During the past few decades, cardiac electrophysiology (EP) has evolved from a specialty that primarily implanted simple pulse generators (pacemakers) for bradycardia therapy to a specialty with an expanded practice in detecting and terminating arrhythmogenicity via complex catheter-based techniques. Representitive of this development are the rapidly evolving treatment options for patients with ventricular tachycardia and fibrillation (VT/VF), the most common cause of sudden cardiac death. Percutaneous catheter ablation has not proven to be an adequate treatment option for many of these patients. Although implantable cardioverter defibrillators (ICDs) are effective in terminating VT/VF, repetitive shocks significantly impair quality of life, and inappropriate ICD shocks in patients with heart failure are associated with increased mortality.1 Percutaneous catheter ablation has evolved as a promising therapy, particularly in patients with recurrent hemodynamically unstable VT refractory to drugs. Developed in the 1970s to guide surgical ablation,2–4 the first percutaneous catheter VT ablation was reported in 1983 in a patient with focal VT originating in the right ventricular outflow tract.5 Since then, the field of VT catheter ablation has seen tremendous growth, partially due to an increase in survival of patients with heart failure and structural heart disease.

This review provides a comprehensive update on currently performed VT ablation procedures and their anesthetic considerations.

VT CLASSIFICATION AND DEFINITIONS

VT is defined as 3 or more consecutive QRS complexes at a rate of 100 beats per minute or more. Different classifications of VT, as well as terminology used in the VT ablation setting, have been well described.6-7 Some of the more frequently encountered terms and definitions associated with VT and ablations are presented in Table 1. Knowledge of specific VT-related terminology is important when planning for an anesthetic, providing information regarding the planned ablation procedure including access route, expected hemodynamic stability (mapping strategy), and inducibility of VT.

PATHOGENESIS OF VENTRICULAR TACHYARRHYTHMIAS

VT and VF manifest as a result of dynamic and complex interactions between an arrhythmogenic substrate (e.g., border zone of a myocardial scar) and electrophysiologic properties of the myocardium (i.e., electrical activation and propagation). Modulating (e.g., increased sympathetic activity, ongoing ischemia, heart failure exacerbation) and triggering factors (e.g., acute electrolyte imbalance, premature ventricular contractions, increased heart rate) further contribute to the clinical manifestation of a VT.5 VTs can occur even in the absence of structural heart diseases, as seen in the genetic channelopathies or in idiopathic/functional VTs.

Regardless of the presence or absence of structural heart disease, the majority of patients presenting for VT catheter ablation have recurrent monomorphic VT (Fig. 1). The mechanism of VT may be either focal or reentrant. Focal VT may arise due to abnormal automaticity or triggered activity from small areas within the myocardium (e.g., right ventricular or left ventricular outflow tract, Fig. 2).6 In patients with ischemic heart disease, acute, delayed, and chronic phases have been associated with different mechanisms of VT and VF.8-17 Recurrent or persistent ischemia, decreased myocardial function,18 and eventually remodeling processes,19 all contribute to late or chronic VT. Areas of normal myocardium adjacent to unexcitable scar tissue are believed to form the substrate of complex reentrant circuits that are able to sustain VT (Fig. 3).20-23 Although initially described in patients with coronary artery disease, reentrant VT may arise from any disease process that results in myocardial scar. Scar-related VT has been described and successfully ablated in patients with dilated cardiomyopathy,
sarcoidosis, arrhythmogenic right ventricular dysplasia/ cardiomyopathy, and hypertrophic cardiomyopathy, which all have been amenable to catheter ablation.24–29

Localizing the areas from where the VTs originate with the help of 3-dimensional (3D) electroanatomical mapping is clinically important and is the basis of catheter-based VT ablation, which will be discussed in more detail.

ABLATION STRATEGIES

Indications for catheter-based ablation of VT have been defined by the EP specialty societies.6 The specific approach (i.e., choice of the mapping and ablation strategy), however, varies and depends on the underlying etiology, mechanism of VT/VF, specific arrhythmogenic substrate, previous attempts or failed ablations, as well as practitioner preferences. It is important for the anesthesiologist to know the etiology of VT and have a basic understanding of the planned ablation strategy, which will not only determine the type of anesthesia but also the choice of hemodynamic monitoring and expected hemodynamic disturbances.

Access

Catheter ablation procedures typically require femoral vascular access. The left side of the heart can be accessed retrograde from the descending aorta or via a transseptal approach from the right atrium. If the VT cannot be terminated from an endocardial approach, and particularly if the source of VT is located closer to the epicardium, then epicardial ablation may be attempted.30–32 Epicardial access is typically established percutaneously from the subxiphoid region using a Tuohy needle.33 However, in patients with a history of cardiac surgery, or inflammatory processes that cause pericardial adhesions, surgical access might be required, via a limited anterior thoracotomy,35 left thoracotomy,36 sternotomy, or surgical subxiphoid window,37 with obvious implications for anesthetic management.

Mapping

Bipolar electrograms (EGMs) recorded by the mapping catheter can help characterize the arrhythmogenic substrate. For instance, low-voltage (amplitude) EGMs represent infarct areas (scar defined as bipolar EGM voltage <0.5 mV, border zone 0.5–1.5 mV, normal myocardium >1.5 mV).38 Within the border zone of the scar, abnormal myocardial fibrils are interspersed among fibrotic tissue, causing electrical activity to proceed in a slow and roundabout manner. This slow conduction is a necessary component of reentrant VT. Critical components of reentrant VT circuits can be identified by the presence of late potentials (i.e., isolated potential occurring 10 milliseconds after the end of the QRS complex)6 or fractionated EGMs (i.e., high-frequency, multicomponent signals of long duration and low voltage), which are targets for catheter ablation.39–41

To facilitate catheter ablation, real-time electroanatomical maps are created using 3D mapping systems (i.e., Carto 3D Mapping System, Biosense Webster, Diamond Bar, CA; EnSite NavX, St. Jude Medical, Inc., Saint Paul, MN)42,43 These systems allow for exact localization and orientation of the catheter tip inside a virtual 3D reconstruction of the

Table 1. Ventricular Tachycardia Definitions and Classification

<table>
<thead>
<tr>
<th>Duration</th>
<th>Definition</th>
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<tbody>
<tr>
<td>Sustained VT</td>
<td>VT lasting longer than 30 seconds and requiring intervention</td>
</tr>
<tr>
<td>Nonsustained VT</td>
<td>VT terminating spontaneously within 30 seconds</td>
</tr>
<tr>
<td>VT storm</td>
<td>3 or more separate episodes of sustained VT within 24 hours each requiring intervention</td>
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<table>
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<tr>
<th>Morphology</th>
<th>Definition</th>
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<tbody>
<tr>
<td>Monomorphic VT</td>
<td>VT of similar beat to beat QRS configuration</td>
</tr>
<tr>
<td>Polymorphic VT</td>
<td>VT with continuously changing QRS configuration</td>
</tr>
<tr>
<td>Pleomorphic VT</td>
<td>&gt;1 specific QRS configuration without continuous change</td>
</tr>
</tbody>
</table>

<table>
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<tr>
<th>Hemodynamic characteristics</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemodynamically stable VT</td>
<td>VT that may pass unnoticed or only cause palpitations</td>
</tr>
<tr>
<td>Hemodynamically unstable VT</td>
<td>VT requiring immediate termination</td>
</tr>
<tr>
<td>Unmappable VT</td>
<td>VT that cannot be induced and mapped while in VT due to hemodynamic instability</td>
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<tr>
<th>Underlying mechanism</th>
<th>Definition</th>
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<tbody>
<tr>
<td>Scar-related VT</td>
<td>Myocardial scar-related reentry circuits as the source of VT</td>
</tr>
<tr>
<td>Focal VT</td>
<td>Focal sites of VT initiation</td>
</tr>
</tbody>
</table>

VT = ventricular tachycardia.
ventricular cavities. Every time the catheter touches the endocardium, the exact catheter tip position within the 3D model, as well as the local unipolar or bipolar EGMs including voltage and local activation time (compared with a reference point), is recorded and added to the map. Maps typically display color-coded representation of the activation sequence (activation map) (Fig. 4) and voltage (Fig. 5).

The following mapping strategies are commonly used either by themselves or as a combination during VT ablation: activation mapping, entrainment mapping, pace mapping, as well as substrate mapping techniques.

Activation mapping refers to mapping during VT (either spontaneous or induced). Depending on the VT, maintaining hemodynamic stability may require significant hemodynamic support. Entrainment mapping specifically refers to a mapping technique that allows a more detailed evaluation of the reentry circuit(s) and its components. Entrainment mapping requires pacing from various sites within the heart at a cycle length shorter (faster rate) than the clinical VT and typically is used to identify reentrant circuits. Hemodynamically unstable VT frequently is considered a contraindication for activation and entrainment mapping. Alternatively, mapping and ablation can be performed in normal sinus rhythm. This is usually accomplished using a pace mapping and substrate mapping approach. In pace mapping, specific myocardial sites within and along the scar border are paced, allowing for analysis of the QRS morphology and activation sequence that are similar to that of the clinical VT. Substrate mapping approach identifies regions of low-voltage signals or late potentials suitable for ablation. Substrate mapping in sinus rhythm frequently is combined with limited activation and entrainment mapping for short periods only to minimize detrimental hemodynamic effects. Epicardial mapping may be added if the endocardial approach is not successful.

**Ablation**

After suitable ablation targets have been identified by the above described mapping techniques, ablation of arrhythmogenic tissue is performed. Focal ablation with limited energy may be all that is required for outflow tract VTs, whereas scar-related VT typically requires more extensive

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Figure 3. Representation of a ventricular tachycardia re-entry circuit. A myocardial scar serves as a fixed anatomic border around which electrical signals can propagate. The regions between the scar are areas of abnormal myocardial tissue (border zone), which displays properties of slow conduction. A ventricular tachycardia (VT) circuit is set up by slow electrical conduction through the region of abnormal myocardial tissue (curved line) and around the scar in normal myocardium and subsequently returns at the entrance point (tip of arrows) to perpetuate VT. The location of the VT exit site from the scar (asterisk) in the left ventricle determines what the electrocardiogram (ECG) morphology of the VT will look like. Therefore, the ECG during VT can help guide ablation by elucidating the location of myocardial scar and the VT exit site. The black square represents the area of excitable gap where myocardial tissue is electrically excitable and not refractory. This excitable gap is required for the maintenance of a reentrant VT circuit.

Figure 4. Activation map of the left ventricular endocardium. An activation map of the left ventricular endocardium during ventricular tachycardia (VT) is shown in panel A. The color scale shows earliest activation in red and late activation in purple. In the region of earliest activation (red arrow), the mapping catheter demonstrated electrograms preceding the onset of the QRS (white arrow). This site was the area of earliest activation seen and was targeted for ablation during VT (red dots). Interestingly, at this site there was no endocardial scar, suggesting that the VT circuit isthmus is intramyocardial or epicardial. During ablation at this site, the VT terminated in <6 seconds of radiofrequency ablation (end of black arrow in panel B).
mapping and tissue lesions. Modern radiofrequency (RF) ablation catheters have steerable, saline-cooled catheter tips, allowing for precise lesions.52 It is important to recognize that these catheters are irrigated continuously at a rate of approximately 2 mL/min and during ablation up to 30 mL/min. During a typical ablation procedure lasting for several hours, this adds a significant fluid load to the patient, further increasing the risk for acute exacerbation of heart failure. At this point, RF alternating current is the preferred energy source. Cryoablation catheters are used frequently during surgical ablations but only play a minor role during the catheter-based approach.53 Rarely, chemical (alcohol) myocardial ablation is performed via branches of coronary arteries after other ablation techniques have failed.54

ANESTHETIC CONSIDERATIONS

Anesthetizing Location
Catheter ablation procedures typically are performed in a non-operating room (OR) setting. If not configured adequately, these remote anesthesia locations may pose problems in case of emergency situations with limited access to supplies and support structures.55 Certain ablation procedures and particularly those that require surgical access may best be performed in a hybrid OR, with the ability to supplies and support structures.55 Certain ablation procedures and particularly those that require surgical access may best be performed in a hybrid OR, with the ability to integrate advanced imaging and other technologies required for ablation procedures into an OR environment.

Anesthesia Provider Model
Currently, there are no specific guidelines by any specialty society regarding the anesthesia coverage required for VT ablations. In an international consensus document on VT/VF ablation,7 the electrophysiologist’s task force members merely recommend the presence of personnel familiar with monitoring patient’s vital hemodynamic variables throughout the procedure. Given the highly complex hemodynamic changes seen during VT ablations necessitating pharmacological and even mechanical support, few institutions will perform complex VT ablations without professional anesthesia coverage. In fact, at many academic centers, it is the cardiac anesthesiologists who either personally administer or supervise anesthesia care for complex VT ablation procedures.

Preablation Assessment
Patients scheduled for VT ablation procedures present with a wide range of comorbidities. The patient without structural heart disease, full exercise capacity, and a benign type of VT will rarely need a preablation anesthesia assessment beyond what has been recommended as the standard preanesthesia evaluation by the American Society of Anesthesiologists.56 On the other extreme, however, there are patients who have severely compromised cardiac function, frequent episodes of unstable VT, or even uncontrollable VT storm requiring emergent intervention. Although institutional practices may differ, the International Consensus Document on VT/VF ablation7 recommends the following assessments before VT ablation: type and burden of VT, VT morphology, and VT cycle length. This information typically is obtained from a 12-lead electrocardiogram and Holter electrocardiogram monitoring. In patients with an ICD, critical information about the clinically relevant VT often can be obtained from the ICD’s automated storage of a VT event. In addition, the task force recommends evaluating the patient for etiology and extent of myocardial disease. This includes assessing the patient for the presence and extent of obstructive coronary artery disease, which typically requires a coronary angiography performed before the ablation procedure. If a cardiomyopathy is suspected, an endomyocardial biopsy may be warranted. A transthoracic echocardiogram and a computed tomography may be required for specific VT ablation procedures. Cardiac magnetic resonance imaging with late enhancement can be particularly useful to delineate scar tissue. Additional recommendations include evaluating the patient for significant peripheral vascular disease, which is important for vascular access planning.

Type of Anesthesia and Anesthesia Depth
The level of consciousness and related impact on the autonomic nervous system can have a profound effect on arrhythmias.57,58 Changing the conscious state and even light sedation may suppress some VTs and, thus, interfere with the ablation procedure. Therefore, the electrophysiologists often request the patient to be awake or to use the least amount of sedation needed for patient comfort when reproducing a clinical arrhythmia in the EP laboratory. An expert consensus on VT ablation developed by the European
Heart Rhythm Association and the North American Heart Rhythm Society cautions to the potential arrhythmia suppressing effects of general anesthesia (GA) while acknowledging the fact that GA may be required in certain patient populations. The panel recommends avoiding GA and deeper levels of sedation in patients with catecholamine-sensitive VTs, and in cases in which previous attempts to induce VT were unsuccessful.6 Adapting the depth of sedation or anesthesia to the various parts of the procedure is encouraged (e.g., mapping for arrhythmogenic substrate may be facilitated by lighter anesthesia). It is also recommended to avoid neuromuscular blocking drugs during anesthesia maintenance when phrenic nerve monitoring is warranted. In general, outflow tract VT ablation is typically accomplished with MAC or light sedation. Complicated scar-related VT ablation procedures can easily take 6 to 8 hours and usually require GA.

**Anesthetic Drug Interaction**

In general, only very limited and low-level evidence data are available on the effect of specific drugs and anesthetic drugs on the inducibility of VT and VT-relevant variables. The majority of the data available are from ablation treatment of supraventricular arrhythmias (SVTs) related to aberrant conduction pathways. Unlike SVTs, however, anesthetic drug effects related to VTs cannot be simply described as a delay or acceleration of electrical signals along established anatomic conduction pathways. Arrhythmogenicity of anesthetic drugs traditionally has been evaluated regarding their effect on action potential duration, specifically the QT interval and other measures of depolarization and repolarization.59 Prolongation of cardiac repolarization has been linked to malignant VTs such as torsades de pointes. Other measures of arrhythmogenic drug properties include direct effects on ion channels, gap junctions, as well as modulating properties, for example, on the autonomic nervous system. Because it is unlikely that anesthetic agents will alter the arrhythmogenic substrate itself (e.g., scar tissue), interference of anesthetic agents with an EP study, and in particular the inducibility of VT, can probably be attributed to modulating factors.

Propofol frequently is administered for sedation or GA during EP studies and ablation procedures apparently with minimal effects on the conduction system.50-52 Compared with volatile anesthetics, propofol does not seem to prolong the action potential duration and QT interval.65-66 Antiarrhythmic properties have been attributed to ion channel inhibition, protective effects on myocardial gap junctions, and suppression of the autonomic nervous system.67-71 In a recent study in patients undergoing SVT ablation, sedation with propofol or ketamine had no effect on the inducibility of the initial arrhythmia.72 However, in patients with clinical VT or VT storm unresponsive to antiarrhythmia treatment, propofol frequently is used to blunt the sympathetic output and help terminate or control the VT.73-74 Etomidate frequently is used for inducing GA in patients with severely compromised cardiac function. Aside from a single case report in which etomidate was used to suppress VT in a patient with VT storm,75 no data on interference with VT ablation procedures are available. Benzodiazepines are used in the EP laboratory for anxiolysis in the preprocedure setting, for sedation, or as part of balanced GA. Effects on the QT interval seem to be minimal.76 The few studies reporting on the use of benzodiazepines in the EP setting showed no clinically relevant effect on EP studies.77-78 Volatile anesthetics commonly are used to maintain GA in the EP laboratory. Although both proarrhythmic and antiarrhythmic effects have been described,79-80 most modern inhaled anesthetic agents seem to suppress ventricular arrhythmias. Sevoflurane, isoflurane, and desflurane have been shown to prolong the action potential duration80 and QT interval.81-83 In animal models, isoflurane reduced the incidence of ischemia, as well as reperfusion-induced arrhythmias,83 and both desflurane and sevoflurane inhibited postinfarction ventricular arrhythmias.84 A recent case report of desflurane use during a VT ablation procedure reproducibly suppressed the arrhythmia.85 Desflurane’s sympathomimetic properties at greater doses and rapidly increasing concentrations, however,86 have led to postulate arrhythmogenic effects in isolated cases.87 Overall, the abundant use of volatile anesthetics for GA during VT ablation procedures in clinical practice seems to imply minimal interference in the majority of cases. In cases an arrhythmia is no longer inducible, modification of the anesthetic and a trial without the use of volatile anesthetic agents can be useful. Opioids are part of many anesthetic regimens. Kappa-opioid receptor-mediated arrhythmogenic and antiarrhythmic action, as well as direct effects on the ion channels independent of opioid receptors, has been discussed.88 Early studies on the arrhythmogenic effects of morphine and fentanyl showed an increased threshold for ventricular arrhythmias in animal models.89-91 Fentanyl and remifentanil seem to minimally affect the QT interval, whereas greater doses of sufentanil may cause QT prolongation.92 A centrally mediated increase in vagal activity causing bradycardia and effects on the autonomic nervous system could theoretically interfere with EP studies, but this does not seem to be of a major concern in the clinical setting.77,93,94 Dexmedetomidine, an α2 adrenergic receptor agonist, has pronounced and clinically relevant effects on the autonomic nervous system and antiarrhythmic properties. Dexmedetomidine reduces the sympathetic tone and heart rate variability.95 The most quoted side effects are bradycardia and hypotension.96 The antiarrhythmogenic properties are thought to be related to central α2A-adrenoceptor-mediated enhanced vagal activity97,98 and sympatholytic effects attributable to presynaptic activation of α2A-adrenoceptors resulting in decreased norepinephrine release. The electrophysiologic properties99,100 and in particular antiarrhythmogenic effects of dexmedetomidine,101 have been described. In a small study in children undergoing heart surgery, patients with perioperative dexmedetomidine administration experienced fewer episodes of VT and VTs.102 Sustained VT in a child with dilated cardiomyopathy was converted successfully to normal sinus rhythm with dexmedetomidine.103 Dexmedetomidine administered for sedation after cardiac surgery resulted in fewer episodes of VT compared with a propofol-based sedation regimen (0% vs 5%).104 In patients with catecholamine-sensitive VTs, the pronounced decrease in epinephrine and norepinephrine plasma levels seen even with low-dose dexmedetomidine infusion105 may be particularly detrimental to VT.
mapping in the EP setting. It seems reasonable to conclude that although there may actually be a role for dexmedetomidine in treating patients with tachyarrhythmias,106 the same properties may be unwanted and counterproductive during VT ablation procedures.

**Monitoring**

The type of monitoring used in patients undergoing VT ablation largely depends on the VT etiology, underlying cardiac dysfunction, as well as planned ablation strategy (i.e., mapping in VT or sinus rhythm). Although large sudden fluid shifts are rare (e.g., catheter-related cardiac perforation), the net fluid gain over prolonged ablation procedures can be significant. Depending on the VT cycle length (i.e., rate) and the underlying cardiac function, large swings in arterial blood pressure and prolonged periods of low cardiac output are frequently seen. Pulse pressure typically is reduced during fast VT, and the mean perfusion pressure significantly decreases. Pulse oximetry readings are often not obtainable, and noninvasive arterial blood pressure monitoring becomes unreliable if possible at all.

With the exception of patients with normal cardiac function and expected short procedure times (e.g., outflow tract VTs), invasive arterial blood pressure should be monitored continuously in patients undergoing VT ablations. If not required for anesthesia induction, arterial access often can be shared with the electrophysiologist, who usually obtains femoral arterial access for the ablation procedure. Similarly, central venous access can be gained from the femoral venous access site.

For more complex ablation procedures and particularly in patients with severely compromised cardiac function, additional monitoring includes means to assess for acute right and left heart failure and adequate end-organ perfusion. Cerebral oximetry has been proposed to provide valuable information in this setting.107,108 Not dependent on pulsatile flow (i.e., during fast VT), obtained values are a noninvasive estimate of oxygen supply and demand ratio.109 Monitoring for decreasing values during VT compared with baseline values recorded during hemodynamically stable sinus rhythm (trend monitoring), as well as a lower limit (absolute threshold), can be used to help guide hemodynamic management.110–112 Maintaining brain saturations at or close to baseline can be reassuring during prolonged periods of VT, whereas significant desaturations may warrant termination of simulated or induced VT.107 The combination of cerebral oximetry and continuous arterial blood pressure monitoring has been used to estimate individual lower limits of autoregulation during cardiopulmonary bypass.113,114 Although this concept has not been evaluated in the VT ablation setting, the principle behind it is appealing because it would allow for a more accurate estimation of the required perfusion pressure during prolonged periods of VT. A pulmonary artery catheter rarely is needed, and technical limitations specific to the VT setting further limit its use. For example, heat generation during RF ablation can interfere with thermodilution cardiac output measurement accuracy. In addition, continuous cardiac output pulmonary artery catheters calculate cardiac output with a significant temporal delay not representative of the acute changes seen with onset and termination of VT during ablation procedures. Less-invasive methods of measuring cardiac output typically are not reliable in patients with arrhythmias.115,116 Monitoring for adequate anesthesia depth and level of neuromuscular block can be useful during VT ablation. The electrophysiologist may ask for GA without neuromuscular blockade during certain parts of the ablation procedure to monitor phrenic nerve function.117 The latter is at risk for being injured during VT ablation procedures due to the nerve’s close proximity to the pericardium. Monitoring for myocardial ischemia, especially during epicardial ablation, is important, given the potential for direct coronary injury from epicardial access or manipulating the catheter in the epicardial space, as well as the epicardial surface ablation.

Intracardiac echocardiography frequently is used by the electrophysiologist in complex ablation procedures. Alternatively, transesophageal echocardiography can be used for hemodynamic monitoring and to detect possible complications from ablation procedures such as pericardial effusion or cardiac tamponade from catheter perforation, or fluid collection from epicardial catheter cooling.

**Postprocedure Patient Care**

Prolonged complex ablation procedures in patients with structural heart disease often are associated with significant volume expansion, electrolyte disturbances, lactate accumulation, and acute exacerbation of heart failure. Measuring the left atrial pressure (LAP) at the end of the procedure can be useful in assessing the degree of cardiac decompensation and guiding postprocedure management. If the LAP is significantly elevated, administering diuretics and possibly extending pharmacological or mechanical circulatory support with assisted ventilation in the postprocedure period may be required. In uncomplicated VT ablations, however, patients are typically tracheally extubated at the end of the procedure.

**Pharmacologic Support**

When choosing a particular inotropic or vasoactive drug, the time point during the procedure has to be considered. After induction of anesthesia and during the mapping and ablation procedure, vasoactive drugs such as phenylephrine and norepinephrine are usually titrated to maintain an adequate perfusion pressure. Drugs with significant chronotropic properties such as dobutamine or epinephrine may interfere with VT mapping and, thus, may not be warranted...
during that part of the procedure. At the end of the procedure, however, particularly in patients with dilated cardiomyopathy, poor cardiac function, and increased LAP, inotropic drugs may be required and even continued in the intensive care unit.

Mechanical Support
The temporary use of mechanical support has been suggested to facilitate ablation strategies that involve extensive mapping and ablation during hemodynamically unstable VT and to prevent acute decompensation of heart failure during prolonged ablation procedures. Various types of mechanical support have been used and are associated with unique advantages and limitations in the VT setting. The intraaortic balloon pump, for example, is of limited value during periods of accelerated heart rates typically encountered during VT ablation procedures. Percutaneously inserted left ventricular assist devices, such as the Impella (Abiomed Inc., Danvers, MA),107,123 the Tandem Heart (Cardiac Assist Inc., Pittsburgh, PA),118,124 and even short-term extracorporeal membrane oxygenation,125-127 have yielded promising results in the VT ablation setting. In general, patients with poor ventricular function, preexisting heart failure, fast VT, chronic kidney disease, and expected prolonged periods of VT during the ablation procedure will benefit the most from mechanical support.119 In a prospective observational study in patients undergoing scar-related VT ablations, fewer and less significant cerebral desaturations and more stable hemodynamics were seen with left ventricular assist device support.120 Although large, prospective randomized studies would be required to prove superior results and outcomes, clinical experience has shown superior hemodynamic stability and less exacerbation of acute heart failure symptoms in patients receiving mechanical support during VT ablation procedures.

Outcome
Outcome reports are highly dependent on the underlying type of VT and whether a substrate can be clearly identified. The primary immediate outcome during the VT ablation is termination of the clinical VT or noninducibility of VT. Long-term outcome is typically reported as recurrence rate, freedom from ICD therapy, hospital readmission rates for VT, event-free survival rates, decrease in VT events, and varies significantly.119,120 In a multicenter observational study in patients with monomorphic VT after myocardial ischemia, immediate ablation success was accomplished in almost half of all patients (49%).132 After a 6-month follow-up, 53% of patients who survived were free of VT. Despite this relative success of catheter VT ablation, the 1-year mortality rate was 18%, and 72.5% of deaths were due to VT or heart failure. In a multicenter prospective VT trial in patients with scar-related VT, immediate ablation success was 81%; however, during the 1-year follow-up 37% of the successfully ablated patients developed new VT or VT recurrence.133 In a most recently published prospective study comparing outcome in patients with nonischemic dilated cardiomyopathy and ischemic cardiomyopathy, the immediate success rate was similar (66.7% vs 77.4%, respectively), but the long-term outcomes in nonischemic dilated cardiomyopathy were significantly worse.134 In patients with arrhythmogenic right ventricular dysplasia/cardio-myopathy, a combined endocardial and epicardial ablation strategy resulted in freedom from ICD therapy during 3-year follow-up period in almost 85% of patients, and 70% of patients did not require antiarrhythmic drugs.135

Complications
Periprocedural complications have been reported in a significant number of patients undergoing VT ablation.130 Complications are mostly related to vascular access, manipulation of the catheter for mapping and the ablation itself, and exacerbation of underlying heart failure or renal insufficiency. One review found that major complications occurred in 6% of catheter ablation procedures, including cardiac perforation, tamponade, thromboembolic events including stroke, and peripheral vascular access complications.136 Access-related injuries include local and more extensive (e.g., retroperitoneal) hematomas, aortic dissection, fistula, and pseudoaneurysm formation.137 Thromboembolic events including strokes, although rare, are more frequently seen in VT ablations compared with SVT ablation procedures.138 Small pericardial effusions are not uncommon but rarely require immediate surgical intervention. Periprocedural mortality is relatively low, typically <5% in large series.130,139,140 Heart failure is a common cause of short- and long-term mortality, particularly in patients with structural heart disease. Other complications include damage to the heart valves and conduction system.

Epicardial VT ablation is associated with inherent risks. Major periprocedural complications have been reported in 4% to 9% of epicardial VT ablations, including acute pericardial bleeding, coronary injury, acute ischemia from coronary vasospasm, and access-related abdominal hemorrhage and organ puncture.142-147 The left phrenic nerve is particularly at risk of being injured during epicardial ablation and prompts routine stimulation and monitoring during epicardial VT ablation procedures.117,148

Management of Patients in VT Storm
Patients presenting with VT storm frequently are hemodynamically unstable and require emergent intervention. Treatment usually includes antiarrhythmic pharmacotherapy and hemodynamic stabilization. The vicious cycle of reentry also is sustained, in part, by sympathetic activation of the autonomic nervous system. Attempts to break this vicious cycle by reducing the autonomic nervous system’s sympathetic output often include deep sedation even requiring mechanical ventilator support. The profound sympatholytic properties of dexmedetomidine could potentially be beneficial for sedation in these patients; however, this has not been formally investigated at this point. In addition, selective sympathetic blockade with high thoracic epidural,150,151 or stellate ganglion blockade,152,153 has shown to successfully terminate VT in selective cases. Bourke et al.154 reported their experience with sympathectomy in 14 patients, 12 in VT storm and 2 with recurrent VT unresponsive to medical or ablation therapy. In 8 patients, an epidural catheter was placed at the T1-2 or T2-3 epidural interspace and further advanced 5 cm in the epidural space.
One milliliter of 0.25% bupivacaine was injected followed by a continuous infusion at 2 to 3 mL/h titrated to effect. The antiarrhythmic effect was observed immediately, and 6 patients had a decrease in VT burden by >80%. When considering neuraxial manipulation in patients presenting in VT storm, particular care must be taken to follow established guidelines for regional, as well as neuraxial, blockade in patients who may be receiving anticoagulation regimens due to their underlying heart disease.

Patients who responded to sympathetic blockade subsequently may undergo surgical sympathetic denervation of the heart. Even renal sympathetic denervation via catheter ablation has been described for treatment of electrical storm,

but this is an evolving field and randomized studies have not been performed at this point.

CONCLUSIONS

In summary, patients undergoing VT ablations comprise a wide range of underlying etiologies and severity of cardiac compromise. The appropriate anesthetic needs must be tailored to the individual patient and planned ablation procedure. The anesthesiologist plays a vital role in maintaining hemodynamic stability and monitoring end-organ perfusion during these procedures.

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