Sustained ventricular tachycardia (VT) is an important cause of morbidity and sudden death in patients with heart disease. Implantable cardioverter-defibrillators (ICDs) terminate VT episodes, reducing the risk of sudden death. Recurrent VT develops in 40% to 60% of patients who receive an ICD after an episode of spontaneous sustained VT. A first episode of VT occurs in ≈20% of patients within 3 to 5 years after ICD implantation for primary prevention of sudden death in high-risk groups. ICD shocks reduce quality of life and are associated with an increased risk of death. Antiarrhythmic drug therapy with amiodarone or sotalol reduces VT episodes but with disappointing incidence of side effects and efficacy. Catheter ablation is useful for reducing VT episodes and can be life-saving when VT is incessant.

Idiopathic VTs occur in patients without structural heart disease and rarely cause sudden death. Electrophysiological study with catheter ablation is often warranted to confirm the diagnosis, to provide further evidence for the absence of ventricular scar or other disease, and often to cure the arrhythmia. Ablation is also an option for symptomatic nonsustained VT and frequent ventricular ectopy in these patients.

The appearance of the VT on ECG often suggests its likely cause and associated heart disease (Figure 1). Monomorphic VT has the same QRS complex from beat to beat, indicating repetitive ventricular activation from a structural substrate or focus that can be targeted for ablation. Most are due to reentry through regions of ventricular scar.

Polymorphic VTs have a changing ventricular activation sequence that can be due to functional reentry without a structural target for ablation; myocardial ischemia, acquired long-QT syndrome, and a number of genetic syndromes are causes. Rarely, ablation of the initiating focus is warranted to control frequent episodes.

Technical Considerations and Complications

The ablation procedure proceeds in steps. After vascular access is obtained, the arrhythmia is induced to confirm the diagnosis and to establish whether abolishing inducible VT is a potential end point for the procedure. Mapping is performed to locate the source, followed by ablation. Testing is then performed to assess the effect of ablation.

Mapping

When VT is hemodynamically stable, mapping can be performed during VT. Many patients with heart disease have VTs that are “unmappable” or “unstable” because they require immediate termination because of hemodynamic intolerance, they cannot be reliably induced, or they frequently change from one VT to another. Substrate mapping or multielectrode mapping approaches have been developed to guide ablation of these VTs.

Mapping systems that recreate the geometry of the ventricles from point-by-point sampling while providing continuous display of catheter position are in common use. Electrophysiological data such as the activation time and electrogram amplitude are color-coded for display (Figure 2). Cardiac motion introduces some error. Papillary muscles and other fine anatomic details are not visible. Registration of preacquired computed tomographic or magnetic resonance images on mapping systems shows promise for improving anatomic definition.

Balloon or basket electrode arrays sample from multiple sites simultaneously, potentially allowing activation to be defined from a single beat or brief run of VT. A “noncontact” mapping system mathematically reconstructs potentials from adjacent sites.

Ablation Technologies

The simplicity and safety of radiofrequency (RF) current make it the most common ablation energy. Solid 4- or 5-mm electrodes are used for ablation of idiopathic VTs, but scar-related VTs often require repeated RF applications and larger, deeper lesions that are facilitated by irrigated RF ablation or larger (eg, 8-mm) electrodes. Large electrodes are convenient but reduce mapping precision and have a greater risk of coagulum formation because of the greater temperature disparities across the electrode compared with smaller electrodes. Irrigated and large-tip electrodes are prone to deep heating within the tissue, causing steam formation that can explode through the tissue (“steam pops”). Tamponade can occur but is rare during...


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ablation of scar-related left ventricular (LV) tachycardias. The risk is likely greater in the thin-walled right ventricle (RV) and with power >40 W. External irrigation has been suggested to reduce the risk of thrombus formation, but administration of intravascular saline requires monitoring volume status and often diuresis.16,17

Experience with catheter cryoablation is limited, but it may pose less risk of injury to adjacent coronary arteries for epicardial ablation than RF ablation.18,19

Endocardial Mapping and Ablation Considerations
Access to the LV for endocardial mapping is commonly achieved retrogradely across the aortic valve. Damage to the aortic valve or a coronary artery ostia is possible but rare.20 Significant vascular access complications, including arterial dissection, significant bleeding, and femoral atrioventricular fistulas, occurred in 2.1% of patients in a multicenter study (Table in the online-only Data Supplement).20 A transseptal approach to the left atrium allows access to the LV through the mitral valve for patients with peripheral vascular disease or a mechanical aortic valve or for insertion of multiple mapping catheters.

Cerebral or systemic embolism is reported in 0% to 2.7% of patients (Table in the online-only Data Supplement).20 Echocardiography should be performed before the procedure to detect mobile LV thrombus, which is a contraindication to LV mapping. If laminated thrombus is suspected, a several-week period of anticoagulation with warfarin may be considered in the hope of reducing the presence of friable thrombus, but no data exist assessing this approach.

During LV mapping and ablation, patients are systemically anticoagulated with heparin. After ablation, continued anticoagulation with aspirin or warfarin is recommended depending on the extent of ablation performed. Atrioventricular block can occur with either RV or LV ablation and may be unavoidable when VT originates from the septum.
Epicardial Mapping and Ablation

Endocardial ablation fails if VT originates from a deep intramural or epicardial source.\textsuperscript{5,21–24} Percutaneous epicardial mapping and ablation are performed as described by Sosa and colleagues.\textsuperscript{22} The pericardial space is entered with an epidural needle under fluoroscopic imaging with contrast injection, followed by placement of a guidewire and introducer sheath for the mapping catheter.\textsuperscript{25} In the absence of adhesions, the catheter moves freely in the pericardial space for mapping (Figure 3).\textsuperscript{22} Epicardial ablation with standard solid RF ablation electrodes can be successful, but absence of cooling from circulating blood often results in low-power heating with limited lesion creation, requiring irrigated RF ablation.\textsuperscript{26}

The risk of injury to epicardial coronary arteries is a major concern and is dependent on proximity to the vessel, overlying fat, and vessel diameter, with smaller vessels more susceptible to thermal injury.\textsuperscript{18,19} Proximity to the coronary arteries is assessed from angiography while the ablation catheter is at the target site; ablation directly on major vessels is avoided.

Pericardial bleeding can occur but rarely requires prolonged drainage or surgical treatment. Rarely, puncture of a subdiaphragmatic vessel can cause significant abdominal bleeding. The left phrenic nerve is potentially susceptible to injury as it courses down the lateral aspect of the LV but can be identified by pacing from the ablation catheter. After ablation, symptoms of pericarditis are common but generally resolve within a few days.

Pericardial adhesions after cardiac surgery often prevent percutaneous access, although limited access to the inferior

Figure 2. LV mapping data from a patient with VT due to prior infarction. A voltage map of the LV is present in the middle of the panel. Purple indicates normal bipolar electrogram amplitude $>1.55$ mV. Amplitude diminishes from blue, to green, to yellow, to red. Gray indicates electrically unexcitable scar (EUS). A large anterior wall low-voltage infarct area is present. A shows VT-2, which has a right bundle-branch block inferior axis configuration. Pacing at the superior aspect of the infarct (arrow) reproduces the VT-2 QRS morphology, indicating that the exit for VT-2 is near the pacing site. B and C show pacing at a site in the middle of the infarct that was a channel for VT-1. During pace mapping (B), the delay of 170 ms from the pacing stimulus to the QRS indicates slow conduction through the scar to the margin of the infarct. The paced QRS has a left bundle-branch block superior axis configuration that matched VT-1. Pacing at the site during VT (C) entrains (resets) tachycardia with a long stimulus-to-QRS interval without changing the QRS morphology and with a postspacing interval that matches the VT cycle length.\textsuperscript{26} D shows that the exit for VT-1 was at the inferior margin of the infarct, where presystolic activity is recorded during VT. E shows the potential channel (black arrow) with exits at the superior and inferior septal infarct margin, which likely used a loop along the border of the infarct (white arrow). Potential ablation targets are the exits (red circles) and/or the channel (blue circles).
A direct surgical approach to the pericardial space via a subxiphoid pericardial window or thoracotomy in the electrophysiology laboratory can be used for mapping and ablation.

Ablation of Monomorphic VT in Structural Heart Disease

Patients with VT and structural heart disease often have depressed ventricular function, heart failure, and coronary artery disease. Induced VT is often poorly tolerated, requiring prompt restoration of sinus rhythm, rendering VT unmappable with catheter techniques. Inability to tolerate long episodes of VT was a deterrent to ablation in the past but is now commonly addressed with substrate mapping approaches (see Substrate Mapping below) or multielectrode catheter mapping. Even so, induction of VT is generally needed to confirm the type of VT and to assess the effect of ablation, placing the patient at risk of hemodynamic deterioration and ischemia during periods of tachycardia and hypotension. Assessment and management of these risks begin before the procedure. Intravascular volume status should be optimized. A recent assessment of potential myocardial ischemia with perfusion imaging or angiography is helpful to guide management decisions if hypotension or pulmonary edema occurs during the procedure. Before the procedure, it is desirable to discontinue antiarrhythmic drugs that may interfere with initiation of VTs, but this may not be safe or necessary when VT occurs despite therapy.

Consideration of risks and benefits should be individualized. Procedural risks are likely to be increased in the elderly and in patients with severe underlying heart disease. In many patients, VT recurrences are acceptably reduced by antiarrhythmic drug therapy, and ablation is not required. Recurrent VT episodes can be a marker for deterioration of heart failure and mortality risk despite an ICD. Some patients warrant assessment for cardiac transplantation or LV assist device placement so that the eligibility of these options is known in the event of further deterioration.

Scar-Related Sustained Monomorphic VT

Scar-related reentry is the most common cause of sustained monomorphic VT in patients with structural heart disease. Causes include old myocardial infarction, cardiomyopathies, and surgical incisions. Ventricular scars are composed of variable regions of dense fibrosis that create conduction block and surviving myocyte bundles with interstitial fibrosis and diminished coupling, which produce circuitous slow-conduction paths that promote reentry. Many scar-related reentry circuits can be modeled as having an isthmus or channel composed of a small mass of tissue that does not contribute to the surface ECG. The QRS complex is inscribed when the excitation wavefront emerges from an exit along the border of the scar and spreads across the ventricles (Figure 2). Reentry circuit configurations and locations vary from patient to patient. Large macroreentry circuits spanning several centimeters are common causes of
slow VTs.

Repeated programmed stimulation typically induces >1 monomorphic VT (Table in the online-only Data Supplement). Multiple VTs can be due to different circuits in widely disparate areas of scar, different exits from the same region of scar, or changes in activation remote from the circuit due to functional regions of block. Ablation at 1 region often abolishes >1 VT.

QRS Morphology as a Guide to the VT Exit

The QRS morphology of VT is an indication of the location of the circuit exit (Figure 1). VTs with a left bundle-branch block–like configuration in lead V1 have an exit in the RV or interventricular septum; dominant R waves in V1 indicate an LV exit. The frontal plane axis indicates whether the exit is on the inferior wall, which produces a superiorly directed axis, or on the anterior (cranial) wall, which produces an inferiorly directed axis. The mid precordial leads, V3 and V4, provide an indication of exit location between the base and apex; apical exits generate dominant S waves; basal exits generate dominant R waves. VTs that originate in the subepicardium generally have a longer QRS duration and slower QRS upstrokes in the precordial leads than those with an endocardial exit.

Mapping During VT

When VT is stable for mapping, an isthmus can be targeted during VT. In activation sequence maps, the circuit exit and isthmus are identified from presystolic (before the QRS onset) and diastolic (earlier than presystolic) activation, respectively, and are confirmed with pacing maneuvers such as entrainment (Figure 2D).

Multielectrode and noncontact mapping systems identify endocardial exit regions of presystolic electric activity in >90% of VTs. Some diastolic activity is identified in approximately two thirds of patients, but complete reentry circuits are defined in <20%.

Because of heterogeneity of scars with multiple potential conduction pathways and channels, electrogram timing alone is not a reliable guide for targeting a specific reentry circuit isthmus. Confirmation that a site is involved in the reentry circuit can be obtained by pacing at the site (entrainment mapping; Figure 2) or reproducible VT termination by mechanical pressure at the site. It is not necessary to define the entire circuit if an isthmus can be identified for ablation.

Substrate Mapping

For multiple and unstable VTs, substrate mapping can be used to identify regions of scar and potential reentry circuit channels during stable sinus or paced rhythm. Voltage maps are created from 3-dimensional anatomic plots of electrogram amplitude (Figures 2 and 3). Low-voltage regions (<1.55 mV in bipolar recordings) identify areas of scar.

The low-voltage region contains the reentry circuit but is usually too large for ablation of the entire region or its circumference. Additional markers of the circuit exit or isthmus are sought for ablation. During sinus rhythm the exit can often be located by pace mapping along the scar border (Figure 2A). Pacing in the exit region replicates the QRS morphology of VT.

A potential isthmus or channel within low-voltage regions can also be identified during sinus rhythm, suggested by low-amplitude isolated potentials and late potentials inscribed after the end of the QRS complex. Pacing in a channel produces a QRS that emerges after a delay due to slow conduction through the channel (Figure 2B). When the stimulated wavefront propagates through a reentry circuit exit region, the paced QRS morphology resembles VT, strongly suggesting that the pacing site is in a reentry circuit isthmus. If the wavefront leaves the scar by another path, the paced QRS morphology may differ from VT or resemble a different VT. Absence of channels may indicate that functional block is present during VT or that the channels are not endocardial.

Areas of dense fibrosis that form some reentry circuit borders can be detected as electrically unexcitable scar, where pacing does not capture (pacing threshold >10 mA at 2-ms pulse width). Marking electrically unexcitable scar areas (gray area on the voltage map in Figure 2) creates a visual map of potential channels, which can then be investigated for ablation. Not all scars have large electrically unexcitable scar areas; narrow bands of fibrosis likely escape detection on the basis of pacing threshold. Areas of block can also be indicated by very-low-amplitude regions on either side of a larger-amplitude channel.

Most recent ablation series include patients with multiple and unstable VTs targeted by these techniques (Table in the online-only Data Supplement). When VT is stable, substrate mapping during sinus rhythm can be used to identify regions for further evaluation during VT, minimizing the time spent in VT. Brief entrainment is often possible, even for unstable VTs, to confirm the location of a reentry circuit, potentially allowing ablation with a smaller number of RF lesions than when ablation is guided only by substrate mapping.

Inducible VTs and Ablation End Points

On average, 3 different monomorphic VTs are usually inducible with repeated programmed stimulation (Table in the online-only Data Supplement). VTs that have been observed to occur spontaneously are often referred to as “clinical VTs”; the others are often designated “nonclinical,” implying that they are unlikely to occur spontaneously. This distinction can be problematic; nonclinical VTs may subsequently occur spontaneously. In many patients, the ECG morphology of spontaneous VT is not known because an ICD promptly terminates VT.

Abolishing incessant VT and inducible clinical VT is generally the minimum end point that defines a successful procedure. Some centers attempt to abolish all inducible VTs or all inducible mappable VTs, aiming to reduce recurrences. These different end points have not been compared directly. After ablation, at least 1 VT is no longer inducible in 73% to 100% of patients, and all inducible VTs are abolished in 38% to 95% of patients (Table in the online-only Data Supplement). Remaining inducible VTs are often faster and are induced by aggressive stimulation.
When the targeted VT remains inducible after ablation, the recurrence risk exceeds 60%. Absence of inducible VT has been associated with a lower but still significant incidence of recurrence, ranging from <3% to 27% in single-center reports. Inducible, nonclinical VTs are associated with increased risk of recurrence in some studies. Healing of initial ablation lesions and reduction of antiarrhythmic medications likely contribute to recurrences, and some patients benefit from a repeat procedure. Even when VT recurs, the frequency of episodes is often reduced. In a multicenter trial in 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 >75% in the majority of patients.

**Purkinje System VT**

A diseased Purkinje system causes VT in ~8% of patients with structural heart disease referred for catheter ablation. VT can be due to macroreentry or focal automaticity that is catecholamine sensitive. VT has an ECG appearance of typical left or right bundle-branch block and is often associated with severely depressed LV function.

In bundle-branch reentrant VT, the circulating wavefront propagates up the left bundle branch and antegrade down the right bundle branch, producing VT with a QRS configuration of typical left bundle-branch block. Less frequently, the circuit revolves in the opposite direction, producing a right bundle-branch block configuration. Ablation of the right or left bundle branch is curative, but ablation of the right bundle is generally preferred to avoid hemodynamic consequences of left bundle-branch block. Associated infranodal conduction delay and the frequent presence of other, scar-related VTs in approximately a third of patients warrant pacemaker or ICD implantation as well.

**Ablation in Specific Diseases**

**Prior Myocardial Infarction**

Patients have generally been referred after failed drug therapy and ICD implantation. Ablation is initially successful, abolishing 1 or more VTs in 77% to 95% of patients (Table in the online-only Data Supplement). During follow-up, previously ineffective antiarrhythmic drugs, frequently amiodarone, are often continued. VT recurs in 19% to 50% of patients, although the frequency is reduced in the majority. Multiple morphologies of VT and unstable VTs are associated with a higher recurrence risk. Failure of endocardial ablation can be due to intramural or epicardial reentry circuits. Epicardial circuits are present in 10% to 30% of patients, more often in inferior wall as opposed to anterior wall infarctions.

Major complications occur in ~5% to 10% of patients (Table in the online-only Data Supplement), including cardiac tamponade, shock, stroke, aortic valve injuries, and vascular injuries. Procedure mortality, 2.7% in I trial, is often due to failure to control VT rather than complications.

The annual mortality after catheter ablation ranges from 5% to >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 and greater LV size and dysfunction increase mortality. The potential for ablation to adversely affect LV function is cause for concern, although assessment of LV ejection fraction after ablation has not shown deterioration. Confining ablation lesions to regions of low-amplitude scar and attention to appropriate medical therapy beneficial to patients with LV dysfunction are prudent.

**Dilated Cardiomyopathy Without Coronary Artery Disease**

Sustained monomorphic VT is not common in nonischemic dilated cardiomyopathies, but ~80% of those that occur are due to scar-related reentry, with the remainder due to bundle-branch reentry or a focal origin. Myocardial scar has been demonstrated with magnetic resonance imaging and is frequently abutting a valve annulus. Progressive replacement fibrosis is a likely cause. VT ablation is often more difficult than in coronary artery disease, but recurrent arrhythmias are controlled in >60% of patients (Table in the online-only Data Supplement). Reentry circuits require epicardial ablation in a third or more of patients (Figure 3). Occasionally, incessant idiopathic VT or ventricular ectopy causes a tachycardia-induced cardiomyopathy that improves after ablation.

**RV Cardiomyopathies**

Scar-related RV tachycardias occur in idiopathic cardiomyopathy, arrhythmogenic RV dysplasia/cardiomyopathy, and cardiac sarcoidosis. VT has a left bundle-branch block configuration. Reentry circuits are often in scars adjacent to the tricuspid or pulmonic annulus. Focal origin VTs or epicardial reentry with a focal endocardial breakthrough also occurs. Areas of scar associated with VT can also be identified from delayed enhancement on magnetic resonance imaging. Ablation is often successful in the short term, but late arrhythmia recurrences are common, suggesting disease progression (Table in the online-only Data Supplement). Cardiac perforation is the major complication but is infrequent.

**Repaired Congenital Heart Disease**

Scar-related VT involving RV regions of repair also occur late after surgical correction of congenital heart disease, notably tetralogy of Fallot. Catheter ablation is feasible and can reduce recurrences, but in a small series only 43% of 14 patients with tetralogy of Fallot remained free of VT. Dense fibrosis or synthetic patches used in the repair may hinder ablation in some patients.

**Idiopathic Monomorphic VTs**

Idiopathic monomorphic VTs tend to present in young, healthy individuals, often precipitated by exertion or emotion. The most common forms have a focal origin in the RV or LV outflow regions. Up to 12% are due to reentry involving LV Purkinje fascicles. Initial evaluation should exclude structural heart disease, particularly arrhythmogenic RV dysplasia (see RV Myocardiopathies above), cardiac sarcoidosis, and other cardiomyopathies.
mality should increase suspicion for underlying structural disease. Areas of delayed enhancement consistent with fibrosis on magnetic resonance imaging suggest scar-related VT. Magnetic resonance imaging in patients with idiopathic RV outflow tract (RVOT) VTs often shows focal areas of thinning, fatty infiltration, and diminished wall motion that can be normal variants. An acquired somatic cell mutation in the mechanism. The prognosis is good and the risk of sudden death is remote provided that underlying heart disease is excluded. Rarely, an RVOT focus is the trigger for idiopathic ventricular fibrillation or polymorphic VT in the presence of an inherited arrhythmia syndrome. Incessant VT or extremely frequent ectopy can cause depressed LV function that improves after the arrhythmia is controlled. Long-term therapy with β-adrenergic blockers, calcium channel blockers, or both or with a membrane active antiarrhythmic drug is often effective. Catheter ablation is an accepted therapy for sustained or symptomatic antiarrhythmias when medications are ineffective or not desired.

**Outflow-Type Idiopathic VT**

Outflow type VTs have an inferiorly directed frontal plane axis (Figure 1). Most originate in the RV and have a left bundle-branch block configuration in V1. VT is often induced with isoproterenol infusion and burst pacing and can be terminated with high-dose adenosine, consistent with cAMP-mediated delayed afterdepolarizations and triggered activity as the mechanism. An acquired somatic cell mutation in the inhibitory G protein G-α-12 has been found in the RV in some patients.

Clinical presentations include paroxysmal exercise-induced VT, repetitive monomorphic VT, with bursts of nonsustained VT separated by 1 or more sinus beats, and frequent premature ventricular beats. The location of the discrete VT focus is suggested by the ECG, but the closest anatomic relations of the RVOT, LV outflow tract, and their great vessels preclude definitive localization from the ECG alone. Success rates for catheter ablation range from 85% to 97%. Long-term follow-up is limited, but recurrence rates are generally low.

The variety of potential locations makes it desirable to perform mapping and ablation during VT. Inability to initiate the arrhythmia in the laboratory, likely related to sedation and systemic absorption of local anesthetic, is a major cause of ablation failure. The first step is to induce VT, confirm the diagnosis, and obtain a 12-lead ECG to allow pace mapping to guide ablation if VT becomes quiescent.

**RVOT Tachycardia**

The free wall of the RVOT is oriented diagonally from posterior to anterior as a catheter moves from right to left. The side of the RVOT that is opposite the free wall sits anterior to the aorta and superior to the LV outflow tract. VT originating from the RVOT has a left bundle-branch block configuration in V1, with a transition to more positive QRS by V4. A free wall origin is suggested by QRS duration >140 ms 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 RVOT. Precise localization relies on combined use of pace mapping and activation mapping. In most patients, a site with a pace map QRS that exactly matches that of VT can be identified at or very close to the VT focus, but excellent pace maps may be obtained several millimeters distant from the earliest activation. Earliest activation during VT or premature ventricular contractions from the focus typically precedes the QRS onset by 15 to 45 ms, with a characteristic QS complex in the unipolar signal. RF application often produces acceleration followed by termination of VT and renders it no longer inducible.

Successful ablation is achieved in 65% to 97% of patients. Serious complications are infrequent but include perforation of the RV free wall. Although the left main coronary artery can be in close proximity to the posterior aspect of the RVOT, we are not aware of any cases of recognized coronary injury. Infrequently, the focus is adjacent to the His bundle, where heart block is a risk of ablation.

**Pulmonary Artery VTs**

VT can originate from sleeves of myocardium extending along the pulmonary artery, above the pulmonary valve, requiring ablation from within the pulmonary artery. VT typically has large R waves in the inferior leads and greater R/S ratio in lead V2 than in RVOT VT. Ablation risks are not well defined for these infrequent VTs.

**LV Outflow Tract VTs**

LV outflow tract VT can originate from the superior basal region of the left interventricular septum or LV free wall, aortic sinuses of Valsalva, or LV epicardium. The QRS has an inferior axis, but the precordial transition to positive R waves is earlier, with prominent R waves often present in V1 or V2 compared with RVOT tachycardia (Figure 1).

LV endocardial VTs originate from foci below the aortic valve. Heart block and aortic valve and coronary artery injury are potential risks but are reported rarely.

**Aortic Cusp VTs**

VT originating from extension of ventricular myocardium above the aortic annulus, requiring ablation from the left or right sinus of Valsalva, caused 21% of idiopathic VTs in 1 series. Pace mapping in the aortic sinus may require high output and may not exactly reproduce the VT QRS. Activation mapping is required and typically shows a 2-component electrogram with the earliest deflection preceding the QRS complex by an average of 39 ms.

The potential for acute occlusion of the left main or right coronary arteries is a major risk consideration. Coronary angiography and intracardiac ultrasound imaging have been used to define the proximity of the coronary ostia to the ablation site. RF ablation has been performed safely at sites >8 mm below the coronary artery ostia with careful continuous monitoring of catheter position during the RF application. Standard RF ablation with tip temperature maintained at <55°C has been suggested to prevent aortic valve damage observed in animal studies.
Epicardial Outflow Tract VTs

Epicardial foci cause VTs with a QRS onset that is often slurred, creating a pseudo–delta wave appearance. The interval from the onset of the QRS to earliest maximal deflection in the precordial leads is delayed, consistent with late access of the wavefront to the endocardial Purkinje system. The VT focus may be adjacent to an epicardial vein that can be cannulated via the coronary sinus, and successful ablation has been performed via this route as well as via percutaneous pericardial access. When proximity to epicardial coronary artery precludes catheter ablation, a direct surgical approach can be an option.

Mitral Annulus VT

Focal VTs from the mitral annulus accounted for 5% of 352 idiopathic VTs in 1 series. VT has a right bundle-branch block or RS pattern in V1 and monophasic R or RS pattern in leads V2 to V6 (Figure 1). The QRS axis depends on the location on the annulus. Endocardial ablation is usually successful; ablation from within the coronary sinus may be required occasionally.

LV Fascicular Idiopathic VT

Verapamil-sensitive idiopathic LV tachycardia typically presents as exercise-related VT between the ages of 15 and 40 years; 60% to 80% of patients are male. VT has a right bundle-branch block configuration, most commonly with a superiorly directed frontal plane axis (Figure 1). VT is due to reentry involving fascicles of the left bundle and an abnormal region, possibly also in the Purkinje system, with slow conduction that is sensitive to verapamil and that is depolarized early in diastole in VT and in sinus rhythm. More than 80% of these VTs involve the posterior fascicles of the left bundle, identified along the inferoseptal aspect of the LV.

Ablation targets the anterograde Purkinje potentials or diastolic potentials during VT, avoiding the proximal Purkinje system to avoid causing left bundle-branch block. Mechanical trauma from catheter manipulation often terminates VT and prevents reinitiation. Ablation then targets the site of mechanical termination, low amplitude, or diastolic sinus rhythm potentials or creates a line of lesions through this region. Efficacy is greater than 80%. Complications are infrequent.

Ablation for Polymorphic VT and Ventricular Fibrillation

Recurrent polymorphic VT causing “electrical storm” not due to ongoing acute ischemia is rare but is seen in idiopathic ventricular fibrillation, long-QT syndromes, Brugada syndrome, and myocardial infarction. VT is often initiated by premature beats from 1 or a few foci that can be ablated if they occur with sufficient frequency to be located (Figure 4). Most appear to originate from the Purkinje system and have sharp presystolic potentials recorded from the focus. Less frequently, an RVOT focus is a trigger. Successful ablation requires the presence of spontaneous ectopic beats for mapping. Electrical storms can wax and wane with long periods of quiescence. Immediate transport of the patient to the laboratory when the arrhythmia is active is warranted if ablation is to be attempted. In selected patients, excellent outcomes can be achieved, with 90% of patients free from recurrent arrhythmias during follow-up, but the number of patients reported is small.

Summary

Advances over the past decade now allow ablation of multiple, hemodynamically unstable, epicardial, and polymorphic VTs, formerly considered unmappable. Specific locations for the origins of idiopathic VT outside of the RVOT have been defined that can be expected to improve overall success for ablation of these arrhythmias. Reported outcome data are from highly specialized and experienced centers and likely suffer from reporting bias. These procedures are often more difficult than ablation of many supraventricular tachycardias and are best approached by experienced operators in experienced laboratories. The field continues to benefit from technological developments. When the expertise is available,
catheter ablation should be considered earlier in the therapeutic armamentarium for treatment of recurrent VT.

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