Belhassen et al. [32] demonstrated that an RBBB, left axis VT occurring in patients with structurally normal hearts could be terminated with intravenous verapamil. This VT typically arises from the posterior fascicle in the LV septum and comprises the majority of idiopathic LV VTs. Reentry involving the fascicle of the LBBB can be demonstrated by multielectrode mapping. The majority of these VTs involve the posterior fascicle, and parts of the circuit can be identified along the inferoseptal aspect of the left ventricle. Two other forms of verapamil-sensitive VTs have been described; a left anterior fascicular VT with RBBB right axis deviation and an upper septal fascicular VT with a narrow QRS configuration and a normal or right axis deviation [33, 34]. For the typical posterior fascicular VT, RF ablation is usually directed at the anterograde Purkinje potentials more apically rather than proximally to avoid injury to the left bundle. Barotrauma from catheter manipulation can terminate tachycardia and render them noninducible. Empiric lines of ablation along the region may have to be created to prevent recurrence.

Papillary Muscle VT

Idiopathic VT arising from the LV papillary muscle is a distinct subgroup and can have a QRS morphology similar to fascicular VT. The arrhythmias can be exercise induced and catecholamine sensitive, but most commonly present as PVCs. They have a focal automatic mechanism. Differentiation from fascicular VT is based on absence of early Purkinje potentials, spontaneous variations in QRS morphology, and early activation located in the region of the papillary muscle identified by echocardiography. Variations in QRS morphology can be subtle and may be due to preferential conduction to different exit sites or multiple regions of origin [23]. Ablation is technically challenging due to unstable catheter contact with the contracting papillary muscle; the use of intracardiac echocardiography is helpful for visualization of catheter positioning. Cooled tip or 8-mm catheters are often necessary for successful ablation due to deeper endocardial origins.

Outcomes of VT Ablation

Outcomes vary depending on the presence or absence of structural heart disease. For VT associated with structural heart disease, ablation typically aims at palliation to reduce recurrent arrhythmias and prevent defibrillator shocks. Thus, interpretation of results of ablation is often clouded by the presence of inducible VTs that have not been seen to occur spontaneously and that are not targeted by ablation. The target VT is rendered acutely noninducible in 70% to 90% of patients [3•, 13, 35].

A recent meta-analysis of four randomized studies and one observational study of VT ablation for structural heart

disease demonstrated a significant 38% reduction in VT recurrence compared with medical therapy [36•]. In addition, adjunctive catheter ablation showed a trend toward reduction in electrical storms provoking multiple ICD shocks. Ablation did not influence mortality in this patient population; over a mean follow-up of approximately 2 years, 12% in the ablation group and 14% in medical therapy group died. In patients with monomorphic VT resulting from infarct scars, there is now substantial experience with VT ablation such that ablation should be considered early in the course of recurrent VT triggering ICD shocks [3•]. Experience with VT ablations for non-ischemic dilated cardiomyopathy, post-surgical correction for congenital heart disease, and primary RV disease is less extensive and confined to data from small single-center series. Acute success rates are comparable but recurrence is higher especially in arrhythmogenic RV cardiomyopathy, although it is hoped that outcomes will be better with increasing use of epicardial ablation for these patients.

Ablation of idiopathic VT is successful in approximately 80% of patients; those arising from the RVoutflow tract and the interfascicular reentrant arrhythmias yield the highest success rates. Failures are usually due to inability to induce the arrhythmia reliably or arrhythmia locations that are beyond safe access with an ablation catheter such as in the epicardial locations close to the coronary arteries.

Avoiding Complications in VT Ablation

Catheter ablation of VT can be challenging. Careful preprocedural evaluation and anticipation of hemodynamic consequences in patients with severe LV dysfunction are important to minimize the risks of serious complications. Procedure-related mortality in patients with structural heart disease approximates 2% to 3% and is primarily related to uncontrollable VT when the procedure fails [3•, 13]. Other complications relate to left heart catheterization and include vascular complications, thromboemboli, cardiac perforation, and valve injury. Damage to conduction system and coronary arteries are particular to the VT locations being targeted. Episodes of induced VT can cause hypotension, myocardial ischemia, and prolonged myocardial stunning. Exacerbation of heart failure may result from VT sustained for mapping and due to fluid administration through the use of externally irrigated ablation catheters. Serious complications are less frequent for idiopathic VT, but RV perforation and tamponade are reported in less than 1% of procedures. Epicardial access is occasionally associated with cardiac perforation. A safe distance from epicardial coronary arteries and the phrenic nerve has to be established by angiography and high-output pacing prior to ablation.

Patients who are being considered for catheter ablation should have a thorough preprocedural evaluation, including tests to exclude significant ischemia and thrombus within the ventricular chambers. If significant vascular disease is suspected, a transseptal approach should be considered. If atrial fibrillation is present, guidelines for anticoagulation should be followed because cardioversion is likely to occur during conversion of VT by external shock.

Conclusions

Catheter ablation is an important treatment option for patients with recurrent ventricular arrhythmias. The approach to ablation and the risks and likely efficacy are determined by the nature of the severity and type of underlying heart disease. It is often curative for idiopathic VT, and successfully reduces VT recurrences and controls incessant VT or VT storm for the majority of patients with structural heart disease.

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References

Papers of particular interest, published recently, have been highlighted as:

- Of importance

1. Poole JE, Johnson GW, Hellkamp AS. Prognostic importance of defibrillator shocks in patients with heart failure. N Engl J Med. 2008;359:1009–17.
2. Takemoto M, Yoshimura H, Ohba Y, et al. Radiofrequency catheter ablation of premature ventricular complexes from right ventricular outflow tract improves left ventricular dilatation and clinical status in patients without structural heart disease. J Am Coll Cardiol. 2005;45:1259–65.
3. • Aliot EM, Stevenson WG, Almendral-Garrote JM, et al. EHRA/HRS expert consensus on catheter ablation for ventricular arrhythmias. Heart Rhyhm 2009;6:886–933. *This is a consensus report from the Heart Rhythm Society and European Heart Rhythm Association on VT ablation with detailed review of VT mechanisms, ablation strategies, indications, and outcomes*.
4. Sosa E, Scanavacca M. Epicardial mapping and ablation techniques to control ventricular tachycardia. J Cardiovasc Electrophysiol. 2005;16:449–52.
5. Robert-Thomson K, Steven D, Seiler J, et al. Coronary artery injury due to catheter ablation in adults: presentation and outcomes. Circulation. 2009;120:1465–73.
6. Fan R, Cano O, Ho SY, et al. Characterization of the phrenic nerve course within the epicardial substrate of patients with non-ischemic cardiomyopathy and ventricular tachycardia. Heart Rhythm. 2009;6:59–64.

7. de Bakker JM, van Capelle FJ, Janse MJ, et al. Slow conduction in the infarcted human heart. "Zigzag" course of activation. Circulation. 1993;88:915–26.
8. Valles E, Bazan V, Marchlinski FE. ECG criteria to identify epicardial ventricular tachycardia in non-ischemic cardiomyopathy. Circ Arrhythm Electrophysiol. 2010;3:63–71.
9. Miller JM, Marchlinski FE, Buxton AE, Josephson ME. Relationship between 12 lead electrocardiogram during ventricular tachycardia and endocardia site of origin in patients with coronary artery disease. Circulation. 1988;77:759–66.
10. Waldo AL. Atrial flutter: entrainment characteristics. J Cardiovasc Electrophysiol. 1997;8:337–52.
11. Stevenson WG, Khan H, Sager P, et al. Identification of re-entry circuit sites during catheter mapping and radiofrequency ablation of ventricular tachycardia late after myocardial infarction. Circulation. 1993;88(Part 1):1647–70.
12. • Stevenson WG, Wilber DJ, Natala A, et al. Irrigated radiofrequency catheter ablation guided by electroanatomic mapping for recurrent ventricular tachycardia after myocardial infarction: the multicenter thermocool ventricular tachycardia ablation trial. Circulation. 2008; 118: 2773–82. *This multicenter observational study of the use of an irrigation RF catheter for ablation of VT is a large experience of endocardial VT ablations for patients with prior myocardial infarction*.
13. Soejima K, Stevenson WG, Maisel WH, et al. Electrically unexcitable scar mapping based on pacing threshold for identification of the re-entry circuit isthmus: feasibility for guiding ventricular tachycardia ablation. Circulation. 2002;106:1678–83.
14. 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.
15. Haissaguerra M, Shoda M, Jais P, et al. Mapping and ablation of idiopathic ventricular fibrillation. Circularion. 2002;106:962–7.
16. Bansch D, Oyang F, Antz M, et al. Successful catheter ablation of electrical storm after myocardial infarction. Circulation. 2003;108:3011–6.
17. Blanck Z, Jazayeri M, Dhala A, Deshpande S, Sra J, Akhtar M. Bundle branch reentry: a mechanism of ventricular tachycardia in the absence of myocardial or valvular dysfunction. J Am Coll Cardiol. 1993;22:1718–22.
18. • Sacher F, Roberts-Thompson K, Maury P, et al. Epicardial Ventricular Tachycardia Ablation. J Am Coll Cardiol. 2010;55: 2366–72. *This multicenter study of epicardial VT ablation outlines the techniques and complication rates in a group of 156 patients*.
19. Cesario DA, Vaseghi M, Boyle NG, et al. Value of high density endocardial and epicardial mapping for catheter ablation of hemodynamically unstable ventricular tachycardia. Heart Rhythm. 2006;3:1–10.
20. Soejima K, Couper G, Coper JM, Sapp JL, Epstein LM, Stevenson WG. Subxiphoid surgical approach for epicardial catheter based mapping and ablation in patients with prior cardiac surgery or difficult pericardial access. Circulation. 2004;110:1197–201.
21. Kay GN, Epstein AE, Bubien RS, et al. Intracoronary ethanol ablation for treatment of recurrent sustained ventricular tachycardia. J Am Coll Cardiol. 1992;19:159–68.
22. Sacher F, Sobieszczyk P, Tedrow U, et al. Transcoronary ethanol ventricular tachycardia ablation in the modern electrophysiology era. Heart Rhythm. 2008;5:62–8.
23. Yamada T, Doppalapudi H, McElderry T, et al. Electrocardiographic and electrophysiological characteristics in idiopathic ventricular arrhythmias originating from the papillary muscle of the left ventricle: relevance for catheter ablation. Circ Arrhythmia Electrophysiol. 2010;3:324–31.
24. Deal BJ, Miller SM, Scagliotti D, Prechel D, Gallastegui JL, Hariman RJ. Ventricular tachycardia in a young population without overt heart disease. Circulation. 1986;73:1111–8.
25. Mehta D, Odawara H, Ward DE, et al. Echocardiographic and histological evaluation of the right ventricle in the ventricular tachycardias of left bundle block morphology without overt cardiac abnormality. Am J Cardiol. 1989;63:939–44.
26. Azegami K, Wilber DJ, Arruda M, Lin AC, Denman RA. Spatial resolution of pacemapping and activation mapping in patients with idiopathic right ventricular outflow tract tachycardia. J Cardiovasc Electrophysiol. 2005;16:823–9.
27. Joshi S, Wilber DJ. Ablation of idiopathic right ventricular outflow tract tachycardia: current perspectives. J Cardiovasc Electrophysiol. 2005;16 Suppl 1:S52–8.
28. Ouyang F, Fotuhi P, Ho SY, et al. Repetitive monomorphic ventricular tachycardia originating from the aortic sinus cusp: electrocardiographic characterization for guiding catheter ablation. J Am Coll Cardiol. 2002;39:5000–508.
29. Hachiya H, Aonuma K, Yamauchi Y, Igawa M, Nogami A, Iesaka Y. How to diagnose, locate and ablate coronary cusp ventricular tachycardia. J Cardiovasc Electrophysiol. 2002;13:551–6.
30. Herendael HV, Garcia F, Lin D, et al. Idiopathic right ventricular arrhythmias not arising from the outflow tract: Prevalence, electrocardiographic characteristics, and outcome of catheter ablation. Heart Rhythm. 2011;8:511–8.
31. Tada H, Ito S, Naito S, et al. Idiopathic ventricular arrhythmia arising from the mitral annulus: a distinct subgroup of idiopathic ventricular arrhythmia. J Am Coll Cardiol. 2005;45:877–86.
32. Belhassen B, Rotmensch HH, Laniado S. Response of recurrent sustained ventricular tachycardia to verapamil. Br Heart J. 1981;46:679–82.
33. Ohe T, Shimomura K, Aihara N, et al. Idiopathic sustained left ventricular tachycardia; clinical and electrophysiological characteristics. Circulation. 1988;77:560–8.
34. Shimioke E, Ueda N, Maruyama T, Kaji Y. Radiofrequency catheter ablation of upper septal idiopathic left ventricular tachycardia exhibiting left bundle branch morphology. J Cardiovasc Electrophysiol. 2000;11:203–7.
35. De Chilllou C, Lacroix D, Klug D, et al. Isthmus characteristics of re-entrant ventricular tachycardia after myocardial infarction. Circulation. 2002;105:726–31.
36. • Mallidi J, Nadkarani GN, Berger RD, et al. Meta-analysis of catheter ablation as an adjunct to medical therapy for treatment of ventricular tachycardia in patients with structural heart disease. Heart Rhythm 2011; 8: 503–510. *This recent publication reviews four randomized clinical trials and one observational study of ablation versus medical therapy for VT.*