Anesthetic Management of Electrophysiological Procedures for Heart Failure

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Introduction

Each year, increasing numbers of patients surviving critical cardiac event or with severe cardiac disease are scheduled for an interventional procedure to manage the underlying conditions. Recent advances in medical technology provided cardiologists with tools that were deemed futuristic only a few decades ago. Nowadays, common procedures in the electrophysiological (EP) laboratory include diagnostic studies, ablations of pathways that are involved in arrhythmias, and placement of pacemakers (PM) or implantable cardioverters/defibrillators (ICD). The complexity of these procedures and the medical condition of the patient population resulted in an increasing demand for the consultation and care provided by anesthesiologists. This allows the EP physician to concentrate solely on the technical aspects of the procedure and share the responsibility for the patient with the anesthesia care team. A detailed knowledge of the procedure is thus of a paramount importance for all medical providers. This article reviews the most common interventions in the EP laboratory and the scientific evidence that form the basis for these procedures. Anesthesia techniques that have a specific impact on the procedures will be discussed.

The expanding demand for the assistance of anesthesiologists in a complex care for a cardiac patient has been reflected by an increasing

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INTERNATIONAL ANESTHESIOLOGY CLINICS
Volume 50, Number 3, 22–42

number of recent publications that reviewed this topic.[1–4] The current role of the anesthesiologist is no longer limited to providing comfort to the cardiac patient. Although this is still the foremost goal of the anesthesiologist's care, it is vital to understand that the particular choice of the anesthesia technique can have a profound effect on the procedure. The anesthesiologist's input thus goes above and beyond a mere concept of providing "sedation" versus "general anesthesia" as falsely perceived by many patients and, unfortunately, also some medical providers. It is certainly true that many minor procedures, for example generator change, can be accomplished under "mild sedation" and/or local anesthesia only provided by the EP physician in an outpatient setting. In contrast, the increasing acuity of the critically ill patients combined with growing complexity of EP procedures may require the presence of dedicated, competent providers that support the patients' vital functions and facilitate the procedure.

A recent survey of EP laboratories of academic centers in the United States revealed that majority of centers use a combined model of care in which both anesthesia and nonanesthesia professionals provide sedation for EP procedures. The main reasons for choosing nonanesthesia personnel were the lack of availability and economic issues, taking priority over patient safety. However, over 25% of respondents indicated that care by anesthesia professionals was warranted most (> 50%) of the time regardless of their current care model.[5] Therefore, it could be anticipated that the anesthesiologists' involvement in providing both sedation and general anesthesia in the off-site locations including the EP laboratory will grow. The anesthesiologists should become familiar with the EP laboratory environment and procedures to be able to provide optimal care to the patient with maximum safety and help to facilitate the procedure and enhance the outcomes.

■ EP Procedures

The most common EP procedures reviewed in this article include implantation of a PM or an ICD, its generator or lead change, implantation of biventricular (BiV) cardioverter/defibrillator for cardiac resynchronization therapy (CRT), and percutaneous catheter-based radiofrequency ablation (RFA) of atrial or ventricular arrhythmias. Profound bradycardia or chronotropic insufficiency can be a sole cause of heart failure, treatable with PM implantation. Heart failure and left ventricular (LV) dysfunctions correlate with risk of sudden cardiac death and are among the most common reasons for ICD implantation today. Implantation of BiV devices is essentially restricted to patients with heart failure. Occasionally, tachyarrhythmias amenable to RFA are a primary cause of heart failure, most commonly in patients with tachycardia-induced cardiomyopathy in

the setting of atrial fibrillation (AF) or atrial flutter with rapid ventricular response. Finally, tachyarrhythmias such as ventricular tachycardia (VT) occur frequently in heart failure patients, even if they are not the primary cause of the heart failure. Therefore, anesthetic management during ablation procedures will be discussed in the latter part of this review.

■ Placement of PM and ICD

A placement of a single-chamber or dual-chamber PM is indicated in patients with rhythm abnormalities according to established guidelines. The incision for the placement of the device is usually performed in the upper chest below the clavicle. A subcutaneous pocket to accommodate the device is created. Vascular access for lead placement is then accomplished by cephalic vein cutdown or by subclavian vein cannulation with Seldinger technique. One or 2 leads are then placed and secured in the right ventricle or right atrium, respectively. CRT (see below) requires BiV lead placement, with one lead placed in the right ventricle and the second lead advanced in the coronary sinus, to allow synchronized pacing of both ventricles. The LV lead placement may be technically more challenging. A long sheath is introduced into the right atrium and advanced into the coronary sinus. A suitable coronary sinus branch is usually cannulated with an angioplasty wire, often preceded by a coronary sinus venogram to delineate the venous anatomy. The LV lead is then advanced into the branch over the wire, although it can be sometimes delivered directly, using a stylet to stiffen the lead. Alternatively, LV lead can be placed epicardially by a cardiac surgeon in the operating room.

Similarly, placement of an ICD is accomplished percutaneously with a technique essentially identical to PM placement. The proper function of the ICD is usually tested at the end of the procedure. This is done by an intentional induction of ventricular fibrillation (VF) under deep sedation to confirm appropriate sensing of the arrhythmia by the device and successful termination with an adequate safety margin.

The device battery becomes depleted over time, requiring generator change. In the absence of lead malfunction, this can be a brief procedure that does not involve intravascular manipulation. In other cases, new leads may need to be placed or old leads removed. Common reasons for lead extraction and/or replacement are lead failure or fracture or an infection. Removal of chronic leads often requires a specialized equipment to liberate the lead from intravascular and intracardiac adhesions, such as the so-called laser sheath. Removal of the lead can result in a catastrophic intrathoracic bleeding due to superior vena cava tear or cardiac perforation.

The rate of serious complications during device-related procedures varies with the patient and procedure type. Procedural mortality with a device placement is $<0.1\%$ but can exceed 1% in a sick patient requiring

extraction of chronic leads. A large pneumothorax, intrathoracic bleeding, and cardiac perforation with tamponade are the most common mechanical complication during the procedure.[6]

A PM-dependent patient is usually transiently supported by a temporary pacing catheter during a generator change. However, some EP physicians may choose not to secure the patient with temporary pacing and opt for a "quick" generator change even in the absence of underlying ventricular rhythm. This can occasionally result in profound bradycardia or asystole. A transthoracic pacing should be immediately available for backup pacing.

■ RFA

These procedures involve thermal destruction of a small amount of myocardial tissue, usually with the aim to interrupt a reentrant tachycardia circuit or destroy the focus of focal arrhythmia. They are too complex and variable to allow detailed description here, but placement of multiple intracardiac catheters is required in most cases and followed by a diagnostic portion of the procedure, when the tachycardia is induced and mapped. Eventually, delivery of radio-frequency energy is performed to destroy the target tissue and often followed by attempts at tachycardia reinduction to confirm its successful elimination. In some cases, destruction of a well-defined anatomic target without tachycardia induction is adequate.

The duration of these procedures ranges from <1 to >6 hours. The desired level of sedation is also quite variable, because certain arrhythmias are much easier to induce and map with minimal sedation only. In contrast, general anesthesia is often requested for complex procedures of long duration.

In addition to the standard complications related to vascular access, cardiac perforation with a catheter or transseptal needle, cardiac wall rupture due to a steam pop, or inadvertent destruction of the atrio-ventricular (AV) mode may occur. Patients requiring RFA of VT in the setting of structural heart disease have frequently marginal hemodynamic status and require intense support.

■ Evidence-based Rationale for ICD Implantation and CRT

Placement of an ICD is a routine EP procedure typically performed primarily to decrease the risk of death due to VT or VF. In principle, ICD provides continuous monitoring and automatic evaluation of ventricular rhythm; it delivers either a shock or a burst of overdrive pacing to terminate ventricular arrhythmias. The effectiveness of ICDs

in protection from sudden cardiac death in properly selected patients is supported by an impressive body of evidence.

The concept of ICD device was developed in the 1970s by Mirowski et al.[7] The nature of the implantation procedure has changed dramatically since the publication of the first clinical report in 1986,[8] when placement of the device leads was performed by a cardiac surgeon in the course of coronary artery bypass graft (CABG) or surgical ablation of VT substrate. The early ICD models usually used 1 or 2 epicardial patches for shock delivery and the device size required placement in the abdominal pocket. The reported mortality associated with placement of the epicardial lead system was 2% to 5%. Development of the endovascular defibrillator leads obviated the need for open-heart surgery and decreased implantation mortality below 1%. Endovascular systems became widespread in early 1990s, but the typical device size (150 to 200 cm^3) still required abdominal pocket placement by a surgeon, resulting in a long subcutaneous course of the lead from the pocket to the vascular access site (usually the left subclavian vein).[9] Decrease in the device size in mid 1990s below 100 cm^3 eventually allowed subcutaneous pectoral placement of the device. This shortened the subcutaneous course of the lead and markedly decreased incidence of mechanical lead failure and other complications. It also allowed for the entire procedure to be performed by a cardiologist in a catheterization/EP laboratory.[10–12]

The current generation of devices has volumes of approximately 30 to 40 cm^3, and the mortality associated with device placement is low (0.02% for death during implantation procedure and 0.27% for inhospital death).[13] Lead dislodgement, pocket hematoma, pneumothorax, cardiac tamponade, and infection are the most common early complications. Approximately 150,000 ICDs were implanted worldwide in 2005; a similar number (> 140,000) were implanted in the United States alone in 2009.[14] The decrease in device size was accompanied by other improvements, including increased battery life, delivery of biphasic (as opposed to monophasic) shocks, provision of painless tachycardia treatment (ie, antitachycardia pacing), and improvements in software responsible for rhythm analysis.

In general, the contemporary ICDs can be classified as single-chamber, dual-chamber, or BiV systems. A single-chamber system consists of a single lead with a tip placed in the right ventricle. The lead is connected to the device, which is now usually implanted in the infraclavicular region, in a pocket created between the pectoral muscle and the subcutaneous tissue layer. The lead is inserted through subclavian vein cannulation or cephalic vein cutdown and allows the system to provide 3 related functions:

(a) Continuous sensing of the ventricular activity from a pair of electrodes at the lead tip;

(b) Pacing of the right ventricle from the same electrode pair, if appropriate; (c) Delivery of high-energy shock, usually between a metal coil located on the lead proximal from its tip, but still in the right ventricle, and the metal casing ("can") of the device itself.

As no atrial pacing is provided by single-chamber ICDs, these systems are typically implanted in patients who are either not likely to require frequent pacing or who are in chronic AF.

Dual-chamber ICD systems differ by the presence of an atrial lead. They provide full functionality of a dual-chamber PM and should be implanted in patients with preserved AV synchrony who are likely to require pacing for bradycardia on top of protection from ventricular arrhythmias, such as patients with sick-sinus syndrome. Occasionally, they are implanted to improve discrimination between VT and arrhythmias of supraventricular origin, for which the treatment with painful shock is often undesirable.

BiV systems are designed to provide simultaneous or near-simultaneous pacing of both ventricles, with the aim of reducing the delay between contraction of different regions of left ventricle, which is frequently observed in patients with left bundle branch block (LBBB). The resulting improvement in LV synchrony can improve cardiac output and ameliorate symptoms of heart failure in certain patients. The LV pacing is usually accomplished through a pacing lead placed in a branch of coronary sinus.

A so-called "leadless ICD" has been recently approved for clinical use in Europe and may become available in the United States in the near future. This device may provide functionality similar to a single-chamber ICD but without the need for intravenous (IV) lead placement and fluoroscopy. It consists of a single subcutaneous lead, placed in left parasternal position and tunneled to a device pocket in left lateral thorax. The device is somewhat bigger than current ICDs (69.9 cm^3) and delivers higher energy (80 J, as opposed to 30 to 40 J for most current ICDs). The system exhibited an acceptable performance in a relatively small pilot study[15]; whether the elimination of issues related to central vascular access outweighs the disadvantages related to long subcutaneous course of the lead, larger device size, and its inability to provide long-term pacing remains to be seen.

■ Evidence-based Literature for ICDs

Antiarrhythmics Versus Implantable Defibrillator (AVID) Trial

Initially, ICDs were implanted solely in patients who survived cardiac arrest or had recurrent episodes of VT resistant to pharmacological therapy. In the absence of reversible cause for the event, these

patients are at fairly high risk of recurrent event. The superiority of ICDs over treatment with amiodarone in such patients was established in the AVID trial.[16] In this study, 1016 patients, who had been resuscitated from VF, suffered VT with syncope or experienced symptomatic sustained VT in conjunction with LV dysfunction, were randomized to treatment with class III antiarrhythmic agents (mostly amiodarone) or ICD placement. At 1 year, survival was better in the ICD group (89.3 vs. 82.3%; $P < 0.02$), corresponding to absolute mortality reduction of approximately 7% per year.

However, most patients who develop cardiac arrest have not experienced prior cardiac arrest or sustained VT episode. Several risk-stratification markers have been explored to allow for primary prophylactic intervention in patients at high risk of sudden death. The risk-stratification efforts have been partially successful; the best validated parameters include low ejection fraction of the left ventricle (LVEF), inducibility of sustained VT during programmed ventricular stimulation, and, under certain circumstances, presence of nonsustained VT during electrocardiography monitoring.

Multicenter Automatic Defibrillator Implantation Trial (MADIT)

The initial data on ICD benefit for primary prophylaxis of sudden cardiac death come from clinical studies that enrolled patients with all these risk factors: the MADIT and the Multicenter Unsustained Tachycardia Trial (MUSTT). The MADIT trial[17] enrolled 196 patients with prior myocardial infarction (MI), LVEF $\leq$ 35%, and nonsustained VT, who had sustained ventricular tachyarrhythmia inducible on programmed ventricular stimulation. Moreover, inducibility of ventricular tachyarrhythmia had to persist despite attempts to suppress it with antiarrhythmic drug. These highly selected patients were randomized to ICD placement or conventional treatment. The ICD patients experienced a dramatic survival advantage, with 54% reduction in all-cause mortality rate over 27 months of mean follow-up ($P = 0.009$) and absolute survival benefit of 17.9% at 1 year.

MUSTT

The MUSTT trial[18] did not actually randomize patients with respect to ICD treatment. Instead, it was designed to assess usefulness of programmed ventricular stimulation in management of arrhythmic risk. A total of 704 patients with significant coronary artery disease, LVEF < 40%, nonsustained VT, and inducible VT during programmed ventricular stimulation were randomized to either antiarrhythmic treatment, selected on the basis of the ability of the drug to suppress

VT reinduction during programmed ventricular stimulation, or no antiarrhythmic treatment. For patients randomized to antiarrhythmic treatment in whom effective drug was not identified, ICD implantation was performed (161 of the 351 patients randomized to antiarrhythmic therapy). The primary end point was cardiac arrest or death from arrhythmia. There was a marginal difference in favor of the antiarrhythmic treatment guided by programmed ventricular stimulation, but on secondary analysis, it turned out that the benefit was limited to patients who received ICDs. The benefit in the ICD patients was impressive: relative risk for primary end point 24% ($P < 0.001$), 58% reduction in all-cause mortality ($P < 0.001$), and absolute survival benefit in the first year close to 10%. The incidence of primary end point and total mortality in patients assigned to antiarrhythmic treatment, but not receiving ICD, was actually somewhat worse than in the patients randomized to no antiarrhythmic treatment.

Taken together, the MADIT and MUSTT trials provided strong evidence that ICD implantation confers a major survival benefit in patients who have not yet experienced cardiac arrest or sustained VT, but carry multiple risk markers of arrhythmic risk. The logical next step was to investigate the use of ICDs for primary prophylaxis of sudden death in less selected patients at somewhat lower risk. The most informative trials in this category were MADIT II and Sudden Cardiac Death in Heart Failure Trial.

MADIT II

MADIT II study[19] included patients with prior MI one month or more before enrollment and LVEF $\leq$ 30%; no programmed ventricular stimulation was required. A total of 1232 patients were randomized in 3:2 ratio to either ICD placement or conventional therapy. Total mortality was the primary end point. Over the average duration of follow-up of 20 months, the reduction in mortality was 31% in the ICD group ($P = 0.016$), with absolute survival benefit of 6% in 2 years. In contrast to the trials described above, Sudden Cardiac Death in Heart Failure Trial[20] included patients with nonischemic LV dysfunction in addition to patients with coronary artery disease. A total of 2521 patients were randomized in 1:1:1 manner to ICD placement, amiodarone, or placebo on top of conventional treatment. Inclusion criteria included heart failure of New York Heart Association (NYHA) class II or III and LVEF $\leq$ 35% despite 3 months of optimal medical treatment. The primary end point was again total mortality. No significant difference in survival was detected between the placebo and amiodarone groups, but the mortality in the ICD group was reduced by 23% over 46 months of follow-up ($P = 0.007$). The absolute survival benefit associated with ICD placement was 7.2% after 5 years. The benefit associated with ICD

placement seemed to be similar in the ischemic and nonischemic patients.

CABG-Patch

In contrast, ICDs seem to be of little benefit in other patient groups. One important study that yielded negative result was CABG-Patch,[21] a trial that evaluated role of ICD shortly after CABG. In this study, 900 patients undergoing CABG, with LVEF < 36% and late potentials on signal-averaged electrocardiography before the surgery, were randomized for either ICD implantation immediately after CABG or no additional treatment. No survival benefit was detected after average follow-up of 32 months. One possible explanation for this negative result may be improvement of LVEF and decrease in arrhythmic risk after CABG in some patients. The fact that the ICDs were implanted in the operating room by cardiac surgeons immediately after CABG completion may have also played a role: the incidence of pneumonia and wound infection was significantly higher in the ICD group. Indeed, the surgical mortality was somewhat elevated in the ICD group, and the decrease in risk of sudden death among ICD patients seemed to be balanced by increase in nonsudden death.

Defibrillator in Acute Myocardial Infarction Trial (DINAMIT)

In patients with acute MI, the risk of sudden death is highest in the first months after the event and subsequently declines with time. It was therefore natural to evaluate the role of ICD during this high-risk period. The strategy of ICD implantation shortly after acute MI was addressed in 2 trials of good quality. In the DINAMIT trial,[22] patients were randomized to either ICD placement or conventional treatment 6 to 40 days after acute MI. In addition, LVEF ≤ 35% and evidence of decreased parasympathetic tone [average heart rate (HR) > 80 beats/min over 24 h or evidence of low HR variability] were required; low parasympathetic tone is a well-established marker for increased total and arrhythmic mortality in this setting. A total of 647 patients were enrolled and followed for average of 2.5 years; there was no difference in overall mortality, the study's primary end point.

Immediate Risk Stratification Improves Survival (IRIS)

The more recent IRIS trial[23] had a similar design: it randomized 898 patients 5 to 31 days after acute MI for ICD placement or conventional treatment. In addition, LVEF ≤ 40% and either increased HR (average over 24 min >90 beats/min) or nonsustained VT were required for inclusion. Again, there was no significant difference in survival after average follow-up of 37 months between the 2 groups. In

both DINAMIT and IRIS trials, the decrease in arrhythmic deaths in the ICD group was compensated by increase in nonarrhythmic mortality. It is possible that patients who die suddenly in the first few months after acute MI are also at high risk of nonsudden death because of large MI size or other factors. It certainly seems that identification of patients who are likely to benefit from ICD placement in the early postinfarction period is difficult.

■ Evidence-based Literature for CRT

The concept of CRT was developed in late 1980s, when acute decrease in LV output with right ventricular (RV) pacing was noted. This was attributed to the time delay in contraction of opposing LV walls, related to slow propagation of depolarization wave-front as the fast-conducting His-Purkinje system is bypassed. Given the similarity between the sequence of LV activation in RV pacing and LBBB, interest has developed in reducing the dyssynchrony of LV contraction in patients with LBBB and systolic heart failure, who comprise nearly one third of all heart failure patients. Studies involving catheter pacing did suggest an acute improvement in LV contractility, as did initial noncontrolled studies with BiV PMs using an LV lead placed into a coronary sinus branch.

Multisite Stimulation in Cardiomyopathies (MUSTIC)

One of the first controlled trials detecting a positive clinical effect of BiV pacing was the MUSTIC study.[24] This was a small trial with cross-over design that followed 48 patients with NYHA III heart failure despite medical treatment, LVEF < 35%, and QRS duration of > 150 ms. The patients were programmed in a way that either minimized or promoted BiV pacing. The programming was changed after 3 months and the patients were followed for another 3 months; the programming sequence was randomized. The patients were blinded to the PM programming. The distance walked in 6 minutes was the primary end point. BiV pacing significantly improved this parameter, as well as quality of life score, NYHA heart failure class, and O_2 uptake during exercise; it also diminished hospital admissions.

Multicenter InSync Randomized Clinical Evaluation

The substantially bigger Multicenter InSync Randomized Clinical Evaluation trial[25] reached a similar conclusion. In this study, 453 patients with LVEF < 35%, NYHA class III or IV, and QRS duration of at least 130 ms were implanted with a BiV PM and randomized to active (ie, BiV pacing) or inactive (no BiV pacing) programming mode in a

double-blind manner for 6 months. The distance walked in 6 minutes, NYHA class, and quality of life score were all improved significantly in the active pacing group after 6 months. Significant improvement in LVEF and hospitalization rate was also noted.

Comparison of Medical Therapy, Pacing, and Defibrillation in Heart Failure

The first large trial of CRT to evaluate its effect on overall survival was the Comparison of Medical Therapy, Pacing, and Defibrillation in Heart Failure trial,[26] in which 1520 patients with severe (NYHA III or IV) heart failure, LVEF < 35%, QRS duration of $\geq$ 120 ms, and recent heart failure admission were randomized at 1:2:2 ratio to optimal medical treatment alone or medical treatment plus implantation of BiV PM or BiV ICD. The primary end point was death or hospital admission. The patients in both BiV PM and BiV ICD groups had a significant decrease in the primary end point (relative risk reduced by 19% and 20%, respectively; $P = 0.014$ and 0.01), whereas overall survival was only significantly improved in the BiV ICD group (by 36%; $P = 0.003$). Rates of heart failure hospitalization and NYHA class were also significantly better in both resynchronization groups.

Cardiac Resynchronization-Heart Failure (CARE-HF)

The CARE-HF trial[27] also studied the effect of CRT on overall survival. It enrolled 813 patients with NYHA III or NYHA IV despite medical treatment, LVEF $\leq$ 35%, LV dilatation, and QRS duration of >150 ms (or >120 ms if echocardiographic criteria of LV dyssynchrony were present). The patients were randomized to implantation of BiV PM or medical therapy. Primary end point was again hospital admission or death; it was reduced by 37% in the BiV pacing group (39% vs. 55% incidence over mean follow-up of 29.4 mo; $P < 0.001$). Importantly, the secondary end point of total mortality was also reduced over the same period (20% vs. 30%; $P < 0.002$).

The resynchronization trials described above established the beneficial effect of LV pacing on mortality, hospital admission, heart failure class, quality of life, and parameters of LV dysfunction in a carefully selected group of patients with severe heart failure, severe LV dysfunction, and wide QRS complex. The magnitude of the survival benefit in the CARE-HF trial (4% per year) seems comparable with ICD benefit in MADIT II. Additional studies have since been performed to assess the effect of BiV pacing in subjects with only mild heart failure symptoms and in patients with mechanical LV dyssynchrony by echocardiographic criteria but with a narrow QRS complex (< 130 ms).

MADIT-CRT

The effect of CRT in patients with mild heart failure was evaluated in the MADIT-CRT trial.[28] In contrast to the earlier trials of CRT therapy, this study enrolled patients in NYHA class I (ischemic LV dysfunction) or NYHA class II (ischemic or nonischemic etiology), LVEF $\leq$ 30%, and QRS duration $\geq$ 130 ms. Patients were randomized in 3:2 ratio to implantation of either BiV ICD or ICD without LV placement. The study recruited 1820 patients, who were followed for an average duration of 2.4 years. The primary end point was a combination of death and heart failure exacerbation (defined as an episode requiring either hospital admission or outpatient IV therapy). The incidence of primary end point was 17.2% and 25.3% in the BiV ICD and ICD-only groups, respectively (relative reduction of 34%; $P = 0.001$). Mortality did not differ significantly between the groups. In this study, CRT also improved LVEF and LV size compared with ICD-only patients.

Resynchronization/Defibrillation for Ambulatory Heart Failure Trial

Similar results were reported by the Resynchronization/Defibrillation for Ambulatory Heart Failure Trial.[29] Initially, this trial recruited patients with heart failure class NYHA II and NYHA III, although the recruitment was later restricted to NYHA II subjects of either ischemic or nonischemic etiology. LVEF $\leq$ 30% and QRS duration $\geq$ 120 ms were also required. Patients were randomized to BiV ICD or ICD alone. Death or heart failure hospitalization was the primary end point. The trial randomized 1798 patients who were followed for mean of 40 months. CRT reduced the primary end point from 40.3% to 33.2% (relative risk reduction of 25%; $P < 0.001$). Total mortality was decreased by BiV pacing (20.8% vs. 26.1%; $P = 0.003$). When the analysis was limited to the 1438 NYHA II patients, the decrease in total mortality remained significant (15.5% vs. 21.1%; $P = 0.006$).

Resynchronization Therapy in Patients With Heart Failure and Narrow QRS (RethinQ)

It thus seems that in the presence of severe LV dysfunction and wide QRS complex, CRT has beneficial effect on incidence of heart failure exacerbations and probably also on total mortality, even in patients with only mild heart failure symptoms.

The role of CRT in patients with narrow QRS complex was addressed in the RethinQ study.[30] A total of 172 patients with narrow QRS (< 130 ms), but with echocardiographic signs of LV dyssynchrony were enrolled. LVEF < 35% and NYHA III status was also required. All

patients were implanted with BiV ICD and randomized for BiV pacing or no BiV pacing. Proportion of patients with improvement in O_2 consumption (by 1 mL/kg/min) after 6 months was the primary end point. With respect to this measure, there was no significant difference between the 2 groups. In patients with QRS duration 120 to 130 ms, O_2 consumption did increase significantly, but there was no difference in the patients with QRS duration <120 ms. RethinQ was a relatively small study, but it did not suggest benefit of CRT in the absence QRS prolongation.

■ Anesthesia-related Aspects Specific to an EP Laboratory

Patient Population

Patients presenting for EP procedures range from essentially healthy young patients with structurally normal heart, undergoing RFA of AV nodal reentry tachycardia or accessory pathway, to hemodynamically unstable patients with end-stage heart failure and multiple comorbidities who require VT ablation because of frequent ICD discharges. A patient presenting for EP procedures could be in generally good clinical shape but unfortunately much more likely will have a rich past medical history with multiple comorbidities, including poor functional status (as one of the indication criteria for implantation of ICD), cardiomyopathy, congestive heart failure, coronary artery disease, hypertension, valvular heart disease as well as cerebrovascular disease, diabetes mellitus, hyperlipidemia, and a plethora of other conditions. Of particular importance is a presence of respiratory disease that could be worsened during prolonged supine position, namely obstructive sleep apnea, chronic obstructive pulmonary disease, and chronic O_2 use. Patients with gastroesophageal reflux disease may be prone to silent aspiration under sedation. Patients with the most severe forms of ventricular arrhythmias may be even supported by extracorporeal devices including intra-aortic balloon pumps or centrifugal pumps on top of inotropic support for the failing heart, presenting a true challenge for the off-site anesthesiologist.

Environment

EP laboratory is usually located off-site in a remote location from operating rooms. This may limit an immediate access to ancillary anesthesia support services, including satellite pharmacy, or difficulties in obtaining laboratory results, advanced airway equipment, or blood products. A thorough anesthetic plan including a sequence of actions for an emergency should be devised and discussed with the EP laboratory personnel.

The EP laboratory itself is usually arranged in 2 sections, one with a "control station" and the patient area with operating table and a dedicated fluoroscopy system that resembles in its shape C-arm imaging device. Sophisticated mapping systems used in many EP laboratories facilitate the procedure but further impede anesthesiologists' access to a patient. Adjacent to the head of the patient is usually the anesthesia machine and a cart containing drugs and anesthesia equipment. It should be noted that some EP procedures require dimmed lights to allow visualizing the anatomy on the remote screens. This could further hamper the clinical evaluation of the patient when the procedure is underway. An excellent review and more detailed description of the EP laboratory could be found elsewhere.[1]

General Preparation and Considerations

Unfortunately, anesthesia providers may not have an unrestricted access to the patient's head at all times. Arms are usually tucked by the body side and restrained, providing limited access to the sites of IV infusions. All lines should be confirmed to be fully functional before a procedure is started given a limited opportunity to rectify mishaps without interrupting the procedure. Infusion pumps with sedative and/or vasoactive drugs should be primed and connected to available IV ports and set to go in case they are needed. Patients' arms, legs, and pressure points should be comfortably padded. A heating blanket can be used to maintain patient's temperature during prolonged procedures under general anesthesia. A placement of a Foley catheter should be weighed carefully, balancing the risks of an infection, discomfort of its placement in a conscious placement, versus the need for fluid balance monitoring and discomfort with overfilled bladder. The anesthesiologist should be monitoring the amount of additional fluids administered by the EP team and adjust the maintenance fluids appropriately.

Type of Anesthesia

The majority of EP procedures could be accomplished either under sedation or under general anesthesia. The plan should be discussed and agreed upon between all parties involved, namely the patient, the EP physician, and the anesthesia care team. It should be recognized that sedation and analgesia comprise a continuum of states ranging from minimal sedation (anxiolysis) through general anesthesia.[31] The term monitored anesthesia care does not describe the continuum of depth of sedation, rather it describes "a specific anesthesia service in which an anesthesiologist has been requested to participate in the care of a patient undergoing a diagnostic or therapeutic procedure."

In the aforementioned survey of academic EP centers, most providers favored sedation/monitored anesthesia care over general

anesthesia except for epicardial VT ablation, for which general anesthesia was preferred. Although the reasons for a particular choice were not directly explored in the study, we can hypothesize that there are 3 major reasons for this choice: first, anesthesia providers are not available at all times to staff the EP laboratory procedures, leaving the choice of sedation as the inevitable only option; second, patients recover sooner from sedation than from general anesthesia and generally do not require high-acuity care and could be scheduled as a same-day or short-stay surgery. This may facilitate the turn-over rate and productivity and decrease the cost to the patient. Third, personal experience of the EP physician with the given procedure, combined with a detailed knowledge of a particular patient, also play a role in the decision-making process regarding the type of anesthesia. For example, minimizing or avoidance of sedation in particular patient may help to facilitate the induction of ectopy originating from the RV outflow tract that could not be induced in anesthetized patients. However, there is a paucity of data that would support that approach, and this rationale should not be used to eliminate anesthesia providers from EP procedures.

The involvement of nonanesthesia personnel has been a matter of recent controversies. American Society of Anesthesiologists (ASA) acknowledged that Medicare regulations permit some nonanesthesiologists to administer or supervise the administration of deep sedation and provided an advisory to serve as a potential guide to its members who may be called upon by administrators or others to provide input in this process. Because of the significant risk that patients who receive deep sedation may enter a state of general anesthesia, privileges to administer deep sedation should be granted only to practitioners who are qualified to administer general anesthesia or to appropriately supervised anesthesia professionals. ASA suggested a framework that could be used for setting up training and granting privileges for administration of moderate or deep sedation to practitioners or nonanesthesia providers.[32,33] However, ASA believes that anesthesiologist participation in all deep sedation is the best means to achieve the safest care.

The importance of the issue is documented by several studies focused on the current practice of sedation for EP procedures. The results show that deep sedation is often administered by nonanesthesia personnel,[34,35] and advanced airway interventions suggesting progressing to general anesthesia are frequently required to rescue the patient from undesired deeper levels of sedation, including general anesthesia.[34,36] A significant proportion of patients scheduled for sedation needed to be converted to general anesthesia, and 40% of patients required airway intervention.[36] Airway emergencies arise even in cases when experienced cardiac anesthesiologist is responsible for the patient care—not because the sedation spins out of control, but because of

unpredicted complications arising during the procedure, for example sudden worsening of congestive heart failure or procedure-related complications.[37] Advanced airway instrumentation may be especially risky in an anticoagulated patient under deep sedation with an unprotected airway. Thus, a comprehensive training and a close surveillance of nonanesthesia providers administering sedation is warranted in cases when anesthesia team is not providing the care.

It should also be noted that an ICD needs to be tested for its proper function after the implantation. This often involves induction of VT and subsequent termination by an internal defibrillation. This is usually accomplished under deep sedation or even general anesthesia to eliminate pain and/or stress. Deepening of preexisting sedation may be accompanied by respiratory depression that may require transient ventilation. Hemodynamically, this testing is usually well tolerated. In severely hemodynamically compromised patients or in patients with AF and transiently discontinued anticoagulation, defibrillation threshold testing is usually not performed.

Very few studies directly compared the short-term and long-term outcome of a particular EP procedure performed under sedation versus general anesthesia, leaving the choice between these 2 to individual preferences of the providers and the patient.

Perhaps the most studied procedures were catheter ablations that usually last several hours and require stable field. Uncontrolled patient respirations with variable diaphragmatic motions could interfere with precise positioning of the catheter during mapping and ablations, respectively. The ablation itself can cause discomfort to the patient. A recent randomized study in 257 patients undergoing RFA for AF showed that general anesthesia was associated with a higher success rate during a single procedure and seemed to reduce the prevalence of pulmonary vein reconnection when repeat ablation was needed. The group subjected to general anesthesia had also shorter fluoroscopy time and procedure time.[38] In contrast, general anesthesia was shown to have an increased risk of esophageal injury caused by RFA as assessed by capsule endoscopy. Monitoring of esophageal temperature and limiting the duration of ablation pulse to <20 seconds, along with other methods, may reduce the risk or prevent this complication of RFA.[39,40]

The use of high-frequency jet ventilation (HFJV) during general anesthesia represents another method of creating a motionless field to facilitate the procedure. At our institution, HFJV is routinely implemented during EP procedures especially when a stable field needs to be established. To evaluate the efficacy of the method in a randomized manner, Goode and colleagues compared 36 patients undergoing RFA in posterior left atrium (LA) that were ventilated with HFJV, with a cohort of patient managed with conventional intermittent positive pressure ventilation (IPPV). Fewer ablation lesions were required in the

HFJV group because of fewer incidences of ablation electrode dislodgment, resulting in significantly decreased procedure time. Direct comparison in additional 10 patients demonstrated that HFJV produced less variation in LA volume, pressure, pulmonary vein blood flow velocity, and posterior LA position than IPPV.[41] This is in concert with previous studies in healthy adults that showed that, in comparison with IPPV, HFJV significantly decreases pulmonary arterial pressure and LA pressure, resulting in significant increases in cardiac output and ejection fraction.[42] Previous studies demonstrated that even critically ill patients with circulatory shock and acute respiratory failure have a more favorable hemodynamic profile during HFJV than during IPPV.[43] In experienced hands, HFJV remains an unsurpassed method to stabilize the field. Most studies documented favorable hemodynamic effects. If considered for the individual case, necessary preparation steps must be made to transition the patient from a conventional inhalational anesthesia during the set-up phase of the EP procedure to total IV anesthesia with neuromuscular blockade before initiating HFJV for the phase when maximum catheter stability is required. The transition from IPPV to HFJV administered using preexisting endotracheal tube is then seamless. We generally use a combination of short-acting anesthetics including remifentanil, propofol, and dexmedetomidine, with neuromuscular blockade provided by cisatracurium. This combination allows easy titration of the drugs to the need of the patient and prevents undesired accumulation of drugs seen with other drugs that have longer context-sensitive half-time index. After a completion of the ablation, anesthesia is usually switched back to inhalational, and IPPV is restarted. The use of short-acting drugs allows targeting the anesthesia toward extubation at the end of the procedure in eligible patients.

Anesthetics

The role of individual drugs on the effect of EP procedure has been largely understudied. A combination of multiple drugs is generally used in a single patient, making strong conclusions difficult. IV general anesthetics including thiopental, propofol, ketamine, or etomidate have been used for induction and/or maintenance of general anesthesia or for maintaining desired level of sedation. Anxiolytics and sedatives including midazolam and other benzodiazepines are used to facilitate sedation along with opioids, namely fentanyl and its derivatives. Nitrous oxide, isoflurane, sevoflurane, and desflurane are volatile anesthetics used in maintenance of general anesthesia. Dexmedetomidine, an ultrashort-acting highly selective α-2 agonist, have been added lately in the anesthesiologists' armamentarium as a novel agent producing sedation and potentiating the effects of other agents, thus allowing to decrease their dose while preserve the respiratory response to increased carbon

dioxide better than other agents. These characteristics made the drug especially useful in sedation cases and in general anesthesia. Its use seems to be appealing namely in obstructive sleep apnea patients who would not tolerate deep sedation with other agents blunting their carbon dioxide responsiveness.[44]

The choice of anesthesia and particular anesthetics should be tailored to the procedure. It is prudent to choose a drug or—more likely—a combination of drugs that do not interfere with the goals EP procedure, especially mapping of tachyarrhythmias. The same principle applies to the choice of preoperative medications that may impede the procedure, namely antiarrhythmics. Neither propofol nor isoflurane anesthesia altered sinoatrial node or AV node function in pediatric patients undergoing RFA.[45] Most tachyarrhythmias remained inducible under propofol anesthesia, except for ectopic atrial tachycardia in children that were not inducible in 4 out of 7 children and the procedure could not be accomplished.[35]

In patients with Wolff-Parkinson-White syndrome undergoing surgical ablation, sufentanil-lorazepam had no clinically significant effect on the EP expression of the accessory pathway. Of the volatile agents, enflurane most, isoflurane next, and halothane least increased refractoriness within the accessory and AV pathways. Therefore, administration of these volatile agents during ablative procedures may confound interpretation of postablative studies used to determine the success of ablation treatment. Conversely, in patients with preexcitation syndrome requiring general anesthesia for nonablative procedures, volatile agents may reduce the incidence of perioperative tachyarrhythmias because of their effects on refractoriness.[46]

Several studies explored the feasibility of dexmedetomidine in children undergoing EP procedures. Hammer and colleagues found that dexmedetomidine significantly depressed sinus and AV nodal function in pediatric patients, resulting in bradycardia and increased arterial blood pressure. They suggested that the use of dexmedetomidine may not be desirable during EP studies.[47] In our experience, cautious use of dexmedetomidine in adult patients did not induce such a deleterious hemodynamic changes that would interfere with EP procedures. Incidental decrease in HR is usually hemodynamically tolerated. The ultra-short half-life of dexmedetomidine enables reconsideration of the anesthetic plan should a major hemodynamic disturbance occur.

■ Summary

Recent advances in EP enabled significant improvement in the care for cardiac patient. Implantation of PMs or ICDs, CRT, and RFA of tachyarrhythmias represent EP procedures that significantly improve

quality of life and outcome in selected patient populations. The complexity of the procedure and the off-site EP laboratory environment create a new, challenging scenario for the anesthesiologist. A complex medical history and a current physical status of a patient presenting for a procedure must be carefully weighed in when discussing the options of anesthesia care. The roles of individual anesthetics and anesthesia techniques need to be further evaluated to facilitate the procedure and optimize patient care.

The authors have no conflicts of interest to disclose.

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