Ventricular Arrhythmias

17

Sohaib Tariq and Howard A. Cooper

Case Presentation

A 71 year old male with a history of hypertension, hyperlipidemia, and coronary artery disease, with coronary artery bypass grafting 15 years earlier, presented with repeated episodes of lightheadedness over the course of several hours. In the emergency department he was found to be in monomorphic VT at a rate of 206 beats per minute (Fig. 17.1). He was urgently cardioverted to normal sinus rhythm and administered a 150 mg bolus of intravenous amiodarone followed by a continuous infusion. A 12-lead electrocardiogram revealed non-specific T-wave abnormalities. Subsequently, he had intermittent episodes of third degree atrioventricular block with a ventricular escape rhythm at 50 beats per minute. Initial laboratory investigation was significant for potassium of 3.9 mEq/L, magnesium of 1.8 mg/dL, white blood cell count of 22.6 K, and cardiac troponin I of 0.13 ng/mL. He was transferred to the cardiac intensive care unit where he continued to have frequent episodes of monomorphic VT.

S. Tariq
Division of Cardiology, Westchester Medical Center, Valhalla, NY, USA

H.A. Cooper (✉)
Inpatient Cardiology, Division of Cardiology, Westchester Medical Center, Valhalla, NY, USA
e-mail: howard.cooper@wmchealth.org

Question What approach should be taken in management of multiple recurrences of ventricular arrhythmias over a short period of time, i.e. VT storm?

Answer Antiarrhythmic medications and correction of the arrhythmia trigger(s).

The patient was initially treated with intravenous amiodarone and external defibrillation for episodes of VT. A temporary transvenous pacemaker was inserted to prevent bradycardia in the setting of intermittent complete atrioventricular block. Electrolytes were repleted. Intravenous lidocaine was added for breakthrough episodes of VT. Benzodiazepines were administered to ameliorate anxiety. Echocardiography showed left ventricular systolic dysfunction, with an estimated ejection fraction of 30%. Right heart catheterization revealed elevated intracardiac filling pressures and a cardiac index of 1.9 L/min/m^2. An endomyocardial biopsy was negative for signs of inflammation or myocarditis. Left heart catheterization demonstrated severe native three vessel disease with an atretic left internal mammary artery (LIMA) graft to the left anterior descending (LAD) coronary artery, and patent vein grafts to obtuse marginal (OM2), diagonal branch (D1), and right coronary artery (RCA). A drug eluting stent was placed in mid LAD. There was no significant elevation in serum troponin levels. No further episodes of VT were observed, even after discontinuation

R.C. Hyzy (ed.), *Evidence-Based Critical Care*, DOI 10.1007/978-3-319-43341-7_17

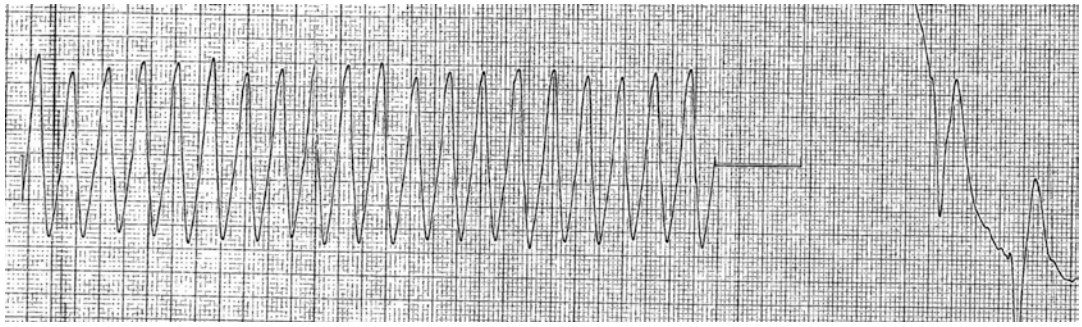

Fig. 17.1 Monomorphic ventricular tachycardia

of lidocaine. The patient received an implantable cardioverter defibrillator (ICD) and was discharged home on a long-acting beta blocker.

Three weeks later the patient presented with pre-syncope and palpitations. ICD interrogation revealed multiple episodes of VT resulting in defibrillator shocks. Intravenous amiodarone was initiated, but VT continued to recur. An intra-aortic balloon pump was placed with subsequent resolution of the VT. He then underwent electro-anatomic mapping followed by radiofrequency catheter ablation, after which he had no further VT.

Principles of Management

Diagnosis

Ventricular arrhythmias include a broad spectrum of rhythm disorders, which may have no clinical consequence or may result in sudden cardiac death. Patients at high risk for ventricular arrhythmias are those with ischemic heart disease, myocarditis, underlying structural heart disease, inherited or acquired channelopathies, drug intoxication, electrolyte derangement, and hyperthyroidism (Table 17.1). However, ventricular arrhythmias may occur in the absence of any of these predisposing factors. The diagnosis of ventricular arrhythmias may be suggested by the clinical presentation, but confirmation requires electrocardiographic recording.

Classification of VT

Ventricular arrhythmias are subdivided into non-sustained (premature ventricular contractions and non-sustained VT) and sustained (defined as VT lasting for more than 30 s or VF). VT may be monomorphic or polymorphic, with *torsades de pointes* representing a specific form of polymorphic VT occurring in the setting of a prolonged QT interval. Electrical storm is defined as 3 or more episodes of sustained VT, VF, or appropriate ICD therapies during a 24-h period. This condition has variably been referred to as "VT storm" or "VT cluster". An important subgroup of electrical storm is incessant VT, which refers to repeated recurrence within 5 min of a technically successful therapy. Management of sustained ventricular arrhythmias is essential aspect of critical care medicine.

Monitoring and Testing

Patients presenting with a suspicion of ventricular arrhythmias should undergo continuous electrocardiographic monitoring in a setting in which appropriate care can be rapidly deployed. A 12-lead ECG should be obtained at baseline and assessed for evidence of ischemia, underlying structural heart disease, and the various channelopathies, such as long QT syndrome or Brugada syndrome (Fig. 17.2). Every attempt should be made to obtain a 12-lead ECG during the occurrence of a ventricular arrhythmia. Confirmation of the presence of a ventricular arrhythmia may require prolonged electrocardiographic monitoring (inpatient or

Table 17.1 Approach to common etiologies of ventricular arrhythmias

Etiology	History	Evaluation	Treatment
Myocardial ischemia	Chest pain, dyspnea	ECG, echocardiography, stress testing, coronary angiography	Coronary revascularization, beta blockers, intra-aortic balloon pump, ablation
Channelopathies	Syncope, family history of sudden death	ECG, exercise testing, provocative pharmacological testing	Removal of QT-prolonging drugs, intravenous magnesium, potassium replacement, temporary pacing, sympathectomy, ICD
Myocarditis	Flu-like symptoms, chest pain	ECG, echocardiography, cardiac MRI, endomyocardial biopsy	Amiodarone, beta blockers, ICD, steroids in selected cases where indicated for subtypes of myocarditis
Electrolyte imbalance	Renal failure, dehydration	Serum electrolytes, ECG	Correction of electrolytes
Cardiomyopathy	Symptoms of heart failure	Echocardiography, coronary angiography, cardiac MRI	Amiodarone, beta blockers, ICD
Sarcoidosis	Lung involvement	Cardiac MRI, biopsy	Steroid therapy, immunomodulating agents, ICD
Arrhythmogenic right ventricular cardiomyopathy	Palpitations, syncope, dyspnea, family history	ECG, cardiac MRI, genetic testing	Beta blockers, ICD, amiodarone
Drug intoxication	Drug abuse, use of QT prolonging medications or digoxin	ECG, serum drug levels	Stop offending agent
Hyperthyroidism	Anxiety, palpitations, heat intolerance	Thyroid hormone studies	Beta blockers, glucocorticoids, anti-thyroid medications

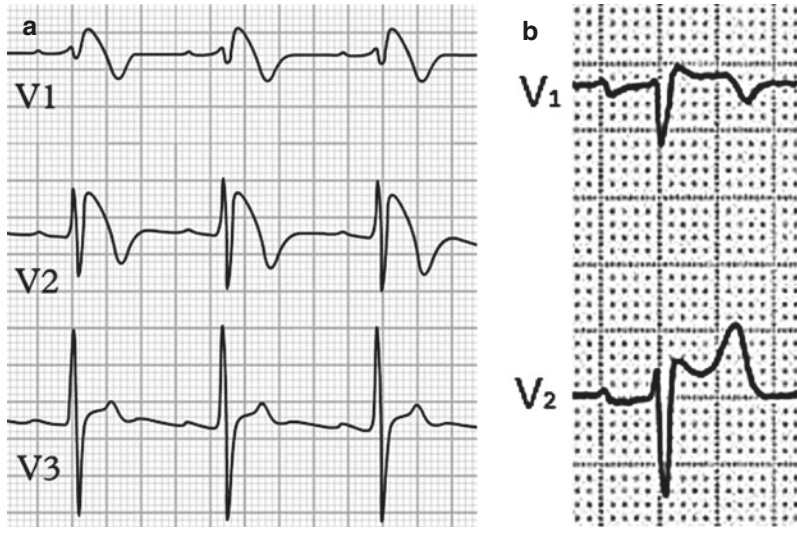

Fig. 17.2 Electrocardiographic tracings in Brugada syndrome. (**a**) Type 1; "coved type" ST-T segment configuration. (**b**) Type 2; "saddle back" pattern

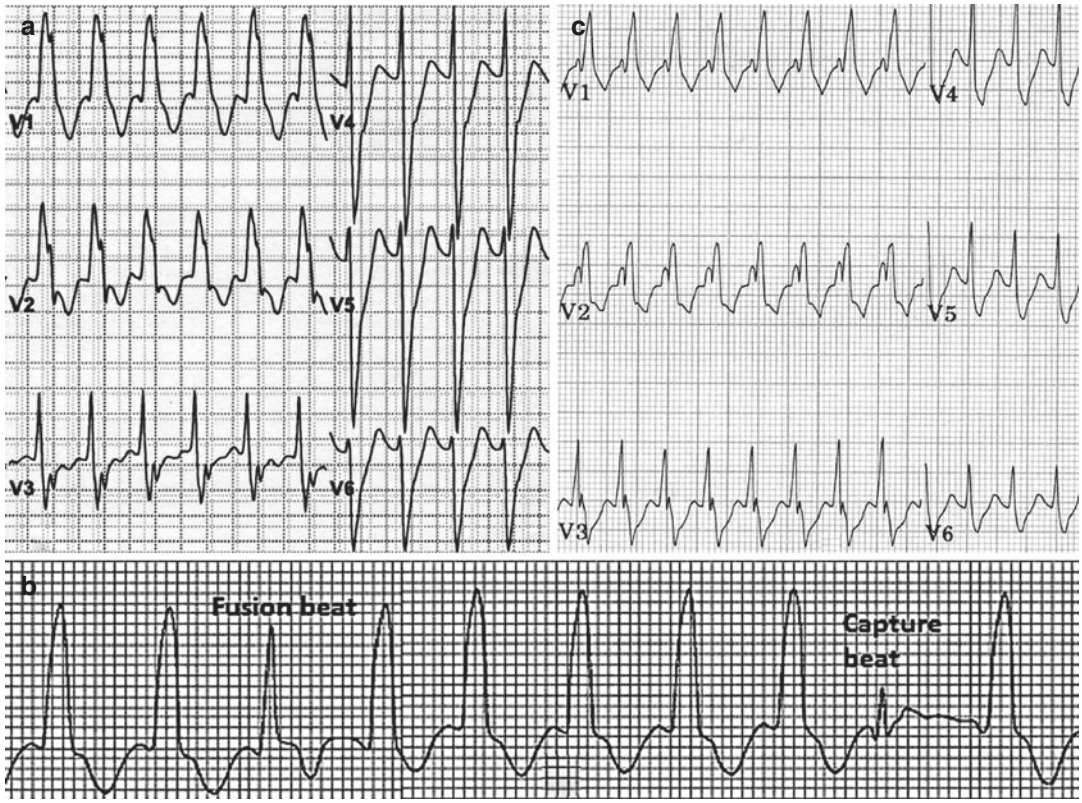

Fig. 17.3 (**a**) Monomorphic ventricular tachycardia. Atrioventricular dissociation can be seen. (**b**) Ventricular tachycardia with fusion beat (hybrid complex of supraventricular and ventricular activation) followed by a capture beat. (**c**) Supraventricular tachycardia with aberrancy. Atrioventricular dissociation and fusion beats are absent. RS complexes in precordial leads are evident

outpatient) (Fig. 17.3), or invasive electrophysiologic testing. Once the diagnosis of a ventricular arrhythmia has been confirmed, further studies should be obtained as clinically indicated in order to elucidate the underlying cause. These may include: serum electrolyte testing, thyroid studies, screening for drugs of abuse, echocardiography, cardiac magnetic resonance imaging, coronary angiography, endomyocardial biopsy, exercise electrocardiography, and provocative pharmacological testing. A family medical history should be assessed for sudden death or known cardiomyopathies. Myocardial ischemia should be considered in all cases of polymorphic VT and ventricular fibrillation with coronary angiography being appropriate early in the evaluation in most cases.

General Measures

Non-sustained VT (Fig. 17.4) is encountered very commonly in the intensive care unit among patients with structural heart disease. In general, no specific therapy is required for asymptomatic patients other than electrolyte repletion and treatment of the underlying condition. For frequent or symptomatic non-sustained VT, however, treatment with beta blockers should be considered. Patients who are not otherwise candidates for an ICD for primary prevention of sudden death, for example, those with non-sustained VT, a history of MI, and moderate left ventricular systolic dysfunction (EF >35 to 40 %), may be considered for electrophysiology study (EPS) when stable in order to determine if an ICD is indicated.

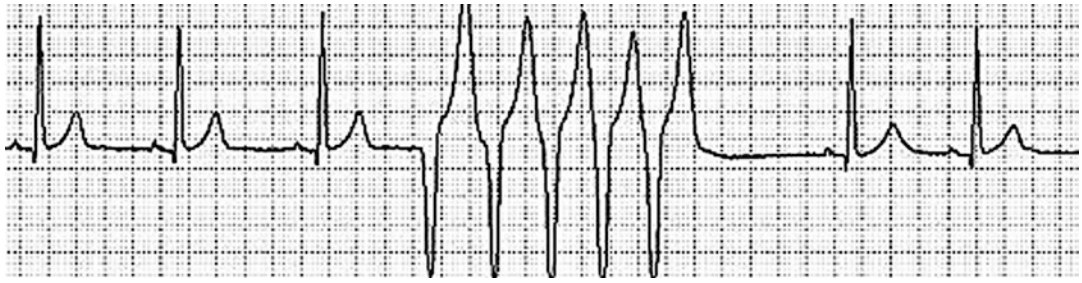

Fig. 17.4 Non-sustained ventricular tachycardia

In patients with sustained ventricular arrhythmias, it is essential that diagnostics and treatments be implemented simultaneously (Fig. 17.5). For patients with cardiac arrest from pulseless VT or VF, guideline-directed ACLS – including rapid defibrillation – should be implemented immediately. When an ICD is present, device interrogation and, if necessary, reprogramming should be performed by a qualified practitioner to ensure the delivery of appropriate therapy and to avoid shocks for non-life-threatening arrhythmias [1]. Triggers of ventricular arrhythmias such as ischemia, electrolyte imbalances, decompensated heart failure, bradycardia, drug intoxication, and hyperthyroidism, should be identified and treated. Heart failure is an under-recognized trigger for new or a significant change in pattern of ventricular arrhythmias in patients with structural heart disease. In patients with VT storm requiring multiple shocks, pain control and sedation with narcotics and/or benzodiazepines should be provided to aid in reducing sympathetic tone. In patients with VT that is refractory to these measures, it is often beneficial to intubate and deeply sedate such patients to further suppress sympathetic activation from pain and anxiety.

A multi-disciplinary approach is appropriate for most patients with sustained ventricular arrhythmias requiring intensive care. The critical care specialist should work closely with the electrophysiology team, and also consider consultation with interventional cardiologists and/or specialists in advanced heart failure as dictated by the specific needs of the patient.

Beta Blockers

Beta blockers should be used in the initial phase of treatment for most patients with ventricular arrhythmias [2]. These drugs play an important role in reducing ischemia, decreasing sympathetic tone, and increasing the fibrillation threshold. Nonrandomized data suggests that intravenous propranolol may be the beta blocker of choice in this setting. However, short-acting metoprolol is a reasonable alternative that is used commonly in clinical practice. In patients with relative hypotension, esmolol is preferred due to its very short half-life [3]. The beta blocker dose should be titrated to maintain a heart rate of 45–60 beats per minute. Insufficient advancement of beta blocking agents is a common error in the management of patients with symptomatic VT. Nevertheless, beta blocker therapy may be limited by bradycardia, hypotension, heart block, and bronchospasm.

Amiodarone

Intravenous amiodarone blocks fast sodium channels and L-type calcium channels, and inhibits norepinephrine release. Amiodarone is highly effective, and has been proven to be superior to other antiarrhythmic agents in suppressing ventricular arrhythmias [4–6]. Amiodarone is given as 150 mg bolus over 10 min (300 mg IV push for cardiac arrest) followed by a continuous infusion, with supplemental boluses given as needed for arrhythmia recurrence. Because of its potential to cause severe phlebitis, intravenous amiodarone

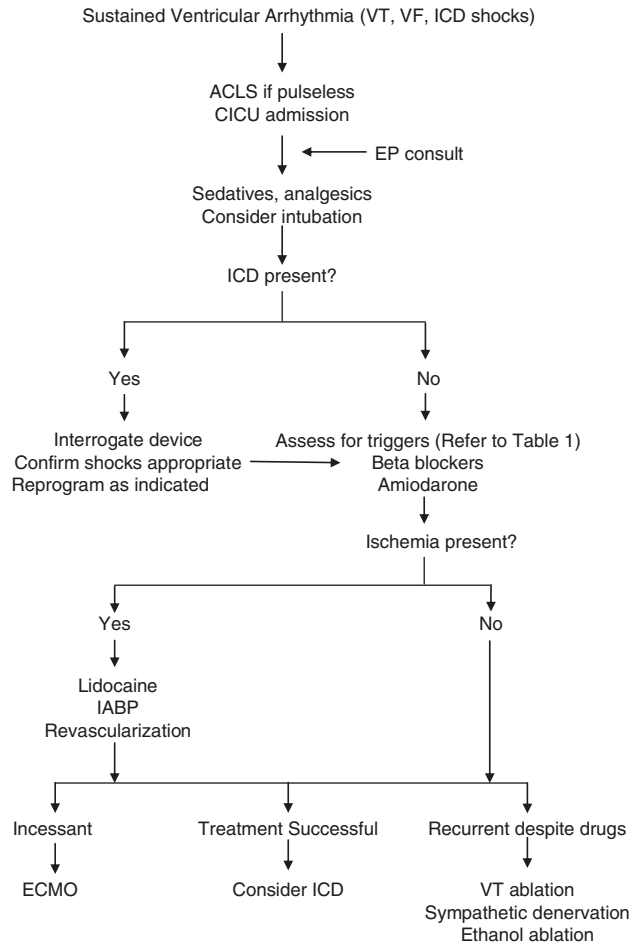

Fig. 17.5 Algorithm for assessment and management of sustained ventricular arrhythmias. *VT* ventricular tachycardia, *VF* ventricular fibrillation, *ACLS* advanced cardiac life support, *CICU* cardiac intensive care unit, *EP* electrophysiology, *ICD* implantable cardioverter defibrillator, *IABP* intra-aortic balloon pump, *ECMO* extracorporeal membrane oxygenation

should be administered via central venous access whenever possible. During short-term use, amiodarone has few side effects but can occasionally cause hypotension. Although amiodarone can increase the QTc interval, precipitation of *torsades de pointes* occurs rarely (<1.0 %) [7].

Lidocaine

Lidocaine blocks fast sodium channels in a use-dependent fashion and does not prolong the QT interval. It is effective in suppressing ventricular arrhythmias occurring in the setting of acute ischemia, but otherwise lidocaine has weak antiarrhythmic properties [5]. In patients resuscitated from VF, administration of lidocaine was associated with reduced survival compared to amiodarone [8]. In the absence of acute ischemia, lidocaine use should be considered if sustained ventricular arrhythmias are refractory to beta blockers and amiodarone. Lidocaine is administered as a bolus dose of 0.5–0.75 mg/kg every 5–10 min (maximum 300 mg total) until the arrhythmia is suppressed, followed by an infusion at 1–4 mg/min. A second bolus of lidocaine 0.5 mg/kg after 20–40 min should be considered because of significant redistribution of the drug. Notable side effects include bradycardia and central nervous system toxicity. Lidocaine is metabolized in the liver and excreted in the urine. Lidocaine should be administered with caution and the mainte-

nance dose adjusted downward in patients with liver disease or decompensated heart failure. Daily serum lidocaine levels should be monitored (therapeutic range: 1.5–5 mcg/mL) and the dose adjusted accordingly.

Digoxin Immune Fab

In patients with sustained ventricular arrhythmias due to digoxin toxicity, anti-arrhythmic drugs are not effective. Digoxin specific Fab antibody should be administered, and temporary pacing should be instituted in the presence of advanced AV block. Common arrhythmias due to digoxin toxicity are:

- Atrioventricular block
- Ectopic atrial tachycardia
- Junctional rhythm
- Ventricular premature beats
- Ventricular tachycardia
- Ventricular fibrillation
- Sinus bradycardia
- Sinoatrial block

Evidence Contour

There are very few randomized trials addressing the acute management of ventricular arrhythmias. Therefore, much of the treatment is based on expert opinion and experience. In particular, the treatment of ventricular arrhythmias which are refractory to the standard treatments outlined above remains an area of great uncertainty.

Anti-arrhythmic Drug Therapy

There are limited randomized data to guide anti-arrhythmic drug therapy of VT. Intravenous amiodarone is widely used as the initial anti-arrhythmic agent due to its high efficacy and excellent short-term safety profile. Beta blockers play an essential role in suppressing sympathetic activity and should be used in addition to amiodarone to maintain rhythm stability. Other than in the setting of acute ischemia, intravenous lidocaine is only modestly effective in terminating ventricular arrhythmias but may be a useful adjunct when amiodarone and beta blockade have been unsuccessful.

Mechanical Circulatory Support – IABP

Placement of an IABP may be useful in the short-term management of patients with refractory ventricular arrhythmias, particularly in the setting of acute ischemia and/or decompensated heart failure, when initial pharmacotherapy and reversal of identified triggers have been unsuccessful. The IABP is placed percutaneously, and may be implanted at the bedside in unstable patients. IABPs increase coronary perfusion, relieve ischemia, and unload the left ventricle, potentially serving as a bridge until a more definitive treatment may be implemented. The effectiveness of the IABP in refractory ventricular arrhythmias has been demonstrated in several case series [9]. Adverse effects are uncommon, but include bleeding, infection, and limb ischemia.

Advanced Mechanical Circulatory Support – ECMO

In rare cases, when VT is incessant and hemodynamically significant, complete mechanical circulatory support may become necessary. Veno-arterial extracorporeal membrane oxygenation (V-A ECMO) involves using a centrifugal pump to remove blood from the venous system, circulate it through an oxygenator, and return oxygenated blood to the arterial circulation. ECMO can be instituted either percutaneously or surgically. In case series, ECMO has shown to facilitate termination of ventricular arrhythmias when other treatments had been exhausted [10].

In addition, ECMO supports the coronary circulation, relieves hypoxia, preserves vital organ perfusion, and allows time to proceed to other definitive treatments. Complications are fairly common, particularly bleeding and limb ischemia.

Radiofrequency Catheter Ablation

Catheter ablation alters the substrate for reentry, and may be useful in critically ill patients with ventricular arrhythmias in whom other modalities have been unsuccessful. This technique, including endocardial and/or epicardial ablation, can be technically challenging, particularly in the setting of multiple reentry circuits or unstable ventricular arrhythmias. Mechanical circulatory support is often useful to allow for proper mapping when the VT is poorly tolerated. Catheter ablation has been demonstrated to be efficacious in treating refractory electrical storm, with a reduction in recurrent VT and cardiac death [11, 12]. Vascular injury, thromboembolism, and cardiac tamponade are potential complications.

Transcoronary Ethanol Ablation

In patients with recurrent VT despite drug therapy and catheter ablation, transcoronary ethanol ablation may be used to treat VT of deep intramyocardial origin. The technique involves the infusion of ethanol – which is toxic to the myocardium – into the coronary artery branch supplying the reentry circuit [13]. Transcoronary ethanol ablation has been shown to prevent the recurrence of VT in a small group of highly selected patients after failed radiofrequency ablation [14]. Complications include myocardial injury, heart block, and ventricular rupture.

Cardiac Sympathetic Denervation

Limited data suggests that surgical sympathectomy may be of benefit in cases of refractory ventricular arrhythmias and VT storm. In a recently published case series involving 41 patients who had previously failed anti-arrhythmic drug therapy and VT ablation, bilateral surgical cardiac sympathetic denervation resulted in a clinically significant reduction in the number of ICD shocks in 90 % of patients and a shock-free survival rate of 50 % at 1 year [15].

References

1. Moss AJ, Schuger C, Beck CA, Brown MW, Cannom DS, Daubert JP, et al. Reduction in inappropriate therapy and mortality through icd programming. N Engl J Med. 2012;367:2275–83.
2. Nademanee K, Taylor R, Bailey WE, Rieders DE, Kosar EM. Treating electrical storm: sympathetic blockade versus advanced cardiac life support-guided therapy. Circulation. 2000;102:742–7.
3. Brodine WN, Tung RT, Lee JK, Hockstad ES, Moss AJ, Zareba W, et al. Effects of beta-blockers on implantable cardioverter defibrillator therapy and survival in the patients with ischemic cardiomyopathy (from the multicenter automatic defibrillator implantation trial-ii). Am J Cardiol. 2005;96:691–5.
4. Connolly SJ, Dorian P, Roberts RS, Gent M, Bailin S, Fain ES, et al. Comparison of beta-blockers, amiodarone plus beta-blockers, or sotalol for prevention of shocks from implantable cardioverter defibrillators: the optic study: a randomized trial. J Am Med Assoc. 2006;295:165–71.
5. Desouza IS, Martindale JL, Sinert R. Antidysrhythmic drug therapy for the termination of stable, monomorphic ventricular tachycardia: a systematic review. Emerg Med J. 2013;68:392–7.
6. Levine JH, Massumi A, Scheinman MM, Winkle RA, Platia EV, Chilson DA, et al. Intravenous amiodarone for recurrent sustained hypotensive ventricular tachyarrhythmias. Intravenous Amiodarone Multicenter Trial Group. J Am Coll Cardiol. 1996;27(1):67–75.
7. Hohnloser SH, Klingenheben T, Singh BN. Amiodarone-associated proarrhythmic effects. A review with special reference to torsade de pointes tachycardia. Ann Intern Med. 1994;121(7):529–35.
8. Dorian P, Cass D, Schwartz B, Cooper R, Gelaznikas R, Barr A. Amiodarone as compared with lidocaine for shock-resistant ventricular fibrillation. N Engl J Med. 2002;346:884–90.
9. Fotopoulos GD, Mason MJ, Walker S, Jepson NS, Patel DJ, Mitchell AG, et al. Stabilisation of medically refractory ventricular arrhythmia by intra-aortic balloon counterpulsation. Heart. 1999;82(1):96–100.
10. Tsai FC, Wang YC, Huang YK, Tseng CN, Wu MY, Chu JJ, et al. Extracorporeal life support to terminate refractory ventricular tachycardia. Crit Care Med. 2007;35:1673–6.
11. Calkins H, Epstein A, Packer D, Arria AM, Hummel J, Gilligan DM, Cooled RF, et al. Multi Center Investigators

Group. Catheter ablation of ventricular tachycardia in patients with structural heart disease using cooled radiofrequency energy: results of a prospective multicenter study. J Am Coll Cardiol. 2000;35:1905–14.
12. Carbucicchio C, Santamaria M, Trevisi N, Maccabelli G, Giraldi F, Fassini G, et al. Catheter ablation for the treatment of electrical storm in patients with implantable cardioverter-defibrillators: short- and long-term outcomes in a prospective single center study. Circulation. 2008;117:462–9.
13. Sacher F, Sobieszczyk P, Tedrow U, Eisenhauer AC, Field ME, Selwyn A, et al. Transcoronary ethanol ventricular tachycardia ablation in the modern electrophysiology era. Heart Rhythm. 2008;5:62–8.
14. Tokuda M, Sobieszczyk P, Eisenhauer AC, Kojodjojo P, Inada K, Koplan BA, et al. Transcoronary ethanol ablation for recurrent ventricular tachycardia after failed catheter ablation: an update. Circ Arrhythm Electrophysiol. 2011;4:889.
15. Vaseghi M, Gima J, Kanaan C, Ajijola O, Marmureanu A, Mahajan A, et al. Cardiac sympathetic denervation in patients with refractory ventricular arrhythmias or electrical storm: intermediate and long-term follow-up. Heart Rhythm. 2014;11:360.