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Review Article

Anesthetic Considerations in the Electrophysiology Laboratory: A Comprehensive Review

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Catheter ablation procedures for arrhythmias or implantation and/or extraction of cardiac pacemakers can present many clinical challenges. It is imperative that there is clear communication and understanding between the anesthesiologist and electrophysiologist during the perioperative period regarding the mode of ventilation, hemodynamic considerations, and various procedural complications. This article provides a comprehensive narrative review of the anesthetic techniques and considerations for catheter ablation procedures, ventilatory modes using techniques such as high-frequency jet ventilation, and strategies such as esophageal deviation and luminal temperature monitoring to decrease the risk of esophageal injury during catheter ablation. Various hemodynamic considerations, such as the intraprocedural triaging of cardiac tamponade and fluid administration during catheter ablation, also are discussed. Finally, this review briefly highlights the early research findings on pulse-field ablation, a new and evolving ablation modality.

Key Words: electrophysiology; anesthesia; ablation; ventilation; pacemakers

There are special considerations for anesthesia administration and monitoring in the electrophysiology laboratory. The cases performed in the electrophysiology laboratory are often long, complex, and associated with serious potential complications, many of which are unique to electrophysiology. Communication between the anesthesiologist and electrophysiologist during the perioperative period is fundamental regarding the specific anesthetic agent used, the mode of ventilation selected, and awareness and management of potential complications. This article will provide a narrative review of the various considerations in the electrophysiology laboratory regarding the following: (1) preoperative anesthetic assessment, (2) anesthetic techniques and modes of ventilation for various procedures, (3) potential complications of various procedures, and (4) future considerations for new techniques.

Preoperative Anesthetic Assessment

The patient's functional status should be assessed as part of the surgical optimization evaluation. Fasting state, anticipated degree of pain, projected case duration, comorbid conditions, and the patient's ability to remain supine for a prolonged period are also important factors to be considered. Patients with atrial fibrillation (AF) often have obstructive sleep apnea (OSA) and are, therefore, at increased risk for sedation-related adverse events.[1] The "STOP-BANG" questionnaire can be used to screen for OSA.[2] In addition, a decreased airway patency and an increased obstructive response to anesthetic agents can lead to difficulty in achieving effective bag mask ventilation and endotracheal intubation.

Prior to any electrophysiology procedure, including pacemaker implantation, lead extraction, or cardiac ablation, a

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transthoracic echocardiogram (TTE) can provide useful data, particularly regarding left ventricular (LV) function. This information can be very advantageous to the anesthesiologist regarding the level of monitoring required, the type of anesthetic administered, and how to manage hemodynamic instability during the procedure. For the electrophysiologist, preoperative TTE also can help determine the type of device to be implanted; for instance, an automatic implantable cardioverter-defibrillator (AICD) in a patient with an ejection fraction of <35% or the need for physiologic and/or conduction system pacing in patients with any degree of LV dysfunction in whom a significant percentage of ventricular pacing is expected (typically >40%).[3] Prior to ventricular tachycardia (VT) ablation, TTE also can be used to determine the presence or absence of an LV thrombus.

The need for routine transesophageal echocardiogram (TEE) prior to AF ablation to rule out left atrial and left atrial appendage (LAA) thrombus has been an ongoing area of interest and research, particularly in patients who have been on uninterrupted anticoagulation for at least 3 weeks. Diab et al. conducted a large (n = 900) observational study in patients with AF, and found that a preoperative TEE is not needed for patients who are on uninterrupted direct oral anticoagulants (DOACs) for at least 3 weeks prior to ablation. These patients had a low thromboembolic event rate of 0.3% postablation.[4] Patel et al. conducted a large (n = 6,186) observational study in patients on uninterrupted DOACs for at least 4 weeks prior to ablation. Intracardiac echocardiography (ICE) was used to rule out LAA and left atrial thrombi in all patients, and revealed spontaneous echocardiographic contrast in 27% of patients.[5] Only 1 patient had a transient ischemic attack, possibly due to a single missed dose of rivaroxaban prior to ablation. The low event rates in both studies are certainly promising; however, these were nonrandomized studies. The 2017 expert consensus statement on catheter and surgical ablation of AF gives a class IIa recommendation for performing a TEE in patients with AF undergoing ablation who are in AF on presentation, even if they have been receiving therapeutic anticoagulation for 3 weeks or longer.[6] The 2020 European Society of Cardiology guidelines for the diagnosis and management of AF recommend therapeutic oral anticoagulation for at least 3 weeks prior to ablation (class I) or use of TEE to exclude LAA thrombus before ablation.[7]

There are special perioperative considerations for patients who have channelopathies undergoing ACID implantation, such as Brugada syndrome and congenital long-QT syndrome (LQTS).[8] A quiet environment prior to and during implantation is needed because sympathetic stimulation, such as abrupt and loud noises, can trigger polymorphic VT (PMVT). Emotional distress and anxiety also can predispose the patient to PMVT; therefore, sufficient anxiolysis should be targeted prior to anesthesia induction. Hypothermia and hyperthermia can trigger PMVT and should be avoided in cases of patients with LQTS or Brugada syndrome, respectively. The QT interval should be monitored throughout the procedure in these patients. Electrolyte derangements—particularly hypokalemia, hypomagnesemia, and hypocalcemia—should be corrected prior to anesthetic induction, as these can prolong the QT interval, predisposing the patient to delayed repolarization leading to PMVT.

Opioid agents generally are considered safe to use in patients with channelopathies.[9] Regional anesthesia and central neuraxial blockade have been used successfully in patients with Brugada syndrome. Volatile anesthetics, depolarizing and nondepolarizing neuromuscular blockers, midazolam, and fentanyl are shown to be safe for LQTS and Brugada syndrome without any adverse effects.[9-12] Drugs that can prolong the QT interval, such as ondansetron, chlorpromazine, amiodarone, ephedrine, epinephrine, norepinephrine, dobutamine, dopamine, isoproterenol, and phenylephrine, should be avoided in the perioperative period.[9] The AICD implantation can be performed under local anesthesia with sedation during testing of the defibrillator and placement in the subpectoral pocket, thus avoiding general anesthesia (GA). The total dose of local anesthetic should be minimized, and systemic absorption limited by the use of lidocaine with epinephrine.[13] General anesthesia with induction agents, such as propofol, thiopental, and etomidate, are shown to be safe; however, ketamine should be used with caution in patients with LQTS or Brugada because it stimulates the sympathetic nervous system.

Proper management of anticoagulation prior to pacemaker and AICD surgery is important to decrease the risk of device pocket hematomas that can result in serious device infection. Patients (n = 681) undergoing pacemaker and AICD implantation in the BRUISE CONTROL-1 trial[14] were randomized to either continued interrupted anticoagulation with warfarin (n = 343) or bridging warfarin with heparin (n = 338).[14] Clinically significant device-pocket hematoma occurred more often in the heparin-bridging group (54 patients, 16%) compared to the continued-warfarin group (12 patients, 3.5%; $p < 0.001$). These results signified an 80% reduction In device pocket hematoma with continued warfarin therapy. Major surgical and thromboembolic complications were rare and did not differ between groups. Results from the BRUISE CONTROL-1 trial led to an increase in the use of DOACs and the subsequent BRUISE CONTROL-2 trial.[15] The BRUISE CONTROL-2 trial investigated whether continued DOAC therapy at the time of device surgery (n = 319) was superior to interrupted DOAC (n = 328) therapy regarding the prevention of clinically significant hematomas. In the interrupted DOAC group, the last dose was taken 2 days prior to surgery. This trial demonstrated that continued DOAC use was not superior to interrupted DOAC use in the periprocedural period in preventing clinically significant hematomas.

Anesthetic Techniques and Potential Complications for AF, VT, and Supraventricular Tachycardia Ablation

General Considerations and Anesthetic Agent Options

The anesthetic used for procedures depends on several factors, including the level of sedation (ie, minimal/moderate/deep sedation or GA) and the method of airway management (ie, facemask, laryngeal mask airway, or endotracheal intubation). The electrophysiologic effects and

special considerations of the most common anesthetic agents are presented in Table 1.[16]

General anesthesia may be the best approach in some device implant cases (eg, prolonged cardiac resynchronization procedures), lead extraction cases, and complex ablation procedures during which patient movement can cause critical shifts in the 3-dimensional (3D) mapping system (Supplemental Table/Figure S1). The proposed length of the procedure and the need for arrhythmia inducibility, as well as patient health factors such as obesity, OSA, airway concerns, and the patient's overall cardiovascular health, must be considered to ensure that the appropriate anesthetics and analgesic agents are selected and to reduce procedure risk and maximize positive patient outcomes.

The anatomic location where the arrhythmia originates is important to consider because it can influence the anesthesiologist's decision regarding sedation. Arrhythmias that originate from a single focus, such as focal atrial tachycardia, can be influenced by autonomic tone and can be suppressed quite easily with various anesthetic agents. Therefore, these ablations are performed usually under minimal sedation. Typical atrial flutter (AFL) is a macroreentrant rhythm originating in the right atrium, and usually is amenable to ablation with an ablation line along the cavotricuspid isthmus. Typical AFL ablation involves a shorter procedure time than AF ablation because it entails a linear ablation line along the cavotricuspid isthmus located in the right atrium, and does not require transseptal puncture. Because AFL is a reentry arrhythmia, mapping and ablation are done anatomically without requiring the patient to have the arrhythmia in the electrophysiology laboratory. Typical AFL ablation can be done either under monitored anesthesia care or GA. Atypical AFL ablation are typically left-sided circuits requiring transseptal puncture, and should be managed from an anesthesia perspective in a similar manner to AF ablation.

For catheter ablation procedures, the mechanism of the arrhythmia is also important to consider, as it can influence the joint decision between the anesthesiologist and the electrophysiologist regarding the level of sedation desired. For instance, ectopic/focal/automatic atrial tachycardias, as well as ventricular ectopic beats, are often suppressed by the level of anesthesia. Therefore, in these types of cases, to allow an adequate induction or frequency of the arrhythmia to ensure accurate mapping, a lighter degree of sedation may be required. In most other cases, including AF, VT, and supraventricular tachycardia ablation (SVT) ablations, GA is recommended. General anesthesia not only increases patient comfort during the procedure, but decreases patient movement critical for accurate mapping, safe transseptal puncture, precise catheter manipulation, and the delivery of ablative energy. Information from the literature regarding the anesthetic strategies for various types of arrhythmias is discussed in more detail below.

Table 1
Anesthetic Agents and Their Electrophysiologic Effects[16]

Anesthetic Agent	Electrophysiologic Effects	Special Considerations
	↑ QTc Enhance ectopic atrial rhythms No effect on SA and AV nodes No effect on accessory pathway	Safe to use
Desflurane	↑ QTc? Inhibitory effects on AV node Tachycardia	Sympathomimetic? arrhythmogenic
Propofol	Inhibitory or no effects on SA node Inhibitory or no effects on AV node No effect on accessory pathway Bradycardia	May not be suitable for ectopic atrial tachycardia ablation; suppresses electrical storm
Midazolam	Vagolysis? Tachycardia?	
Rocuronium	Minimum effects on automaticity	Avoid during phrenic nerve pacing
Vecuronium	Minimum effects on automaticity Bradycardia	Avoid during phrenic nerve pacing
Succinylcholine	Inhibitory effects on AV node Bradycardia or tachycardia	
Fentanyl	↑ Vagal tone	No issues in EP procedures when combined with midazolam
Sufentanil	May ↑ QTc No effect on accessory pathway	
Dexmedetomidine	Enhance vagal activity ↓ ↓Norepinephrine release ↓ Sympathetic tone Bradycardia	Antiarrhythmic in pediatric patients; may not be suitable in EP laboratory
Ketamine	Minimal effects on SA and AV nodes ↑ Atrial conduction time	↑ Heart rate ± BP

Abbreviations: AV, atrioventricular; AVNRT, atrioventricular nodal reentrant tachycardia; AVRT, atrioventricular reentrant tachycardia; BP, blood pressure; EP, electrophysiology; SA, sinoatrial; QTc, corrected QT-interval

Anesthetic Techniques for AF Ablation

Di Biase et al.[17] found that GA has a greater AF ablation success rate than sedation. The authors randomized 257 patients undergoing radiofrequency (RF) catheter ablation for AF to either conscious sedation with fentanyl or midazolam (n = 128) or GA (n = 129).[17] Patients who received GA had a greater single procedure success rate than patients who received sedation (88% *v* 69%, respectively; $p < 0.001$). Among patients who needed another pulmonary vein isolation (PVI) procedure, those who received GA had a lower prevalence of pulmonary vein electrical reconnection than patients who received sedation (19% *v* 42%, respectively; $p < 0.001$). In addition, patients who received GA had shorter fluoroscopy (53 ± 9 minutes *v* 84 ± 21 minutes, respectively; $p < 0.001$) and procedure times (2.4 ± 1.4 hours *v* 3.6 ± 1.1 hours, respectively; $p < 0.001$) compared to patients who received sedation.[17] The authors indicated that the benefits of GA were due to increased catheter stability, with consistent, regular diaphragm movement, and less patient movement.

One possible drawback to GA over conscious sedation is the potential increased risk of esophageal injury with GA. Natale et al.[18] compared GA using propofol (n = 25) versus conscious sedation with fentanyl or midazolam (n = 25), and found that the proportion of patients with esophageal damage detected by capsule endoscopy was greater in those who received GA compared to conscious sedation (12 [48%] *v* 1 [4%], respectively; $p < 0.001$). The authors proposed that the greater incidence of esophageal injury in the GA group may have been due to reduced esophageal motility and deglutition, which could result in ablation at the same esophageal location during the duration of the ablation lesion. The authors also proposed that the lack of deglutition during GA may prevent physiologic cooling, resulting in an increased likelihood that the ablation lesions can extend to the esophagus.[18] However, results from this study should be interpreted with caution due to its relatively small sample size.

Based on the current evidence, GA should be used in most AF ablation cases. This approach ensures patient comfort throughout the procedure. It also allows for mechanical deviation of the esophagus, leading to consistency in ablation lesions and a decrease in tidal volumes, which stabilizes the ablation catheter and avoids patient movement that would result in critical shifts in the 3D mapping system. In cases in which the proceduralist and anesthesiologist deem sedation to be appropriate, studies have examined the combination of dexmedetomidine with fentanyl or remifentanil during AF ablation. Dexmedetomidine is a highly selective alpha-2 agonist with sedative and analgesic effects. The benefit of dexmedetomidine is that it provides sedation and analgesia without significant respiratory depression. The side effects of dexmedetomidine include hypotension, bradycardia, and conduction disturbances.[19] Dexmedetomidine slows sinoatrial (SA) node and atrioventricular (AV) nodal conduction more than propofol; however, it does not appear to affect conduction in the His-Purkinje system.[20] Bradycardia and hypotension tend to be transient and do not typically require treatment. Cho et al. randomized 90 patients to either dexmedetomidine and remifentanil (n = 45) or midazolam and remifentanil (n = 45).[19] Dexmedetomidine and remifentanil provided deeper sedation, less respiratory depression, better analgesia, and higher satisfaction for the proceduralists who were blinded to the sedation regimen. The only significant adverse effects were increased hypotension in the dexmedetomidine and remifentanil group, and an increased incidence of desaturation (ie, oxygen saturation rate <90%) in the midazolam and remifentanil groups.[19]

Potential Complications of AF Ablation

Esophageal Injury

Esophageal injury can occur in up to 47% of patients after PVI[21] due to the general proximity of the esophagus to the posterior wall of the left atrium (Fig 1).[22] Di Biase et al. randomized 50 patients undergoing AF ablation to either receive GA (propofol induction with an inhalation agent via endotracheal tube; n = 25) or sedation with midazolam and fentanyl (n = 25). Patients received a capsule endoscopy the day after the procedure to evaluate for endoluminal esophageal damage.[18] There was a higher incidence of esophageal injury among patients who received GA compared to patients who received sedation (48% *v* 4%, respectively; $p < 0.001$), with normalization of all lesions at repeat endoscopy 2 months after the procedure.[18]

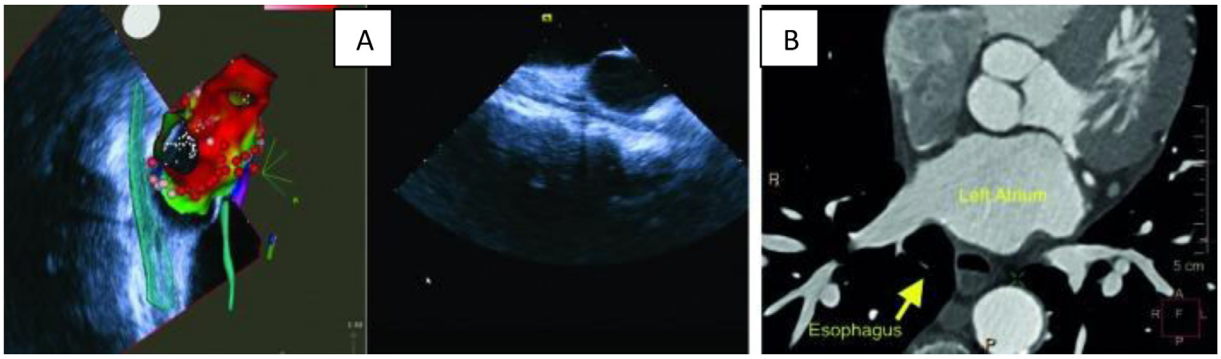

Figure 1. Anatomic relationship between the esophagus and the left atrium (A) Three-dimensional electroanatomic map of the left atrium and the esophagus. (B) Sagittal view of chest computed tomography showing the close proximity of the left atrium to the esophagus. Reproduced with permission from Romero et al.[22]

A rare but potentially fatal complication that typically occurs within 1-to-4 weeks postablation[23] is atrial-esophageal fistula (AEF) formation, with an estimated prevalence of 0.02-to-0.11%.[6] The most common presenting signs and symptoms of AEFs include fever, neurologic symptoms from brain emboli, sudden-onset chest pain, fatigue, malaise, nausea, vomiting, dysphagia, odynophagia, hematemesis, melena, and dyspnea.[24-26] The overall mortality rate of AEFs is approximately 55%—as high as 97% with noninterventional management, 65% with endoscopic management, and approximately 33% for patients undergoing surgery—and typically is caused by air and bacteria entering the left atrium from the esophagus, leading to stroke and mediastinal infection.[23] Due to the high mortality rate of AEF formation, it is important to be familiar with methods to prevent this complication. The risk of AEF formation potentially can be decreased by use of a rapidly responding temperature probe to allow for continuous esophageal luminal temperature monitoring. For example, the CIRCA S-CATH (CIRCA Scientific, Inc, Englewood, CO) esophageal temperature probe has multiple temperature sensors that span a wide area of the esophagus.[27] These sensors respond very rapidly to rises in temperature and can alert the operator to esophageal heating or cooling, which can signal esophageal injury.

Although there is no established standard practice for using multisensor or single-sensor probes, multi-sensor esophageal probes have been shown to provide a superior thermodynamic profile compared to single-sensor temperature probes (Fig 2).[27] Multisensor probes provide greater sensitivity than single- electrode probes (100% *v* 60%, respectively), and similar specificity (60%) for detecting esophageal ulceration.[27] Additional approaches can be used to reduce the risk of esophageal injury when ablating in the posterior atrial wall, including the following: (1) reducing the amount of RF power delivered (eg, ≤25 watts); (2) shortening the RF application time (eg, ≤20 seconds); (3) decreasing contact force (eg, ≤10 grams) using contact force sensing technology; and/or (4) esophageal deviation.[6] (Supplemental Table/Figure S1).

There are various strategies that can be used during AF ablation to laterally displace the esophagus away from the ablation site to decrease the risk of esophageal injury. Mechanical approaches to deviate the esophagus include an endotracheal stylet placed within a thoracic chest tube,[28] displacement with an endoscope,[29] vacuum suction, DV8 inflatable balloon retractor (Manual Surgical Sciences, Minneapolis, MN), and a malleable metal stylet within a plastic tube.[30] Houmsse et al.[31] demonstrated a novel technique that used vacuum suction in 4 animal models. The technique was relatively safe, with only minor (1 mm) esophageal injury after 1 hour of continuous suction, and a 0.2˚C luminal esophageal temperature increase during high-power ablation.

The same authors who used an off-the-shelf malleable metal stylet[30] subsequently reported on the feasibility and effectiveness of a novel esophageal balloon retractor, DV8 (Manual Surgical Sciences, Minneapolis, MN) for manual esophageal deviation in 200 consecutive PVIs.[32] The DV8 inflatable balloon retractor comprises a polyurethane balloon wrapped with a silicone sleeve (Fig 3).[32] It is used to manipulate the esophagus laterally away from the target area of ablation after administration of intravenous heparin and transseptal puncture before creating a geometric 3D mapping of the left atrium.[32] Esophageal deviation was performed to maximize the distance from the trailing esophageal edge to the closest point of the ablation line ($MED_{Effective}$), and correlated with occurrences of luminal esophageal temperature elevation (Fig 4).[32] The esophageal temperature was monitored throughout the procedure by placing a multielectrode temperature probe along the trailing edge of the esophagus. The RF energy delivery on the posterior atrial wall was terminated if the esophageal temperature was >38˚C. The authors reported no instances of AEF formation or clinically evident esophageal trauma. Esophageal deviation also has been shown to improve the durability of posterior atrial wall lesions during AF ablation, decreasing the rate of posterior atrial wall pulmonary vein electrical reconnections compared to only using esophageal temperature monitoring.[33]

Active esophageal cooling is another method to prevent and potentially reduce the risk of severe endoscopically-detected esophageal thermal lesions (EDELs). Tschabrunn et al.[34] conducted a randomized, controlled pilot study of 44 patients undergoing AF ablation who received either esophageal cooling (n = 22) or standard temperature monitoring (n = 22). The EDELs were detected in 5 (22%) patients who received

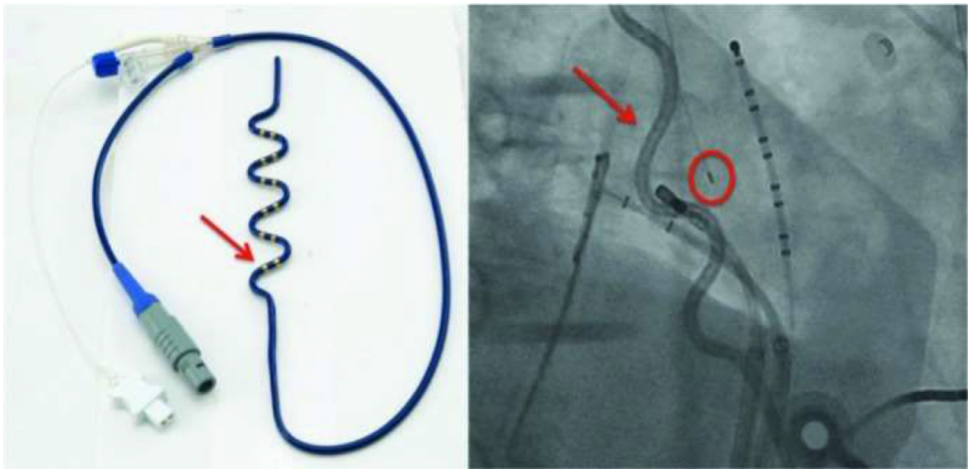

Figure 2. (Left) 12-sensor, sinusoidal temperature probe. (Right) Fluoroscopy image showing the 12-sensor sinusoidal (arrow) and a single-sensor temperature probe (circle). Reproduced with permission from Tschabrunn et al.[27]

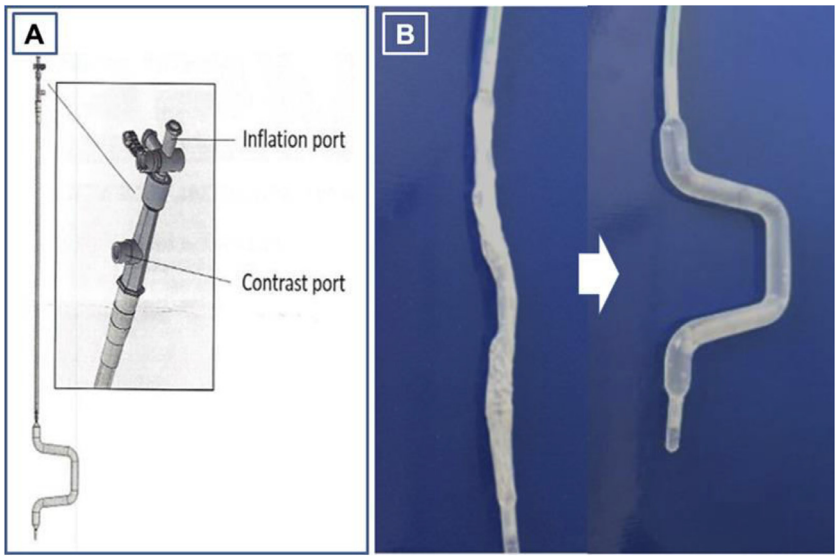

Figure 3. DV8 Balloon Esophageal Retractor Device.

(A) Schematic of the device revealing both the contrast port and the inflation port. Instilling contrast into the former helps delineate the esophageal lumen; while dilute contrast into the latter causes the device to assume its deviated pose. (B) The device is shown both deflated (left) and inflated/deviated (right).

Reproduced with permission from Bhardwaj et al.[32]

esophageal cooling, and in 8 (36%) patients who received standard monitoring. However, severe thermal injury occurred in 3 (60%) of the EDEL patients in the standard monitoring group and in only 1 (12%) patient in the cooling group. These pilot data suggest that active esophageal cooling may reduce the occurrence of severe EDELs.

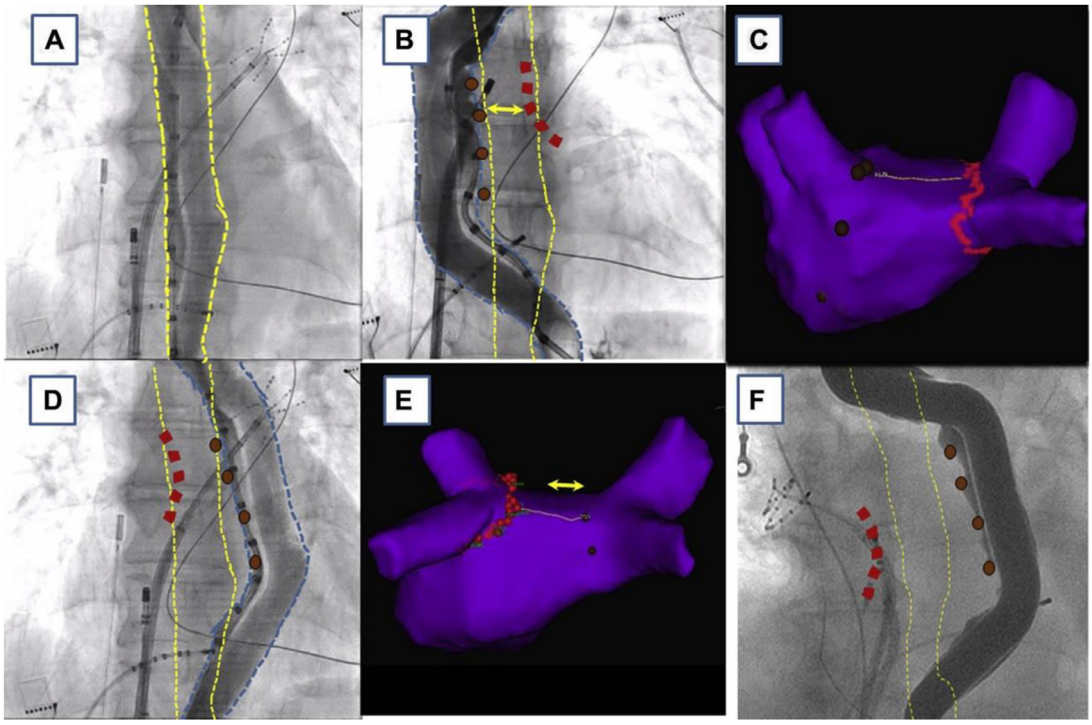

Figure 4. Determining the mechanical esophageal deviation distance from the trailing esophageal edge-to-ablation line.

The balloon retractor is shown (A) deflated just before (B) rightward or (D) leftward esophageal deviation. After identifying the trailing edge of the esophagus by fluoroscopy in the anteroposterior view, a mapping catheter is placed at several vertical points along this edge (brown dots in B and D) and then marked on the electroanatomic map (C and E). Once the ablation lesions are placed, the shortest distance between the trailing esophageal edge and the ablation line is defined as the MED Effective (the distance from the trailing esophageal edge to the closest point of the ablation line). A multielectrode temperature probe was placed along this trailing edge of the esophagus to assess whether the luminal esophageal temperature rose. An example of leftward deviation is shown in another patient without the temperature probe (F) with brown points on the esophageal trailing edge and red dots where ablation was performed. Yellow dotted lines outline where the esophagus was before deviation. Reproduced with permission from Bhardwaj et al.[32]

Phrenic Nerve Paralysis

Phrenic nerve (PN) paralysis is one of the most common complications of AF ablation, occurring more frequently in cryoablation (Supplemental Table/Figure S1). than RF ablation, with a reported incidence of 3.5%-to-7%.[35,36] The right PN can be injured when the right superior pulmonary vein is ablated during RF ablation or cryoablation. The right PN also can be injured by ablation along the posterolateral wall of the right atrium.[37] The left PN can be injured from ablation near the LAA or over the epicardium of the LV.[37] Because the course of the PN varies between patients, high-output pacing should be performed from the ablation catheter before ablation in areas of concern to ensure a safe distance from the PN.

During cryoablation, PN function is monitored by advancing a pacing catheter to an optimal site of PN capture and pacing at reasonably high output to ensure capture.[38] This pacing catheter must always be placed above the level of ablation, capturing the PN at twice the capture threshold. The optimal site for PN capture during ablation of right-sided pulmonary veins is the anterolateral portion of the superior vena cava above the site of ablation. At this location, the PN is separated from the superior vena cava wall only by the pericardium.[38] Given the importance of avoiding ablation near a diaphragmatic nerve, paralytics should not be administered to these patients. Paralytics prevent accurate assessment of PN location during ablation. The PN also can be "mapped" in the nonparalyzed patient by using high-output pacing from the ablation catheter and marking the points on the mapping system where phrenic stimulation occurs.

During implantation of cardiac resynchronization devices, a lead usually is placed into one of the venous branches of the coronary sinus; very often these branches are near the left PN, and, therefore, PN stimulation (PNS) can occur.[39] A PNS occurs in 2%-to-37% of implanted patients and is not always detected in the supine position during implantation.[40] The lateral and posterior veins are at a higher risk of PNS than the anterior veins, and apical positions are at a higher risk than basal positions.[40] It is essential to evaluate the possibility of PN capture during implantation of LV leads. In patients in whom paralytics are given before LV lead placement, the identification of PN stimulation can be impossible until the effects of the paralytic agent have waned or have been reversed. If this goes unnoticed during the case, PNS may become evident at the end of the case or in the recovery room at a time when lead repositioning may be challenging, and would require a repeat procedure for lead repositioning.

Anesthetic Techniques for VT Ablation

The depth of anesthesia and the specific agents used have effects on VT induction and the overall success of the procedure. Propofol causes respiratory and cardiovascular inhibitory effects, which can suppress ventricular arrhythmias and is, therefore, counterproductive for inducing VT. Specifically, propofol is a myocardial depressant and causes a reduction of systemic vascular resistance. Propofol also has been shown to convert both SVT and VT. Although the underlying mechanism of these effects remains unclear, it is likely related to the suppression of both sympathetic and parasympathetic tone and effects on sodium, calcium, and potassium channels in the myocardium.[41] Opioids also inhibit sympathetic reflexes and can suppress VT induction. Depth of anesthesia and patient selection are also important factors. Patient movement during the ablation procedure can affect the 3D mapping system and cause maps to shift, leading to excess procedure duration due to the need to remap the arrhythmia. Patient movement also can affect ablation catheter stability and contact force, thereby limiting the success of the procedure.

Nof et al.[42] retrospectively compared 226 patients with sustained VT and structural heart disease who underwent RF ablation for scar-related VT under intravenous sedation (n = 155) or GA using propofol or isoflurane (n = 71). There were no significant differences in VT inducibility, complications, or elimination of VT between the sedation and GA groups. General anesthesia was associated with an increased need for hemodynamic support without negatively impacting outcomes of VT stability. Data were then prospectively collected from a subgroup of patients with scar-related VT and an implanted defibrillator (n = 73) who underwent noninvasive programmed stimulation (NIPS) for VT induction prior to ablation. These patients then received GA and underwent invasive programmed electrical stimulation prior to VT ablation, which allowed for comparison of NIPS to invasive programmed stimulation after GA. Of the 56 patients who were inducible with NIPS and under GA, 28 (50%) had the same induced VTs, and 28 (50%) had different induced VTs. The clinical VT was reproduced with NIPS in 17 of 23 patients (74%), and under GA in 13 of 23 patients (59%). The authors concluded that GA does not prevent inducible VT in most patients.

Potential Complications of VT Ablation

Patients undergoing VT ablation often have poor cardiac reserve, with a complex substrate (ie, areas of significant myocardial fibrosis), multiple comorbidities, including concomitant heart failure, and incessant or recurrent VT treated by multiple ICD shocks, which can increase the risk of periprocedural hemodynamic decompensation. Acute hemodynamic decompensation occurs in approximately 11% of patients, and is associated with an increase in mortality.[43] The "PAAINESD" risk score was developed to help identify patients undergoing scar-mediated VT ablation who are at increased risk of hemodynamic compensation and may benefit from mechanical circulatory support (MCS).[43] The benefit of hemodynamic support for unstable VT with a percutaneous LV assist device or extracorporeal membrane oxygenation should be weighed against the potential adverse effects of these support devices, such as vascular injury, hemorrhage, and stroke.[44] Decreasing the risk of hemodynamic deterioration during VT ablation by cautious use of cardiodepressive medications such as propofol could decrease the need for MCS devices.[44] The benefit of MCS versus the risks should be assessed for each individual patient.

During the induction of sustained VT in patients with ischemic and nonischemic cardiomyopathy, hypotension can occur due to the purposeful induction of a nonperfusing rhythm, which results in a temporary decrease in end-organ perfusion.[43] One strategy to address this issue is to use MCS, such as an Impella (Abiomed Inc, Danvers, MA) cardiac support system or venoarterial extracorporeal membrane oxygenation, but these are typically not needed if the scar/substrate is mapped. For long (eg, 4-6 hours) complex VT ablation cases, and when mapping and ablating VT in patients with structural heart disease, the use of GA is suggested while considering the aforementioned complications—the length of the procedure and patient stability. The use of GA allows for epicardial access for the mapping and ablation of VT circuits based on the epicardial surface. However, if GA is chosen and the arrhythmia is suppressed, the case may need to be aborted or the operator may perform a scar-based ablation.

Monitoring patients undergoing complex VT procedures is performed with standard American Society of Anesthesiologists monitors. In addition, invasive pressure monitoring is recommended to evaluate for hemodynamic instability during VT induction. Defibrillator pads also should be placed on the patient prior to the procedure. Though MCS devices may be necessary, complex VT cases may be performed with intraprocedural vasopressor or inotropic infusions. There are no established guidelines for which agents to use, and, therefore, the selection of agents is left to the discretion of the anesthesiologist and electrophysiologist.[45]

Anesthetic Techniques for Supraventricular SVT Ablation

The SVT ablations can be performed with GA or sedation with benzodiazepines and opiates. A benzodiazepine-opiate combination has a less-negative impact on SVT inducibility compared to GA.[46] However, remifentanil has demonstrated conduction slowing of both the SA and AV nodes.[47] The use of propofol during SVT ablation has been shown to cause dose-related depression of the sinus node and His-Purkinje system function, with no effect on AV nodal function.[48] This dose-related depression does not suppress inducibility in most cases of SVT.[48] Lai et al.[49] examined the use of propofol infusion in 150 patients who underwent an electrophysiology study. Of the 152 tachyarrhythmias induced, 148 underwent successful RF ablation.[49] However, 4 out of 7 ectopic Ats terminated after propofol infusion, suggesting that this type of arrhythmia may be sensitive to suppression when propofol is administered.

Two studies[50,51] have examined the use of inhalation anesthetic agents for GA during EP cases. Both studies compared the effects of propofol to isoflurane on cardiac conduction in patients undergoing RF ablations for tachyarrhythmias, including SVT. Neither study found differences between the 2 agents for arrhythmia inducibility, SA node conduction time, AV node conduction time, or accessory pathway conduction. To the best of the authors' knowledge, there are no studies that have compared inhalation agents to intravenous sedation in patients implanted with pacemakers or ICDs.

When used as a continuous infusion, dexmedetomidine can achieve both analgesia and sedation without significant respiratory depression.[52] In a retrospective review of 163 consecutive SVT ablations in which dexmedetomidine was the primary sedative used, SVT inducibility during the electrophysiology study was not suppressed.[53] Regardless of the type of anesthesia used for SVT ablation, the appropriate balance between patient comfort and a level of sedation that will not suppress arrhythmia induction should be a primary focus.

Ventilatory Considerations During Ablation Procedures

There is a significant respiratory motion of the heart during spontaneous or positive-pressure ventilation. Evidence suggests that decreasing respiratory excursion leads to better catheter stability, more durable and complete lesions, and reduced procedure time.[54,55] Poor contact force can result in edema and transient injury without adequately ablating the arrhythmogenic substrate, resulting in long-term arrhythmia recurrence. There are 3 ventilatory methods to help overcome respiratory variation, including high-frequency jet ventilation (HFJV), high-frequency, low-tidal-volume ventilation (HFLTV), and ventilatory breath-hold and/or apnea.

HFJV

Ablation catheter contact force can be affected due to the regional myocardium to be targeted being displaced by cardiac and respiratory motion. During HFJV, small amounts of gas can be introduced into the airway through a noncompliant catheter or cannula at relatively high frequencies of 100-to-130 breaths/min.[56] Jet ventilation minimizes diaphragm movement, reduces ablation catheter displacement, and improves tissue contact, leading to shorter ablation times and a decrease in AF recurrence.[55]

The HFJV was first used during PVI for AF in 2006 by Goode et al.[55] The authors found that HFJV produced less posterior left atrial motion and increased ablation electrode-endocardial contact time compared to intermittent positive-pressure ventilation (IPPV). In addition, HFJV decreased left atrial volume, the maximum change in left atrial pressure, and pulmonary vein flow compared to IPPV. The HFJV also decreased motility of the left and right interpulmonary vein saddle sites, the number of RF applications, and ablation procedure time.[55]

Elkassabany et al.[57] performed a retrospective analysis examining the use of HFJV in 188 consecutive patients undergoing PVI for AF. An HFJV was performed successfully for 175 ablations, with 13 (7%) patients converted to IPPV due to carbon dioxide (CO_2) retention, resulting in respiratory acidosis.[57] Body mass index did not correlate with respiratory acidosis in these cases. However, a prior study found that a body mass index >26 is associated with CO_2 retention during suspension laryngoscopy using HFJV.[58]

Aizer et al.[59] conducted a small study of 20 patients who underwent AF ablation, and found cardiac pacing reduced catheter-tissue contact variability and enhanced impedance reduction. Given the independent benefits of HFJV and cardiac

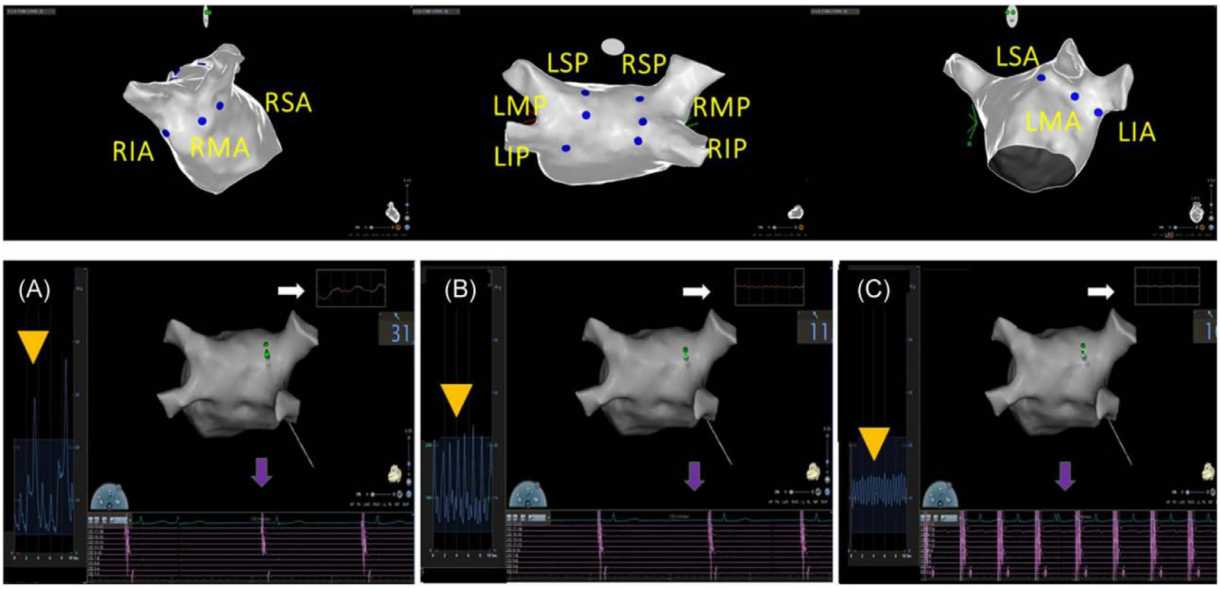

Figure 5. Anatomic locations of lesions across the left atria with the representative effect of HFJV combined with rapid pacing. In images (A), (B), and (C), the white arrow indicates the respiratory pattern of each image, the purple arrow indicates the cardiac rhythm, and the orange triangle indicates the contact force curve. (A) Significant contact force variability (orange triangle) with standard ventilation and a nonpaced rhythm. (B) With the initiation of HFJV (white arrow), there are large reductions in peak contact force and overall variability. (C) With the addition of rapid pacing (purple arrow) to HFJV, there is an additional reduction in contact force variation.

Abbreviations: A, anterior; HFJV, high−frequency jet ventilation; I, inferior; L, left; M, mid; P, posterior; R, right; S, superior. Reproduced with permission Aizer et al.[54]

pacing on catheter stability during AF ablation, Aizer et al. subsequently investigated if there was an additional benefit from combining cardiac pacing and HFJV among 40 patients who underwent AF ablation.[54] The authors found that the combination of rapid atrial pacing and HFJV (n = 20) improved catheter stability compared to standard ventilation (n = 20; Fig 5).[54] Limited expertise with HFJV among many anesthesiologists is one potential caveat to widespread implementation of HFJV at this time.

Potential Complications of HFJV

Although there are no absolute contraindications, there are relative contraindications to HFJV. An HFJV should be avoided in patients with intracranial or pulmonary hypertension, in which periods of hypercarbia would worsen their disease states.[60] An HFJV also can lead to potential complications and subsequent effects from these complications (Table 2).[61] Potential complications include barotrauma, inadequate humidification of gasses, and impaired ventilation and oxygenation.[61]

Due to possible variations in oxygenation and ventilation associated with HFJV, the patient's oxygenation and ventilation should continue to be assessed. It is recommended to perform serial arterial blood gasses 30 minutes after the initiation of HFJV and afterwards.[16] It is important to establish the arteriolar−end-tidal CO_2 ($ETCO_2$) gradient in order to use the $ETCO_2$ as a measure of assessing the adequacy of ventilation. The $ETCO_2$ can be measured during HFJV by stopping jet ventilation and delivering a series of tidal volume breaths. If an increase in $ETCO_2$ is identified, jet ventilation should be discontinued, and normal tidal volume breaths should be given. The F_IO_2 can be adjusted if the SpO_2 decreases.

HFLTV

There are several barriers preventing the widespread use of jet ventilation, including proper equipment, provider comfort, and anesthetic agent choices during jet ventilation. In an attempt to address these barriers, Gabriels et al.[62] retrospectively examined data from 44 consecutive patients who underwent RF ablation (Supplemental Table/Figure S1). of paroxysmal or persistent AF using a standard ventilator with high respiratory rates (40-50 breaths/min) and low tidal volumes (200-250 mL). The catheter stability and clinical outcomes of these patients were compared to an age- and sex-matched control group (n = 44) from the same period who

Table 2
Potential Complications and Subsequent Effects of High-Frequency Jet Ventilation

Barotrauma	Inadequate Gas Humidification	Impaired Ventilation
Pneumothorax	Mucosal trauma	Hypercarbia
Pneumomediastinum	Tracheal necrosis	Hypoxemia
Pneumopericardium	Atelectasis	Elevated intracranial pressure
Subcutaneous emphysema		Elevated pulmonary artery pressure
Hypotension		
Right ventricular failure		

NOTE. Table adapted from Conlon, 2012[61]

underwent AF ablation with normal ventilator settings (tidal volume: 6-8 mL/kg, respiratory rate: 14-15 breaths/min). General anesthesia was maintained in both groups with sevoflurane and remifentanil without paralytic agents. The results indicated that HFLTV ventilation significantly decreased contact force variability and led to reductions in ablation, procedural, and fluoroscopy times, but did not reduce AF recurrence. Additional sedation can be given if the patient is overbreathing the ventilator. Alternatively, the patient can be paralyzed after consulting with the electrophysiologist (see PN considerations section).

The HFLTV ventilation has multiple advantages over jet ventilation. Jet ventilation does not allow for monitoring of $ETCO_2$. As a result, serial blood gas samples should be collected to monitor pH and CO_2 levels.[62] In addition, there is no method to deliver volatile anesthetics during jet ventilation. Therefore, there is a reliance on total intravenous anesthesia, which likely explains the increased vasopressor use in jet ventilation. Jet ventilators are also more expensive, at an average cost of $35,000, and the use of these ventilators requires specialized training. The HFLTV ventilation uses a standard ventilator and does not incur additional costs or require additional training.[62]

Potential Complications of HFLTV

During low-tidal-volume ventilation, end-tidal inhaled anesthetic and $ETCO_2$ may not correctly reflect the concentration of these gases in the alveoli. A significant portion of the ventilated lung volume during HFLTV is deadspace; therefore, it is reasonable to maintain $ETCO_2$ at 20-to-25 mmHg to prevent the patient from overbreathing the ventilator. Because anesthetic agents are not absorbed in lung deadspace, higher end-tidal inhaled anesthetic levels are needed. Low-tidal-volume ventilation also may predispose patients to atelectasis. If hemodynamics allow, positive end-expiratory pressure can be added to prevent atelectasis.

Ventilatory Breath-hold and/or Apnea During Ablation

During challenging catheter positions with decreased stability, the electrophysiologist may request that the anesthesiologist perform a breath-hold to maintain chest stiffness during the ablation process by temporarily suspending chest wall movement due to respiration. In these cases, the anesthesiologist should communicate concerns regarding hypercarbia and hypoxia and when resumption of normal respiration is necessary.

Additional Potential Complications in the Electrophysiology Laboratory

Cardiac Perforation and Pericardial Tamponade

One of the most important and serious complications in the electrophysiology laboratory is perforation of the heart, resulting in cardiac tamponade. This complication can occur during device implantation, lead extractions, or during any type of ablation. Cardiac tamponade has a reported incidence of 1% in PVI procedures,[63] 0.1%-to-5.2% during permanent pacemaker and/or ICD implantations,[64] and 0.3%-to- 1% of transvenous lead extractions.[65,66] Accurate blood pressure assessment is critical during high-risk procedures such as transvenous lead extractions, and an arterial line is highly recommended. Radial arterial line blood pressure monitoring can be considered for patients with structural cardiac disease undergoing VT ablation and AF ablation. Invasive blood pressure monitoring improves the ability to rapidly and accurately detect hypotension, which can result in an earlier assessment of possible pericardial effusion/ pericardial tamponade. There are no established guidelines on the optimal modality for blood pressure monitoring for patients with LV systolic dysfunction undergoing cardiac ablation for AF. The 2017 Heart Rhythm Society expert consensus statement indicated that 60% of the writing group members used an arterial line for blood pressure monitoring during catheter ablation for AF.[6] In patients in whom the risk of cardiac tamponade is increased, it is critical that the electrophysiologist is notified immediately when hypotension develops to investigate the etiology of the hypotension and ensure timely treatment.

To assist with determining the etiology of hypotension, and, specifically, excluding cardiac tamponade as the cause, an intraprocedural TEE should be performed, which allows for a quick assessment of the pericardial space when hypotension occurs. An ICE has become a standard tool during ablation procedures.[67] Either rotational or phased-array ICE can be used to evaluate for a pericardial effusion[67] should persistent hypotension occur. If ICE is not available, TTE can be used.

Fluoroscopy also can be used to aid in the diagnosis of cardiac tamponade by revealing a reduction in the excursion of the cardiac silhouette. Huang et al.[68] found that a reduction in the excursion of the cardiac silhouette can be an early sign of cardiac tamponade and, in some instances, an even earlier sign than hypotension. Therefore, if there is an intraprocedural concern for tamponade, an accurate assessment of the excursion of the cardiac silhouette should be performed before initiation of the procedure to obtain a baseline for comparison. Another useful fluoroscopic sign is the "pericardial stripe," which can be derived from direct visualization of pericardial effusion. The "pericardial stripe" (Fig 6) refers to visualizing a fluid density between epicardial and pericardial fat (specificity 94%, sensitivity 12%), and is due to fluid displacement of the epicardial fat pad.[69]

The management of cardiac tamponade is emergent pericardiocentesis via a subxiphoid approach. Therefore, a pericardiocentesis kit should be available during any case in which cardiac perforation and tamponade are a possibility. In procedures such as AF ablation, in which systemic intraprocedural anticoagulation via continuous intravenous infusion of heparin is required, administration of the reversal agent protamine should be administered. If blood continues to leak into the pericardium, the use of a cell-saver with "autotransfusion" may be of use to maintain intravascular volume and hematocrit. Cardiothoracic surgery consultation also should be obtained in these cases to evaluate the need for a surgical

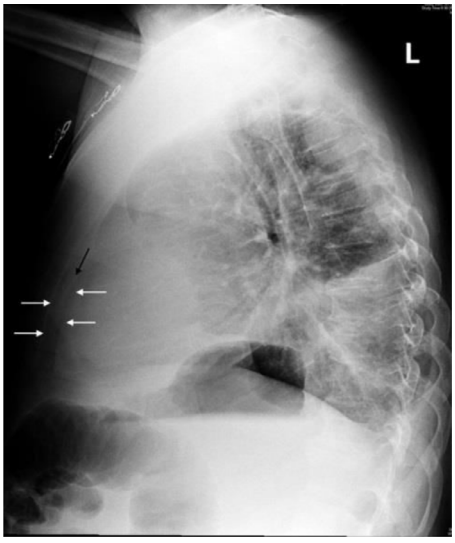

Figure 6. Lateral chest radiograph demonstrating a pericardial effusion by visualization of the "pericardial stripe" (radiopaque region (black arrow), which is the fluid density between the radiolucent epicardial and pericardial fat stripes (white arrows). Reproduced with permission from Li et al.[69]

pericardial window, which has been reported in approximately 16% of procedures.[6] In cases in which periprocedural oral anticoagulation is given, reversal of these agents should be considered by using fresh frozen plasma and/or 4-factor prothrombin complex concentrate for reversal of warfarin, idarucizumab for patients on dabigatran, and andexanet alfa for patients on apixaban or rivaroxaban.[6,70,71]

Vascular Complications

The most common complications during catheter-based electrophysiology procedures are vascular access-related, which include hematomas, bleeding, pseudoaneurysms, arteriovenous fistulae, and retroperitoneal hematomas. Sharma et al. found that the use of ultrasound-guided access was associated with a decreased 30-day risk of vascular complications, with an overall femoral vascular access-related complication rate of 3.2% and 1.2% rate of major complications (Bleeding Academic Research Consortium 2+ bleeding/hematoma, AV fistula, or pseudoaneurysm). Compared with conventional anatomic landmark-guided vascular access, the use of ultrasound guidance decreased rates from 5.3% to 1.1% for all complications and from 2.5% to 0.6% for major vascular access complications.[72] Of the vascular access complications, groin hematomas are the most common, with symptoms and effects ranging from anxiety and comfort to pseudoaneurysms, arteriovenous fistulae, and hypovolemic shock.[6]

Thromboembolism and Air Embolism

In patients who are undergoing AF or VT ablation in which transseptal access is common, systemic anticoagulation with intravenous unfractionated heparin should be administered intraprocedurally, regardless whether anticoagulation was used preprocedure. This reduces the risk of thrombus formation on the catheters, sheaths, left atrium, and at ablation sites. During AF catheter ablation, heparin should be administered prior to or immediately after transseptal puncture, and should be monitored and adjusted to achieve and maintain an activated clotting time of at least 300 seconds (class I recommendation).[6] Protamine also may be administered at the end of the ablation to reverse the effects of heparin. For VT ablation, unfractionated heparin also should be administered after sheath insertion as an initial bolus (empirical dose 5,000-10,000 IU or 50-100 IU/kg), followed by intermittent bolus and/or continuous infusion to maintain a target activated clotting time of longer than 250-to-350 seconds.[44] Additionally, continuous flushing of all sheaths with heparinized saline should be considered, including aspirating and flushing sheaths with any catheter exchanges.[73]

An air embolism complicating catheter ablation is an uncommon but potentially fatal event, with an incidence of $<1\%$, which can occur as a result of air entry into the vasculature.[6] Particularly in procedures such as AF, which requires transseptal access to the left atrium, the most common mechanism of air entry is the introduction of air via the transseptal sheath. Other mechanisms include the entry of air through the infusion line and negative suction when catheters are removed. Signs and symptoms suggestive of an air embolism into the cerebral vasculature include altered mental status, seizures, and focal neurologic deficits, which may be difficult to recognize depending on the depth of sedation. Intraprocedurally, a common presentation of air embolism during AF ablation is acute coronary ischemia, which could present clinically as acute ST elevations, ventricular fibrillation, or heart block.[6] The right coronary artery is usually the culprit coronary artery involved, likely due to the superior position of the right coronary artery ostium in the supine patient. Air embolisms are managed mostly with supportive care, usually resulting in complete resolution of symptoms and signs within minutes. However, cardiac pacing and cardiopulmonary resuscitation might be needed in cases of persistent hypotension, AV block, or ventricular fibrillation. Postoperatively, an atrioesophageal fistula should be ruled out if an air embolism is documented after the ablation. Preventative measures should be used in all catheter ablation cases, such as monitoring all infusion lines for bubbles and withdrawing the catheter slowly to minimize suction effects, along with the simultaneous aspiration of the fluid column within the sheath.[6] In the case of cerebral air embolisms, the most important initial step is to maximize cerebral perfusion by administering intravenous fluids and supplemental oxygen, which increases the rate of nitrogen absorption from air bubbles. Other treatment measures are to briefly suspend the patient in a head-down position and use of hyperbaric oxygen.[6]

Hemodynamic Considerations

During electrophysiology procedures, phenylephrine is a typical and effective alpha-agonist used to counteract vasodilatation and hypotension related to anesthetic agents.[74] As previously discussed, if vasopressor requirements to maintain blood

pressure increase at any point during the procedure, pericardial tamponade needs to be suspected and ruled out immediately.

Isoproterenol is often used to help induce SVT or "AF triggers" during catheter ablation procedures. During AF ablation procedures, graded infusion doses of isoproterenol as high as 20-to-30 μg/min can be administered.[6] However, this commonly results in hypotension related to vasodilatation, which can be counteracted with phenylephrine.

It should be noted that during AF ablation procedures, whether RF or cryoablation, significant vagal responses can be elicited, most commonly associated with ablation near the left superior pulmonary vein. This response is due to stimulation of the ganglionated plexi that lie in the antra of the pulmonary veins. This effect is transient and does not warrant any treatment. Interestingly, there are data that suggest vagal responses may be associated with an increased success rate of the procedure and decreased risk of AF recurrence.[75] It is important that the procedure team be aware of a potential vagal response to avoid the unnecessary treatment of transient hypotension and bradycardia.

Volume Overload Due to Excess Fluid Administration

A large volume of fluid (≤4 L) can be infused into the patient during ablation of arrhythmias with irrigated catheters, especially RF ablation of AF or VT. The volume from intravenous medications and continuous sheath flushing can be especially high. However, it is important that the anesthesiologist limit the volume of fluid administered to decrease the risk of hypervolemia. The patient also should be monitored for signs of fluid overload.

Biase et al.[76] randomized 138 patients undergoing ablation for long-standing persistent AF to 1 of the 3 following groups: group 1 (n = 46) received ablation with a standard 3.5-Fr open irrigated catheter, and was discharged with 40 mg twice daily of furosemide; group 2 (n = 46) received ablation with a standard 3.5-Fr open irrigated catheter and was discharged with 80 mg twice daily of furosemide; and group 3 (n = 46) received ablation with the Surround Flow (Biosense Webster, Diamond Bar, CA) open irrigated catheter and was discharged with 40 mg twice daily of furosemide. At ablation completion, all 3 groups were given 40 mg intravenous furosemide in addition to fluids (groups 1 and 2 received 4.4 L; group 3 received 1.7 L). No patients in group 3 were rehospitalized for fluid overload, whereas 22% in group 1 and 11% in group 2 were rehospitalized for fluid overload. Group 3 also had significantly less weight gain than groups 1 and 2.

Previous ablation catheter irrigation systems used a flow system that delivered the irrigating fluid only to the distal surface of the tip electrode. This resulted in an inhomogeneous flow, which was associated with excessive saline perfusate volumes and the possibility of thrombus formation on the nonirrigated part of the electrode.[77] The Surround Flow entire tip system features widespread distribution of the irrigating solution by increasing the number of irrigation holes from 6 to 56, which enables homogeneous cooling, protection from thrombus formation with lower flow rate requirements, and reduces the incidence of steam pop (Supplemental Table/Figure S1)..[77-79] Per manufacturer recommendations, the SF ablation catheter uses 17 mL/min of fluid to cool the ablation catheter, which is an almost 50% decrease from the conventional irrigation rate of 30 mL/min.[80,81] Despite this technology, fluid administration is still significant during these procedures. The fluids required to flush sheaths (eg, transseptal sheaths) typically are flushed continually throughout the procedure and during the use of vasopressors and anesthetic agents. Patients also tend to retain fluid, which may be due to atrial natriuretic peptide depletion because of ablation. As a result of these factors, the most common cardiac etiology of hospital readmission after AF ablation is congestive heart failure.[82] Therefore, it is the authors' routine practice to administer intravenous furosemide at the end of most AF ablation procedures and to discharge patients on oral diuretics for approximately 3-to-5 days.

Other Perioperative Considerations

Antibiotic Prophylaxis

Prophylactic antibiotic administration has been shown to reduce complications due to infections in patients undergoing implantation of pacemakers or cardioverter-defibrillators.[83] Prospective and retrospective studies indicate that the incidence of infection related to permanent pacemakers ranges from 0.13%-to-19.9%, with serious complications, including sepsis and endocarditis, occurring in approximately 0.5% of patients.[84-86]

De Oliveria et al. conducted a prospective, randomized, double-blind study that examined the efficacy of cefazolin in patients undergoing device implantation. Patients were randomized to receive cefazolin (n = 314) or placebo (n = 335), with follow-up occurring at 10 days, 1, 3, and 6 months after discharge. The study was stopped early due to a significant difference in the number of patients with infections favoring the cefazolin group (2 of 314 patients, 0.63%) compared to the placebo group (11 of 335, 3.28%; risk ratio = 0.19; p = 0.016).[83]

To the best of the authors' knowledge, the efficacy of antibiotic use for catheter-based electrophysiology procedures has not been studied. As a result, there is no consensus among healthcare centers regarding the administration of antibiotics prior to the procedure, and future studies are warranted.

Regional Anesthetic Techniques for Subcutaneous Implantable Cardioverter-Defibrillator

Placement of the Subcutaneous Implantable Cardioverter-Defibrillator (S-ICD; Boston Scientific, Marlborough, MA) was developed as an alternative to the transvenous ICD (TV-ICD) for patients who do not need the pacing function in a device. The perioperative management of S-ICD is different from TV-ICD due to the extent of tissue dissection required for S-ICD devices. The optimal anesthetic technique for this procedure has not been studied; however, regional anesthetic techniques have been evaluated to increase patient comfort and tolerance of the procedure.[87]

In contrast to the small infraclavicular location of TV-ICD placement, the S-ICD requires a larger area of coverage. Local infiltration is generally sufficient for the placement of TV-ICD devices, though it may not be enough for S-ICD devices. The S-ICD placement requires coverage of the left anterolateral chest wall as well as the left parasternal region for tunneling of the electrodes. Possible regional techniques include but are not limited to thoracic epidural or paravertebral block and/or catheter placement, or truncal nerve blocks (eg, pectoral nerve blocks I and II, serratus anterior plane block, or transverse thoracic muscle plane blocks). Though both the epidural catheter and paravertebral catheter or block placement would provide dense coverage to the desired area for S-ICD placement, the patient's anticoagulation status often precludes the use of these techniques. Additionally, the use of epidural placement is less common in ambulatory patients, and the sympathectomy that occurs with epidural anesthesia may not be tolerated by this subset of patients.

There are limited data evaluating the utility of regional anesthesia for S-ICD placement.[87] Despite regional blockade of the target areas, patients may still require anesthetic techniques ranging from minimal sedation to GA. These regional techniques are likely best used as analgesic adjuncts which lower the sedation requirement of the procedure rather than a primary anesthetic.

Postoperative Period Care

Cardiac ablation procedures and cardiac implantable electronic device implants can be associated with varying degrees of postoperative pain, especially during early postoperative care.[88] Untreated or inadequately treated pain can result in emotional distress, immobility, and a prolonged hospital stay. Therefore, postoperative analgesia is an important measure of clinical performance and patient satisfaction.

Bode et al.[88] aimed to quantify postinterventional pain for 102 patients who underwent cardiac ablation and cardiac implantable electronic device implant and/or explants, using a numeric rating scale (NRS 0-10) every 2 hours, over 24 hours postintervention. Forty-nine patients underwent ablation with deep sedation using intravenous propofol, fentanyl, and midazolam; and 53 device surgeries were performed with local anesthesia using 20 mL of mepivacaine 1%, along with additional intravenous fentanyl for analgesia if necessary. Pain was classified as moderate-to-severe if the NRS was >3. Postinterventional analgesia with nonopioid and opioid analgesics was used at the discretion of the treating physicians. Sixty patients (59%) suffered from moderate-to-severe pain within the first 24 hours after the procedure, and the most commonly reported symptoms were back pain (44%) and pain at the device pocket site (39%).[88] This study highlighted the relatively high prevalence and poor predictability of postoperative pain in this patient population despite the treating physician's discretionary use of periinterventional analgesics for pain management.

The management of postoperative anticoagulation is important for decreasing the risk of potential postoperative complications, including thromboembolism. An AF ablation results in a prothrombotic state that can last for several days to months, and, therefore, it is recommended that patients be anticoagulated for at least 2 months after AF ablation regardless of their CHA_2DS_2-VASc score or rhythm status.[6] Patients on warfarin who have a subtherapeutic international normalized ratio the day of the procedure can be given any of the following: (1) direct thrombin inhibitor, (2) factor Xa inhibitor several hours after ablation, (3) low-molecular-weight heparin, or (4) intravenous heparin as a bridge to the resumption of warfarin, with an international normalized ratio between 2.0 and 3.0.[6] After more extensive VT ablation in which ablation was performed over an area with >3 cm between ablation sites, antithrombotic therapy is recommended with either aspirin 325 mg/d or anticoagulation with warfarin.[44]

Appropriate triaging for postprocedure disposition can affect the length of stay and hospital costs. Same-day discharge is appropriate after an uncomplicated right-sided VT ablation, whereas at least 24 hours of monitoring on telemetry is appropriate after an uncomplicated left-sided VT ablation due to the need for transseptal puncture. For more complex cases, patients who are hemodynamically unstable or in need of ventilatory support, treatment in an intensive care unit is appropriate.[44] Patients who undergo an uncomplicated SVT, AFL, and/or AF ablation usually can be discharged the same day. Disposition after device implantation is typically provider- and case-specific, whereas patients with uncomplicated device implants can be discharged the same day. If there is any hematoma development, tenuous hemodynamic or respiratory status, patients can be monitored on a telemetry floor until discharge is appropriate.

Future Considerations

Pulsed-field ablation (PFA) is a promising new ablation modality that uses a nonthermal form of energy delivery that targets myocardial cells by creating cell membrane pores (electroporation) that cause myocardial cell death and "spares" other types of cells. Cardiac myocytes appear to have a lower threshold for electroporation, enabling cardiac ablation while minimizing the potential for collateral damage of other tissues such as the esophagus and PN.[89] Animal models have shown preservation of PN and esophageal tissue despite exposure to clinical PFA levels.[89,90] This technology does not require fluid administration to irrigate and cool the ablation catheter electrode. It appears that PFA is safe to use on the posterior wall of the left atrium without the need for esophageal temperature monitoring, esophageal deviation, or esophageal cooling, as this technology is not thermal-based and appears to be specific to ablating cardiac myocytes and not cells comprising the esophagus.[91]

The first pilot study using PFA in humans included 38 patients who underwent PVI, and acute electrical isolation was achieved in all 152 (100%) of the pulmonary veins, with no serious adverse events (eg, PN injury, esophageal injury, stroke, or death) after 30 days.[92] These results indicated that PFA is a promising new modality that has the potential to

circumvent several of the current previously discussed challenges of ablation procedures.

Conclusions

The role of the anesthesiologist in the electrophysiology laboratory has continued to evolve over the past 2 decades. Coordination and communication between the anesthesiologist and the electrophysiologist are essential to ensure an optimal balance of patient comfort, arrhythmia inducibility, hemodynamic tolerance, patient safety, and catheter stability. The anesthesiologist must also be mindful of the various procedural considerations when using specialized technologies and techniques needed to perform ablation and device procedures. Therefore, a multidisciplinary approach to electrophysiology procedures involving the electrophysiologist and anesthesiologist should always be used to optimize patient outcomes.

Conflict of Interest

None.

Supplementary materials

Supplementary material associated with this article can be found in the online version at doi:10.1053/j.jvca.2022.10.013.

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