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Journal

Journal of cardiothoracic anesthesia, 28(1)

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Publication Date

2014-02-01

DOI

10.1053/j.jvca.2013.08.019

Peer reviewed

Published in final edited form as:
J Cardiothorac Vasc Anesth. 2014 February ; 28(1): 69–75. doi:10.1053/j.jvca.2013.08.019.

Anesthetic Considerations for Thoracoscopic Sympathetic Ganglionectomy to Treat Ventricular Tachycardia Storm: A Single-Center Experience

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Abstract

Objective—The aim of this study was to determine the pertinent anesthetic considerations for patients undergoing surgical sympathectomy for electrical storm (incessant ventricular tachycardia (VT) refractory to traditional therapies).

Design—This is a retrospective review of a prospective database.

Setting—This single-center study took place in a university hospital setting.

Participants—Twenty-six patients were enrolled.

Interventions—Fifteen patients underwent left-sided sympathectomy, whereas 11 patients underwent bilateral sympathectomy.

Measurements and Main Results—Anesthetic management of these patients was quite complex, requiring invasive monitoring, transesophageal echocardiography, one-lung ventilation, programming of cardiac rhythm management devices, and titration of vasoactive medications. Paired t test of hemodynamic data before, during, and after surgery showed no significant difference between preoperative and postoperative blood pressure values, regardless of whether the patient underwent unilateral or bilateral sympathectomy. Eight patients remained free of VT, three patients responded well to titration of oral medications, and one patient required 2 radiofrequency ablations after sympathectomy to control his VT. Three patients continued to have VT episodes, although reduced in frequency compared with before the procedure. Four patients were lost to followup. Overall, five patients within the cohort died within 30 days of the

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procedure. No patients developed any anesthetic complications or Horner's syndrome. The overall perioperative mortality (within the first 7 days of the procedure) was 2 of 26, or 7.7%.

Conclusions—The anesthetic management of patients undergoing surgical sympathectomy for electrical storm can be quite complex, because these patients often present in a moribund and emergent state and cannot be optimized using current ACC/AHA guidelines. Expertise in invasive monitoring, transesophageal echocardiography, one-lung ventilation, cardiac rhythm device management, and pressor management is crucial for optimal anesthetic care.

Keywords

sympathectomy; stellate ganglionectomy; ventricular tachycardia; electrical storm

DESPITE MULTIPLE RECENT advances in treatment, ventricular arrhythmias leading to sudden cardiac death remains the leading cause of death in the United States, even eclipsing the overall mortality of all cancers combined.[1,2] "Electrical storm" refers to ventricular arrhythmias refractory to medical treatment, for which the need for electrical therapy may range from twice in a 24-hour period to nearly continuous shocks.[3] Such arrhythmias classically are treated with a combination of antiarrhythmic drugs, defibrillation, and/or rapid pacing. However, class I antiarrhythmics often fail, and amiodarone may take days to achieve sufficient rhythm control[4]—a luxury of time not afforded in the case of electrical storm. Although the implantable cardioverter/defibrillator (ICD) remains the standard in treatment for recurrent ventricular tachycardia (VT), it is not curative therapy, and the risk of recurrent arrhythmia remains unaffected. Furthermore, the occurrence of frequent ICD shocks has been tied to increased mortality and decreased quality of life.[5] Recently, electrical storm refractory to medical and electrical therapies has been treated successfully via catheter ablation,[4,6,7] although the failure rate of this approach remains high, thus necessitating alternative treatment. Stellate ganglionectomy has been introduced as a definitive surgical approach to ameliorate sympathetically mediated VT in patients refractory to conventional therapies, and its use is gaining momentum.

Although most patients in electrical storm have a low immediate mortality,[8] patients who are candidates for this approach (having typically failed pharmacologic catheter-based interventions with persistent life-threatening arrhythmias) universally present on both an emergent basis and often moribund cardiac state. Therefore, perioperative optimization of these patients cannot be undertaken using the American College of Cardiology/American Heart Association (ACC/AHA) guidelines and, thus, pose a unique challenge to the perioperative care team.

In this single-center study, the authors retrospectively reviewed a prospectively collected database to determine anesthetic considerations in managing patients undergoing sympathetic ganglionectomy via video-assisted thoracic surgery (VATS) for treatment of electrical storm. The challenges in perioperative care and surgical, electrophysiologic, and anesthetic management for these complex patients are described, and clinical outcome measures are reviewed to determine if these patients could be ushered safely through the perioperative period despite the significant challenges posed by patient comorbidities and surgical and anesthetic complexity.

OPERATIVE CASE CONSIDERATIONS

Twenty-six consecutive patients undergoing bilateral or unilateral sympathetic ganglionectomy via the VATS approach were recruited for this study. Arterial access was obtained in all patients before induction of anesthesia. Induction of general anesthesia was achieved using titrated doses of lidocaine (1-1.5 mg/kg), fentanyl (1-3 ug/kg), and etomidate (0.1-0.2 mg/kg) and/or propofol (1-2 mg/kg). In general, fentanyl and etomidate were preferred for patients with significantly depressed ejection fraction (EF); otherwise, fentanyl and propofol were preferred. Neuromuscular blockade was maintained by administration of a nondepolarizing agent (rocuronium, vecuronium, or cisatracurium). All patients were intubated via direct laryngoscopy and placement of a left-sided double-lumen tube for single-lung ventilation. Patients without pre-existing central venous access received a 9-French Cordis introducer in anticipation of either possible initiation of inotropic/vasopressor support and/or pulmonary artery catheterization.

Anesthesia was maintained using potent inhaled anesthetic agents (0.7-1.3 minimum alveolar concentration) in 100% oxygen. Hemodynamic monitoring consisted of standard monitors as described by the American Society of Anesthesiologists with the addition of invasive arterial monitoring. Because most patients had implanted devices and were, thus, at risk of electromagnetic interference due to electrocautery, device interrogation and reprogramming were performed after the patients were anesthetized. Reprogramming consisted of disabling electronic antitachycardia therapies after placement of cutaneous defibrillator pads, disabling rate responsiveness, if present, and adjusting electronic bradycardia therapies on a patient-specific basis. Adhesive defibrillator pads were placed in the anterior-posterior orientation, so as not to interfere with the surgical field and to minimize the risk of damage to the patients' electronic pacing systems. Transesophageal echocardiography (TEE) was performed in all patients to assess cardiac function and guide intraoperative hemodynamic management.

Patients then were placed in the lateral decubitus position with the operative side up. For patients undergoing concurrent, bilateral procedures, the left was performed first. Upon completion of the left-sided VATS, patients then were repositioned toward the opposite side for performance of the contralateral VATS.

At the end of the surgical procedure, patients who were not intubated before surgery were assessed for standard extubation criteria. Once criteria were met, these patients were extubated. Patients then were transferred to the postanesthesia care unit (PACU) or intensive care unit (ICU), depending on level of acuity.

METHODS

After institutional review board (IRB) approval, volunteers within the Department of Anesthesiology collected a prospective database of consecutive patients meeting inclusion criteria of undergoing unilateral or bilateral sympathetic ganglionectomy via VATS for treatment of electrical storm due to any cause. Given the observational nature of the study,

there were no exclusion criteria. The authors then retrospectively reviewed the data collected from this cohort including all pertinent preoperative , intraoperative, and postoperative data.

A total of 26 patients undergoing thoracoscopic sympathectomy for ventricular tachycardia storm from April 2009 through December 2011 were enrolled. Data were collected pertaining to patient age, gender, ASA physical status, preoperative left ventricular EF, cause and prior treatment of arrhythmia, and presence of an implanted cardioverter/ defibrillator.

Data were collected pertaining to type of procedure (unilateral versus bilateral sympathectomy), surgical time, anesthesia time, estimated blood loss (EBL), intravenous fluid administration, transfusion, and inotropic and vasopressor support. Any intraoperative occurrence of significant arrhythmia or hypoxemia also was noted, along with measures used to maintain hemodynamic stability. Performance of intraoperative TEE was deemed significant, and any abnormalities were recorded in the database. Finally, data were collected regarding successful extubation at the end of surgery versus requirement of prolonged ventilatory support.

Postoperative disposition was noted, as was length of ICU stay. Data were collected pertaining to residual arrhythmia and subsequent treatment, as well as mortality.

Hemodynamic data were gathered for each patient, including lowest blood pressures (1) in the final 24 hours before surgery, (2) intra-operatively before sympathectomy, (3) intraoperatively after sympathectomy, and (4) in the first 24 hours after surgery, to assess hemodynamic effects of sympathectomy.

Significant complications requiring further intervention (such as chest tube placement or dialysis) were recorded. Change in EF after sympathectomy was noted, as were any signs or symptoms of development of Horner’s syndrome.

Numeric values in the database were analyzed for proportions and expressed as percentage, median, and range. Paired t test was used to determine significant changes between preoperative and postoperative values.

RESULTS

A total of 26 patients underwent thoracoscopic sympathectomy. Table 1 shows patient demographic data. Mean age was 58 ± 11 years. Twenty-four patients (92%) were male, and all patients were deemed ASA class 4E. Mean preoperative EF was 31% ± 14%, with a median of 25% and a range of 15% to 59%. The primary cause of VT was predominantly ischemic cardiomyopathy (23%) or nonischemic dilated cardiomyopathy (54%). Most patients (92%) had an ICD. In all patients, the indication for thoracoscopic sympathectomy was recurrent VT requiring multiple ICD shocks despite maximal medical management and failed catheter ablation.

Table 2 shows relevant intraoperative clinical information. Eleven of 26 patients underwent bilateral sympathectomy (42%). Median surgical time for the procedure was 164 minutes,

with a range of 91 to 296 minutes, and median anesthesia time was 229 minutes with a range of 132 to 358 minutes. Median estimated blood loss (EBL) was 50 mL with a range of 0 to 400 mL, and median IV fluid (IVF) administered was 1,100 mL with a range of 150 to 2,500 mL. No patients required blood transfusion. In this patient population, 4 patients (15%) required inotropic support before surgery, consisting primarily of norepinephrine, epinephrine, milrinone, dopamine, or vasopressin. During the procedure, however, 50% of patients required inotropic or vasopressor support, consisting of epinephrine, vasopressin, norepinephrine, and/or dopamine. Seven patients (27%) required postoperative inotropic support (within the first 24 hours of surgery), consisting of vasopressin, dopamine, epinephrine, phenylephrine, norepinephrine, and milrinone. One patient required extracorporeal membrane oxygenation 5 days before sympathectomy because of severe hemodynamic instability after attempted catheter-based radio-frequency ablation for VT. Three patients required intraaortic balloon pump support for severe hemodynamic decompensation before sympathectomy, and one required an intra-aortic balloon pump because of hemodynamic instability after attempted VT ablation after sympathectomy already had taken place.

Three of 26 patients had sustained VT during the procedure, with one patient suffering circulatory arrest requiring advanced cardiac life support measures. This patient recovered but continued to have nonsustained VT; however, the sympathectomy proceeded without incident. One patient was unable to tolerate one-lung ventilation; therefore, right-sided sympathectomy was aborted. TEE exams in most patients resulted in no new findings when compared with preoperative exams, although grading of valvular lesions was noted to vary slightly. One patient had a flail posterior mitral valve leaflet and another had a diffusely hypokinetic left ventricle, but these were not changed from previous echocardiograms. Twenty of 26 patients (77%) were extubated in the OR. The six who remained intubated had arrived in the operating room intubated and, therefore, were not extubated after the procedure. Paired *t* test of hemodynamic data before, during, and after surgery found no significant difference between preoperative and postoperative blood pressure values, regardless of whether the patient underwent unilateral or bilateral sympathectomy (Fig 1).

All patients were admitted to the ICU postoperatively, with or without recovery in the PACU. Postoperative ICU stay averaged 7 ± 7 days. Two patients who previously had been listed for orthotopic heart transplantation due to recurrent episodes of VT received their transplants after sympathectomy. Four patients developed multisystem organ failure with subsequent withdrawal of care before end-of-life due to residual, intractable VT. One patient developed a right-sided pneumothorax, pneumonia, and, subsequently, septic shock; he ultimately suffered pulseless electrical activity arrest and passed away 10 days after sympathectomy. Three patients with continued VT responded well to titration of oral medications, including carvedilol and amiodarone. One patient required two radiofrequency ablations postoperatively to control his VT. Three patients continued to have episodes of VT, although reduced in number after sympathectomy. Eight patients had no further episodes of VT, and four patients were lost to follow-up.

In terms of complications, one patient developed a pneumothorax intraoperatively requiring chest drain insertion because of a difficult surgical dissection, and one patient developed a

hemothorax on postoperative day 1. Another patient, as mentioned previously, developed a right-sided pneumothorax on postoperative day 9. One patient developed a left hemothorax before sympathectomy due to a supratherapeutic activated partial thromboplastin time while on a heparin drip. Five patients had chronic renal insufficiency, and four patients had preoperative acute renal failure secondary to periods of hypoperfusion during episodes of VT. There was no exacerbation of renal insufficiency post-sympathectomy. No patients in this study developed Horner's syndrome, a theoretical complication of the surgical procedure. Although there was no 24-hour mortality, two patients died within 7 days (8%) and three within 30 days (12%). Of note, no anesthetic complications were sustained in the 26 patients in this study.

DISCUSSION

This observational study describes the unique perioperative and anesthetic concerns for patients undergoing unilateral or bilateral stellate ganglionectomy. To the authors' knowledge, this is the first study to explore what encompasses safe anesthetic practice for these extremely challenging patients undergoing such a complex surgical procedure.

Surgical Considerations

François Franck first suggested the utility of surgical sympathectomy in treating angina in 1899.[9] However, successful surgical removal of the stellate ganglion for cardiac indications was not reported until 1916, when the ganglion, as well as the last cervical and first thoracic ganglia, were removed for the treatment of severe angina and recurrent arrhythmias, both of which resolved after surgery.[10] Subsequently, in 1929, LeRiche and Fontaine performed bilateral cardiac sympathectomy to treat persistent supraventricular tachycardia.[11] In the decades immediately following, stellate ganglionectomy continued to be performed for indications such as severe angina and abnormalities of the sympathetic nervous system such as Raynaud's disease.[12] However, the advent of beta-adrenergic blocking drugs subsequently allowed physicians to provide patients with a "medical sympathectomy" without the risk of surgical complications.[13,14]

In the 1950s, studies revealed that stimulation of sympathetic structures in the setting of coronary ischemia led to an increase in ventricular arrhythmias.[15] The first successful human sympathectomy for treatment of ventricular arrhythmia was carried out in 1961 by Estes and Izlar, although initial enthusiasm over this procedure soon was attenuated as subsequent studies reported inconsistent success rates.[16]

Because sympathetic innervation of the heart has been shown to play a key role in production of fatal arrhythmias, surgical sympathectomy increasingly has gained popularity as a treatment modality. Even so, this procedure often is considered only as a final option after a patient has failed medical treatment and catheter-based ablation. Surgical stellate ganglionectomy differs from blockade via injection, because in surgical excision, only the lower portion of the ganglion is removed, along with thoracic sympathetic ganglia 2-4 (the "cardiac accelerator" fibers); in contrast, injection of local anesthetic or alcohol eradicates the stellate ganglion in its entirety. As such, the physiologic effects can differ significantly.

Currently, surgical stellate ganglionectomy is approached via VATS. Patients are placed in the lateral decubitus position, with the operative side up. The chest cavity is entered through 3 separate ports: The first in the mid-chest line in the seventh intercostal space, the second in the posterior chest line in the seventh intercostal space, and the third in the anterior chest line in the fourth intercostal space. The first through fourth ribs are identified, as well as the subclavian artery and vein and the thoracic aorta. The sympathetic ganglia and chain then are identified and dissected, taking care to avoid injury to the intercostal vessels. The rami communicantes of ganglia 1 through 4 are cut and the chain divided caudally below the fourth rib. After hemostasis is achieved, a chest tube is inserted through one of the ports and the remaining ports are sutured closed.

Electrophysiology Considerations

Ventricular arrhythmias may be due to varying etiologies, including underlying ischemia, severe congestive heart failure, myocarditis, or congenital abnormalities.[15] However, it is known that ventricular arrhythmias generally are sensitive to, and can be triggered by, sympathetic stimulation.[17] The left cardiac sympathetic system is recognized as predominantly regulating arrhythmogenic potential, whereas the right-sided system primarily regulates heart rate.[18-20] Left sympathectomy is, therefore, usually the initial surgery of choice, although staged or concurrent right-sided sympathectomy also can be performed when the etiology of the ventricular arrhythmia is deemed to be more complex (a synergistic interaction between arrhythmogenic stimulation and heart rate, for example) or particularly sensitive to sympathetic stimulation. This differs from recommendations for ganglionic blockade, for which bilateral blockade absolutely is contraindicated because of concerns for fully eradicating sympathetic tone to the heart. Sympathectomy has indeed been shown to downregulate adrenergic neurotransmitters, such as norepinephrine, epinephrine, and dopamine.[21,22] Further studies have suggested that surgical sympathectomy can lead to prolongation of ventricular refractory time and shortening of the action potential duration.[21,23] Multiple studies and case reports have described the use of surgical sympathectomy for treatment of VT due to prolonged QT syndrome as well as catecholaminergic polymorphic ventricular tachycardia, mediated via decreased intra-cardiac norepinephrine release.[17,24-26]

Besides surgical sympathectomy, which often is considered as a last resort, strategies in management of electrical storm include pharmacologic sympathetic blockade, antiarrhythmic drugs, optimization of cardiac rhythm device programming, management of reversible causes such as electrolyte imbalance, catheter ablation, and possible deep sedation or general anesthesia. In addition, thoracic epidural infusion of local anesthetic may provide a temporizing measure to treat intractable arrhythmias.[27,28]

Anesthetic Considerations

Although data have indicated that immediate mortality in all patients suffering electrical storm is low,[8] patients considered for surgical sympathetic ganglionectomy typically have failed aggressive medical and catheter-based ablative therapies with persistent, life-threatening ventricular arrhythmias and, thus, present with a higher immediate risk of sudden cardiac death than most patients with electrical storm. Indeed, even when admitted

from home, these patients immediately were diagnosed by their physician as suffering intractable ventricular arrhythmias and emergently admitted for surgical intervention. As a result, these patients universally fell into the American Society of Anesthesiologists (ASA) physical status 4E category at minimum, with Goldman Cardiac Risk Index (CRI) scores of 3 or 4 (quoted perioperative mortality of 11 and 22%, respectively).[29] While successful surgical termination of the arrhythmia ostensibly decreases the short-term, arrhythmia-related mortality of these patients, those fitting this ASA physical status and CRI profile still are appreciated to have perioperative mortality near 20% for any surgery.[30,31] Therefore, as VATS for sympathetic ganglionectomy becomes more commonplace, it would behoove anesthesiologists to define safe practice for perioperative management of these cases.

To the authors' knowledge, this is the largest database of patients undergoing sympathetic ganglionectomy via VATS approach and is the first report to specifically delineate different anesthetic considerations and approaches for these very difficult patients. In addition, the authors present their unique demographic and intraoperative characteristics, both of which have not been reported previously in such a large-scale study. Because all of these patients presented for surgery in a moribund cardiac state, all received arterial catheters to assist in hemodynamic monitoring and management, as well as frequent blood sampling for management of single-lung ventilation. Intraoperative TEE was performed to assess baseline cardiac function as well as to rule out unanticipated structural pathology (eg, valvular disease, thrombus, intra-cardiac shunt). Considering that all of these patients had abnormal hearts, and many had depressed EF, it is the authors' opinion that TEE is a clearly indicated monitoring modality to elucidate causes of intraoperative hemodynamic instability.

Most of these patients had implanted devices, all of which required reprogramming to prevent electromagnetic interference leading to inappropriate antitachycardia therapies. In this center, perioperative electrophysiology services are provided by cardiovascular and thoracic anesthesiologists; and, therefore, most cases were managed by those personnel. For the time period during which antitachycardia therapies were disabled, all patients were monitored, and adhesive defibrillator pads were applied. Once electrocautery was no longer needed, device reinterrogation was performed to rule out major changes in capture or sensing thresholds, as well as to reinitiate anti-tachycardia therapies and optimize antibrachycardia therapies for the immediate postoperative period.

The major challenges in anesthetic management of these patients center around the risk of cardiac events (hemodynamic instability, arrhythmia, and cardiac arrest), as well as management of single-lung ventilation and cardiopulmonary physiologic interaction. Given that these cases require single-lung ventilation in patients with low cardiac output, interventions geared toward maintaining arterial oxygen saturation were undertaken frequently by the anesthesiologist, such as recruitment maneuvers, CPAP to the operative lung, PEEP to the non-operative lung, and in one case, aborting the procedure due to persistent hypoxia on single-lung ventilation. Also, as a large number of the patients requiring inotropic and vasopressor support, an advanced understanding of the indications and use of these drugs on the part of the anesthesiologist is crucial for optimal perioperative care. Furthermore, the ability and willingness to initiate ACLS protocols are absolutely necessary to maximize perioperative survival in these patients. In this study, the two

perioperative mortalities were due to intractable VT progressing to death despite appropriate management, while other patients appropriately treated for hemodynamic instability survived past the postoperative period. Indeed, the overall perioperative mortality (2 of 26) was far less than the quoted mortality for patients fitting this demographic. The other mortalities occurring within the first 30 days were associated with unsuccessful termination of arrhythmias and resulting multiorgan system failure, as opposed to any anesthetic-related cause.

Two theoretical risks of this surgical procedure include Horner's syndrome (due to loss of sympathetic tone from the stellate ganglion) and loss of myocardial contractility (due to decreased sympathetic innervation of the heart). In this study, no patients reported development of symptoms suggesting Horner's syndrome. Although there was an overall trend toward decreased EF after surgery, this finding was not statistically significant. Indeed, several patients actually had an increase in EF after surgical sympathectomy.

Also, with decreased sympathetic outflow to the heart, theoretical concern exists for precipitous hemodynamic collapse, especially in the setting of bilateral sympathectomy. Although 50% of patients required some type of inotropic support, the data showed no statistically significant change in hemodynamics either intraoperatively or within the first 24 hours after surgical sympathectomy compared with preoperative values for either unilateral or bilateral sympathectomy. Furthermore, bilateral sympathectomy did not portend greater likelihood of requiring inotropic or vasopressor support when compared with unilateral sympathectomy. However, because 50% of patients required intraoperative initiation of such support, it is reasonable to infer that expertise with and willingness to administer these medications are necessary to maintain perioperative hemodynamic stability.

Limitations

This study was performed in a single center, and, thus, the care these patients received was contingent on the practice and expertise of the group in this center. These results may not translate to centers with different areas of specialization.

CONCLUSION

Presentation of the patient in electrical storm for thoracoscopic sympathectomy poses many unique challenges to the anesthesiologist. Perioperative management of complex hemodynamic derangements necessitates familiarity with TEE, invasive monitoring, inotropes, vasopressors, cardiac rhythm management devices, single-lung ventilation, and ACLS protocols. In this center, the personnel best fitting these demands are the cardiovascular and thoracic anesthesiologists. The results suggest that care provided by anesthesiologists with expertise in the aforementioned areas allows for maximized successful perioperative outcomes for these challenging cases, with mortality much lower than the anticipated incidence given the complexity of surgery and degree of patient illness.

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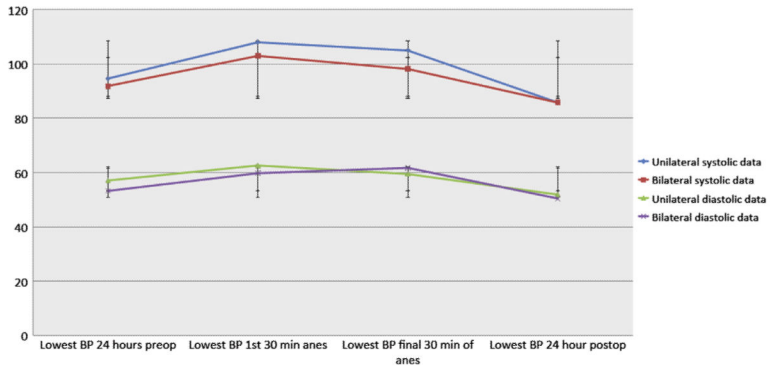

Fig 1.

Hemodynamic data for unilateral versus bilateral sympathectomy expressed as mean systolic and diastolic values. Error bars span one standard deviation above and below the mean. Abbreviations: anes, anesthesia; BP, blood pressure; postop, postoperatively; preop, preoperatively.

Table 1

Patient Demographic Data

Characteristic	
Male (%)	92
Female (%)	8
Age (years)	
Median	58 ± 11
Range	34-75
ASA class	4
LVEF (%)	
Median	25
Range	15-59
ICD (%)	92
Cause of VT (%)	
ICM	23
NICM	54
Hypertrophic CM	8
Other	15
Number of antiarrhythmic drugs (%)	
3 or more	27
2	38
1	31
0	4
Number of VT ablations	
Median	2
Range	0-5
Preoperative renal insufficiency (%)	35
On hemodialysis (%)	12
Diabetes mellitus (%)	31
Preoperative CVA (%)	27
Admitted from home (%)	27
Admitted from ICU (%)	73

NOTE. Data are expressed as percentage, median, and/or range.

Abbreviations: ASA, American Society of Anesthesiologists classification; CM, cardiomyopathy; CVA, cerebrovascular accident; ICD, implantable cardioverter-defibrillator; ICM, ischemic cardiomyopathy; ICU, intensive care unit; LVEF, left ventricular ejection fraction; NICM, nonischemic cardiomyopathy; VT, ventricular tachycardia.

Table 2

Perioperative Data

Characteristic	
Bilateral sympathectomy (%)	42
Length of surgery	
Median	164
Range	91-296
Length of anesthesia	
Median	229
Range	132-358
EBL (mL)	
Median	50
Range	0-400
IVF (mL)	
Median	1,100
Range	150-2500
Blood products transfused (mL)	0
Inotropes before surgery (%)	15
Intraoperative inotropes (%)	50
Postoperative inotropes (%)	27
Days in ICU before surgery	
Median	6
Range	0-29
Days in ICU after surgery	
Median	4
Range	0-30
Extubated in OR (%)	77
EF change(%)	
Median	−15
Range	−38 to +40
Perioperative lABP (%)	15
ECMO (%)	4
Perioperative mortality (%)	
24 hours	0
7 days	8
30 days	12
1 year	23

NOTE. Data are expressed as percentage, median, and/or range.

Abbreviations: EBL, estimated blood loss; ECMO, extracorporeal membrane oxygenation; EF, ejection fraction; IABP, intra-aortic balloon pump; ICU, intensive care unit; IVF, intravenous fluid; OR, operating room.