Review

Anesthetic Management

in Radiofrequency Catheter Ablation of Ventricular Tachycardia

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Radiofrequency catheter ablation is increasingly being used to treat patients who have ventricular tachycardia, and anesthesiologists frequently manage their perioperative care. This narrative review is intended to familiarize anesthesiologists with preprocedural, intraprocedural, and postprocedural implications of this ablation.

Ventricular tachycardia typically arises from structural heart disease, most often from scar tissue after myocardial infarction. Many patients thus affected will benefit from radiofrequency catheter ablation in the electrophysiology laboratory to ablate the foci of arrhythmogenesis. The pathophysiology of ventricular tachycardia is complex, as are the technical aspects of mapping and ablating these arrhythmias. Patients often have substantial comorbidities and tenuous hemodynamic status, necessitating pharmacologic and mechanical cardiopulmonary support. General anesthesia and monitored anesthesia care, when used for sedation during ablation, can lead to drug interactions and side effects in the presence of ventricular tachycardia, so anesthesiologists should also be aware of potential perioperative complications. We discuss variables that can help anesthesiologists safely guide patients through the challenges of radiofrequency catheter ablation of ventricular tachycardia. ***(Tex Heart Inst J 2016;43(6):496-502)***

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Ventricular tachycardia (VT) is often associated with severe heart disease. The progression of VT degenerates to ventricular fibrillation and accounts for 80% of sudden cardiac arrests.[1] Implantable cardioverter-defibrillators (ICDs), with or without adjunct antiarrhythmic-drug therapy, are the first-line treatment for most VT patients who have structural heart disease. Advances in technology have enabled radiofrequency catheter ablation (RFCA) to improve results—both as a sole treatment for VT without structural heart disease and in combination with an ICD or antiarrhythmic therapy for scar-related VT associated with structural heart disease.[2] Because anesthesiologists are frequently required during RFCA in patients with VT, this review discusses concerns related to anesthetic management throughout the procedure.

Pathophysiology of Ventricular Tachycardia

Ventricular tachycardia, part of a heterogeneous group of wide-complex tachyarrhythmias, can be morphologically classified either as monomorphic VT (all beats look identical or nearly identical in each lead of a surface electrocardiogram [ECG]) or as polymorphic VT (beat-to-beat variation in each lead of a surface ECG). In monomorphic VT, 70% of cases are caused by ischemic heart disease and 20% by other structural disease (including dilated nonischemic cardiomyopathy, valvular or congenital heart disease, sarcoidosis, and Chagas disease); 10% are idiopathic.[3] Most VT is due to a reentrant circuit at the border of a myocardial scar and is usually monomorphic, although acute myocardial infarction can sometimes also cause polymorphic VT.[4,5] This contrasts with idiopathic VT, which usually originates from the right ventricular (RV) or left ventricular (LV) outflow tract and typically presents in adolescents.[6] Genetic disorders such as long QT or Brugada syndrome can predispose patients to polymorphic VT.[7] Table I shows causes of arrhythmias.

Treatment of Ventricular Tachycardia

Currently, there are 4 options to treat VT: antiarrhythmic medications (β-blockers, calcium channel blockers, and class I or III antiarrhythmic agents), ICD support, catheter ablation, and surgery. Selection of the appropriate therapy to manage the various arrhythmias (premature ventricular contractions [PVC], nonsustained VT, sustained monomorphic or polymorphic VT, and ventricular flutter or fibrillation)

is guided by the cause and mechanism of the arrhythmia and the risk associated with each relevant therapy. Many patients benefit from combined treatments.[8] Patients undergo ICD implantation chiefly on the basis of LV ejection fraction (LVEF), the cause of LV dysfunction, and New York Heart Association (NYHA) functional class (Table II).[9]

Radiofrequency Catheter Ablation

Clinically and technologically, RFCA has improved over the last 20 years, and indications for its use have greatly increased. Currently, RFCA is widely used to manage VT associated with structural heart disease when ICDs or antiarrhythmic drugs have failed, and it is usually the sole treatment for idiopathic VT.[2] As RFCA outcomes improve, catheter ablation is being used earlier in VT treatment.

The approach to catheter-based ablation can be endocardial, epicardial, or both, depending on the likely origin of the arrhythmia. In most circumstances involving ischemic heart disease, endocardial ablation is sufficient. For this procedure, catheters are usually inserted into the heart chambers through sheaths placed in the femoral veins or arteries. The RV can be reached by advancing the catheters antegrade from the femoral vein, and the LV can be reached via an antegrade transseptal or retrograde transaortic approach (Fig. 1). The cath-

TABLE I. Causes of Different Ventricular Arrhythmias

Type	Cause
Premature ventricular contractions	Ischemia
	Cardiomyopathy
	Idiopathic
	Exercise-induced
Monomorphic VT	Scar-related from ischemia
	Structural heart disease Dilated cardiomyopathy Hypertrophic cardiomyopathy Valvular heart disease Sarcoidosis Chagas disease
	Idiopathic origin (nonstructural) RVOT/LVOT Fascicular Papillary muscle Epicardial
Polymorphic VT	Acute myocardial ischemia
	Idiopathic RVOT origin Purkinje Moderator band
	Inherited syndromes: Long QT Brugada Catecholaminergic

LVOT = left ventricular outflow tract; RVOT = right ventricular outflow tract; VT = ventricular tachycardia

TABLE II. Indications for ICD to Prevent Sudden Cardiac Death

Patient History	Indication for ICD
Prior MI	LVEF ≤0.30, >40 days post-MI, and NYHA functional class I
	LVEF ≤0.35, >40 days post-MI, and NYHA functional class II or III
	LVEF ≤0.40, nonsustained VT, and inducible VF or sustained VT during electrophysiologic study
Nonischemic dilated cardiomyopathy	LVEF ≤0.35 and NYHA functional class II or III
Genetic conditions*	Hypertrophic cardiomyopathy, congenital long QT syndrome, catecholaminergic polymorphic VT, Brugada syndrome

ICD = implantable cardioverter-defibrillator; LVEF = left ventricular ejection fraction; MI = myocardial infarction; NYHA = New York Heart Association; VF = ventricular fibrillation; VT = ventricular tachycardia

*An ICD is usually indicated if the patient's genetic condition is associated with one or more risk factors.

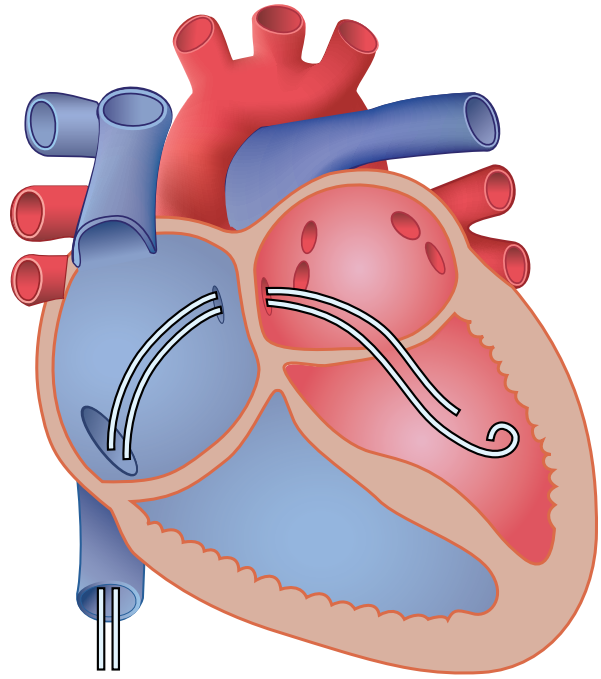

Fig. 1 *Drawing shows endocardial placement of radiofrequency ablation catheters from the antegrade transseptal approach (catheters have been inserted through the femoral vein and guided across the atrial septum into the left ventricle).*

eters are then used to map the chamber and to ablate the appropriate region.[10]

Radiofrequency catheter ablation is more challenging in nonischemic cardiomyopathies when circuits are intramural or epicardial in origin. This is also true in other cases, such as arrhythmogenic RV cardiomyopathy, where up to 40% of VTs are epicardial in origin.[11] In these circumstances, the epicardial approach can be useful. To begin, a small mark is made 2 to 3 cm below the xiphoid process. After local anesthesia is given, a 17G Tuohy epidural needle is inserted from the mark toward the pericardial space, under fluoroscopic guidance. When the needle has reached the pericardial space (confirmed by fluoroscopy and lost resistance), a guidewire is inserted through the needle, and an 8F or 9F sheath is placed over the guidewire with use of the standard Seldinger technique. After sheath position is confirmed by fluoroscopy, a catheter is inserted for mapping and ablating the cardiac surface.

Identifying the VT's precise location is crucial. This can be done by activation mapping, pace mapping, entrainment mapping, substrate mapping, or combinations of these. Activation and pace mapping are typically used for focal mechanisms; substrate, substrate + pace, and entrainment mapping are more suitable for reentrant mechanisms. In stable VT, activation and entrainment mapping during VT are used to find the crucial circuit that will guide ablation. For multiple and unstable VTs, substrate + pace mapping can reveal scarred regions and potential reentry-circuit channels during sinus or paced rhythm.

In activation mapping, the electrical activation sequence is recorded during VT, and the earliest site of activation is marked as the origin of tachyarrhythmia.[12] Pace mapping analyzes the activation sequence while pacing in the absence of VT. The point where the paced QRS morphology is similar to the VT marks the origin of a focal VT or the exit of a scar-related reentry.[13,14] Entrainment mapping, which is attained by pacing at rates faster than the VT, is useful for finding reentry circuit sites and bystander sites that are adjacent to the reentry circuit isthmus. Entrainment is the continuous resetting of a reentrant circuit by a pacing train. The QRS morphology and electrogram timing during entrainment show whether the pacing site is in the reentry circuit isthmus or at bystander sites.[15] Substrate mapping uses an electroanatomic mapping system to find an area of abnormal myocardial conduction on the basis of anatomic and electrographic characteristics (usually voltage) during stable sinus or paced rhythm. Low-voltage areas are the scarred regions. Substrate mapping can be especially helpful in the ablation of multiple, pleomorphic, and unmappable VTs (hemodynamically unstable or not inducible).[16] Electroanatomic mapping of the heart is safe and effective in arrhythmic patients: it shows intracardiac electrical activation in connection with an anatomic position in the cardiac chambers. It is widely used in mapping idiopathic VT and VT associated with structural heart disease.[2]

The best catheter ablation strategy is still undetermined, and there has been no randomized trial to compare ablation during VT with different substrate-based ablation approaches, such as linear ablation, short lines, targeting of conductive channels, or encircling technique.[13,14,17] When patients have inducible VT at the beginning of ablation, the procedural endpoint is noninducibility of all clinical VTs during postablation induction with programmed electrical stimulation or catecholamines.[2,17] In patients with incessant VT, restoration of stable sinus rhythm is a reasonable clinical endpoint, regardless of the outcome of postprocedural programmed electrical stimulation. In patients with noninducible VT, the ablative endpoint can be defined as the absence of all channels inside the targeted region, or as ablation with linear lines on the basis of pace mapping at designated sites along the infarct scar.[2]

The success rate of VT ablation varies, depending upon the mechanism of the VT. In patients with scar-related VTs, 70% will have a reduced or abolished burden after ablation, whereas up to 90% of idiopathic VTs will be abolished.[18]

Preprocedural Considerations for Anesthesia

The anesthesiologist's preparation starts with reviewing the patient's medical history and physical examination results (Table III). Special attention should be given to recent medication use, because some anticoagulants and antiarrhythmic agents can cause QTc prolongation. Patients are often taking diuretic agents and digoxin, so electrolyte values should be obtained to exclude major abnormalities in sodium and potassium levels.

Most patients undergoing ablation will be in sinus rhythm. If they are in clinical VT, the electrophysiologist can compare a current ECG with VTs induced during the procedure, to see if they are from the same probable origin. If the patient already has an ICD, stored and real-time intracardiac electrograms can be obtained during device interrogation to compare with induced VTs during the procedure. Recent echocardiograms can rule out intracardiac thrombus and reveal ventricular and valvular function. Preprocedural magnetic resonance imaging (MRI) can help to characterize myocardial scarring. (In the past, MRI was contraindicated in patients who had ICDs; however, the U.S. Food and Drug Administration has approved the first ICD device that is compatible with full-body MRI scanners.) The images can then be merged with the electroanatomic mapping system during substrate mapping intraprocedurally. If acute ischemia is suspected, angiography can be performed to rule out reversible VT causes.

Patients initially presenting with arrhythmia can have widely varying symptoms, depending on their heart rate

TABLE III. Preoperative Evaluation for VT Ablation

Variable	Details
History	Type of VT, symptoms (chest pain, palpitations, etc.), triggers, past ablation attempts, history of MI, CHF, and valvular disease
Physical examination	Signs of decompensated heart failure, murmur, gallops, general physical state, sternotomy scar, vascular access, and presence of support devices such as IABP or pVAD
Medications	Current antiarrhythmic agents, diuretic agents, heart failure medications, digoxin, anticoagulants, and infusions of vasopressors or inotropic agents
	QTc-prolonging medication: antipsychotic agents, antibiotics (macrolides, fluoroquinolones), antiemetics, antiarrhythmic agents, and antidepressants
Vital signs	Hemodynamic stability
Laboratory tests	Electrolytes, creatinine, troponin, TSH, toxicology assay
Electrocardiogram	Determine baseline rhythm; if in clinical VT, determine rate and morphology of QRS complexes of VT.
Imaging	Chest radiography: evaluate cardiomegaly and pulmonary edema or effusion. Echocardiography: evaluate ventricular function, valvular disease, and presence of thrombus. CT/MRI: identify location of myocardial scarring and evaluate cardiomyopathies.

CHF = congestive heart failure; CT = computed tomography; IABP = intra-aortic balloon pump; MI = myocardial infarction; MRI = magnetic resonance imaging; pVAD = percutaneous ventricular assist device; TSH = thyroid-stimulating hormone; VT = ventricular tachycardia

and degree of underlying cardiomyopathy. Slow VT might cause palpitations, syncope, or angina, whereas fast, sustained VT can lead to hemodynamic instability and cardiovascular collapse. Hemodynamically unstable patients might be getting continuous infusions of vasopressors and inotropic fluids along with amiodarone, lidocaine, or procainamide to suppress arrhythmia. In patients with VT storm (>3 VT episodes within 24 hr), sedation and insertion of an intra-aortic balloon pump (IABP) are often necessary to decrease adrenergic stimulation and improve hemodynamic stability.

Types of Anesthesia

Radiofrequency catheter ablation has been successful with patients under monitored anesthesia care (MAC) and general anesthesia (GA). Advantages of MAC with sedation include avoiding anesthetics that can depress hemodynamic stability and decrease the inducibility of VT.[19] Much ablation for ventricular arrhythmia targets symptomatic focal PVCs rather than sustained VT, so the inducibility of arrhythmia is important. Volatile anesthetics such as sevoflurane and isoflurane can prolong action-potential duration, delay atrial and ventricular repolarization, and decrease the inducibility of tachyarrhythmias in vitro; however, the clinical significance of this is unknown.[20,21] Fentanyl, often used in GA and MAC, increases vagal tone and prolongs sinus node recovery.[22] Certain types of VT, such as outflow-tract VT, are extremely sensitive to sedation, so minimal usage of anesthetic drugs is optimal. If target PVCs are seen, they can be added to the recording system as templates before anesthetic drugs are administered, and this information can be used later for pace mapping if ectopy is suppressed by sedation. In MAC, midazolam and short-acting opioids have typically been used for sedation. Mandel and colleagues[23] reported the successful use of a remifentanil infusion with intermittent midazolam boli for epicardial VT ablation in a case of arrhythmogenic RV cardiomyopathy.

General anesthesia is typically preferred when prolonged and complex procedures are performed in patients who have unstable rhythms or tenuous cardiorespiratory reserve, or when difficult airways are anticipated. In addition, if the transseptal approach is chosen, GA will enable control of the patient's ventilation and might decrease the risk of air embolism during septal puncture or sheath exchange. We routinely use MAC with minimal sedation for ablations involving stable VTs or VTs that are suppressed by volatile anesthetics, and we use GA for more complex or unstable-VT ablations. When recurrent VT is refractory to medication and ablation, high thoracic epidural anesthesia has been shown to decrease the arrhythmia burden.[24]

Intraprocedural Considerations

Ablation is performed with standard American Society of Anesthesiology monitors in place.[25] Arterial line placement is generally indicated for hemodynamic monitoring, especially if VT induction is planned. Central venous access might be necessary in patients who have moderate-to-severe cardiomyopathy, substantial ventricular dysfunction, or valvular abnormalities. Defibrillator pads should be properly positioned in advance. The patient's temperature should be closely monitored

for intraprocedural hypothermia. In patients under GA, an esophageal temperature probe can reveal increases in temperature and minimize the risk of esophageal injury.[26]

After the intra-arterial and transseptal sheaths are inserted, anticoagulation is necessary to prevent thrombus formation (the reported prevalence of stroke during catheter ablation is 0.4% to 1%).[27] Typically, a 100-U/kg bolus of heparin is given before infusion, to target an activated clotting time between 300 and 400 s.[28] The heparin infusion can be discontinued after the catheters are removed from the LV (retrograde transaortic approach) and left atrium (antegrade transseptal approach). Depending on the most recent activated clotting time, reversal with protamine may be performed. The intra-arterial sheaths are typically kept in place for some time after the procedure.

Patients who are not in spontaneous arrhythmia in the electrophysiology (EP) laboratory undergo VT induction with the use of intravenous isoproterenol or epinephrine, sometimes with programmed stimulation or burst pacing. Many anesthetics might affect cardiac conduction and interfere with the clinical inducibility of VT. Volatile anesthetics should be avoided.[20] Propofol and etomidate have been used to suppress electrical storm, and dexmedetomidine is contraindicated in EP procedures because it can significantly depress nodal functions.[29] If the electrophysiologist cannot induce VT, options include switching anesthetic agents, using MAC, or using other mapping techniques (such as substrate mapping) for ablation. Neuromuscular blockade agents should be avoided when the planned ablation site is close to the phrenic nerve, because phrenic-nerve pacing is used to locate that nerve and avoid damage from ablation.

Use of Mechanical Support

Patients with VT often have poor cardiovascular reserve. This condition, GA, and the induction of VT for mapping can lead to hemodynamic instability and abortion of the procedure, so proactive hemodynamic management is crucial. Intraprocedural vasopressor or inotropic infusions might be necessary to maintain hemodynamic stability and prolong the procedure. No guidelines exist regarding specific agents, so the anesthesiologist and electrophysiologist may choose them. If mechanical support is desired, early IABP usage might be helpful; however, IABPs provide modest hemodynamic support at best and are often useless during rapid VT.[30]

Percutaneous ventricular assist devices (pVADs) like the TandemHeart™ (CardiacAssist, Inc.; Pittsburgh, Pa) and the Impella® 2.5 (Abiomed, Inc.; Danvers, Mass) can support hemodynamic stability, improve safety, and prolong procedural time to enable extensive mapping and ablation (Table IV and Fig. 2).[30,31] Limitations apply when using these devices in VT ablation. The TandemHeart's transseptal placement precludes the same access for ablation catheters, so only the retrograde approach from the femoral artery is feasible. The device can also dislodge, causing the cannula to slip into the

TABLE IV. Comparison of TandemHeart and Impella 2.5*

Variable	TandemHeart	Impella 2.5
Access	Retrograde through femoral vein to RA, then transseptal to LA	Retrograde through femoral artery, then across AV into LV
Pump flow	Centrifugal	Axial
Flow rate (L/min)	2.5–4	2.5
Arterial flow	Retrograde up the aorta	Anterograde down the aorta
Contraindications	Severe AR; aortic dissection; RA or LA thrombus	Mechanical AV or severely calcified AV; aortic dissection; LV or LA thrombus

AR = aortic regurgitation; AV = aortic valve; LA = left atrium; LV = left ventricle; RA = right atrium

*Both nonpulsatile-flow devices necessitate the use of anticoagulation and are approved by the U.S. Food and Drug Administration for up to 6 hours of partial circulatory support.

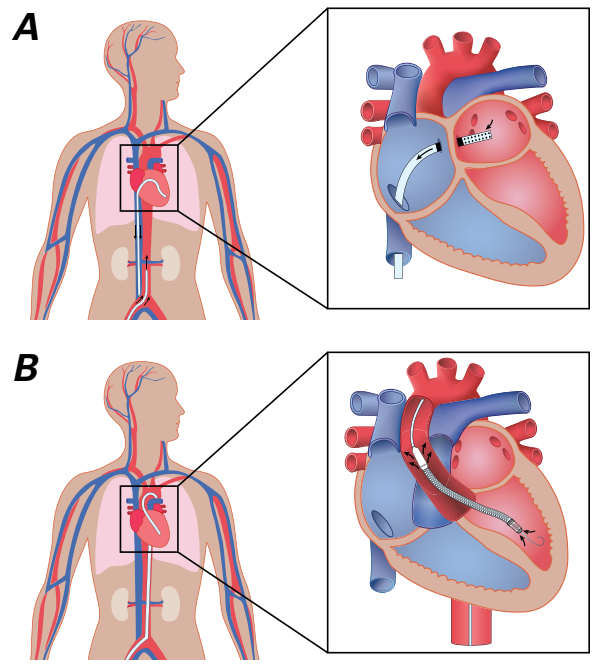

Fig. 2 ***A****) Drawing of a TandemHeart. The cannula, placed transseptally into the left atrium, withdraws oxygenated blood that is pumped retrograde into the descending aorta by an external centrifugal pump (not shown).* ***B****) Drawing of an Impella 2.5. The cannula, placed transaortically into the left ventricle, withdraws oxygenated blood and pumps it into the ascending aorta, augmenting cardiac output.*

right atrium, which can lead to cardiogenic shock and death.[32] The Impella can irritate the heart and cause ectopy, and its electromechanical interference with the mapping and ablation catheters might necessitate turning it off altogether. Finally, GA will be necessary, because transesophageal echocardiography is typically used to guide pVAD management intraoperatively. Despite these limitations, patients undergoing pVAD-assisted ablation often have more VTs mapped and ablated and have less VT recurrence than do control groups.[33,34]

Side Effects and Complications

Complications of VT ablation include a 1% to 3% risk of death and major morbidities (cardiac perforation, stroke, or myocardial infarction), a 3% risk of conduction block that necessitates a permanent pacemaker, and a 5% risk of major bleeding.[35] Potential injury includes RV free-wall perforation and damage to the aortic cusps or coronary ostia.[2,36,37] Ablation treatment fails most often because of incomplete mapping or the inability to induce arrhythmia.[6] The outflow tract anatomy in particular is complex, and the electrogram at target sites can easily be missed because of low amplitude.[18] Esophageal injuries from thermal injury and atrioesophageal fistula are rare.[38]

Several considerations apply when ablation is performed via the epicardial approach. First, to avoid injuring the left phrenic nerve, phrenic-nerve pace mapping should be performed on the anterior and lateral borders of the LV, to identify the nerve before ablation. Second, most ablation catheters have irrigated tips that continuously infuse saline solution during the procedure, so periodic aspiration is necessary to prevent excessive fluid accumulation and tamponade. Third, the risk of vascular puncture or tearing is high in patients with histories of cardiac surgery, pericarditis, or chest radiation, so percutaneous epicardial access might be contraindicated in this population. Pericardial access can be obtained through a small surgical approach, usually subxiphoid.

Postprocedural Care

After RFCA, hemodynamically stable patients are often extubated in the EP laboratory. Because of possible bleeding, the femoral sheath is often left in place for several hours, and patients must lie recumbent during that time. Patients may be monitored in the postanesthesia recovery unit or in the cardiac intensive care unit, for new arrhythmias, cardiac tamponade, and groin or retroperitoneal bleeding. They should also be observed for signs of fluid overload or heart failure: ablation catheters can infuse substantial volumes of crystalloid solution that might be unaccounted for by the anesthesiologist. If the patient was in VT storm or exhibited substantial hemodynamic derangement before or during the procedure, continued intubation and sedation might avert a catecholamine surge associated with immediate postprocedural emergence. If a pVAD was used during ablation, its removal depends on the patient's hemodynamic stability.

Summary

Radiofrequency catheter ablation has become increasingly useful and important in the treatment of patients with VT. The involvement of an anesthesiologist is often necessary during RFCA. With a thorough understanding of the techniques and potential complications of RFCA, the anesthesiologist can facilitate this life-saving procedure while ensuring patient safety.

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