Review

The Evaluation and Management of Electrical Storm

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Electrical storm is an increasingly common and life-threatening syndrome that is defined by 3 or more sustained episodes of ventricular tachycardia, ventricular fibrillation, or appropriate shocks from an implantable cardioverter-defibrillator within 24 hours. The clinical presentation can be dramatic. Electrical storm can manifest itself during acute myocardial infarction and in patients who have structural heart disease, an implantable cardioverter-defibrillator, or an inherited arrhythmic syndrome. The presence or absence of structural heart disease and the electrocardiographic morphology of the presenting arrhythmia can provide important diagnostic clues into the mechanism of electrical storm. Electrical storm typically has a poor outcome.

The effective management of electrical storm requires an understanding of arrhythmia mechanisms, therapeutic options, device programming, and indications for radiofrequency catheter ablation. Initial management involves determining and correcting the underlying ischemia, electrolyte imbalances, or other causative factors. Amiodarone and β-blockers, especially propranolol, effectively resolve arrhythmias in most patients. Nonpharmacologic treatment, including radiofrequency ablation, can control electrical storm in drug-refractory patients. Patients who have implantable cardioverter-defibrillators can present with multiple shocks and may require drug therapy and device reprogramming. After the acute phase of electrical storm, the treatment focus should shift toward maximizing heart-failure therapy, performing revascularization, and preventing subsequent ventricular arrhythmias. Herein, we present an organized approach for effectively evaluating and managing electrical storm. ***(Tex Heart Inst J 2011;38(2):111-21)***

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Electrical storm is a life-threatening syndrome that involves recurrent episodes of ventricular arrhythmias. It is defined as 3 or more sustained episodes of ventricular tachycardia (VT), ventricular fibrillation (VF), or appropriate implantable cardioverter-defibrillator (ICD) shocks during a 24-hour period.[1] Sustained VT lasts 30 seconds, involves hemodynamic compromise, or requires intervention to terminate the episode. Management of electrical storm is challenging and requires an approach tailored to the underlying cause. The condition can manifest itself during the acute phase of a myocardial infarction (MI) and in the presence of structural heart disease, an ICD, or an inherited arrhythmic syndrome. The major symptoms are palpitations, dizziness, and often syncope. The clinical presentation might be dramatic and can involve cardiac arrest or multiple episodes of potentially fatal arrhythmias. Patients who have an ICD can present with recurrent shocks. Effective management of electrical storm requires knowledge of arrhythmia mechanisms, therapeutic options, ICD programming, and emerging techniques for the treatment of refractory cases. Herein, we present an organized approach for effectively evaluating and managing electrical storm.

Incidence and Prognostic Implications

The incidence of electrical storm varies depending upon the populations that are studied. The condition occurs in 10% to 20% of ICD recipients.[2] Patients who are experiencing acute MI or have had an MI or those who have an inherited arrhythmic syndrome are also susceptible. As the prevalence of congestive heart failure continues to rise, even more patients will undergo ICD implantation.[3] The incidence of electrical storm is lower when ICDs are placed for primary versus secondary prevention.[4] In a MADIT-II substudy of 719 patients,[4] 4% developed electrical storm over an average of 20.6 months. There were no differences in baseline characteristics be-

tween patients with electrical storm and those with isolated episodes of VT and VF. In another trial,[5] 20% of patients who received ICDs for secondary prevention experienced electrical storm during a 31-month period. Intracardiac electrograms that were recorded during those episodes showed that most cases were due to VT and that they occurred an average of 9.2 months after ICD implantation.

Data on the prognostic significance of electrical storm strongly suggest that these patients have a poor outcome. Electrical storm might be an independent risk factor for cardiac death. In the AVID trial,[5] patients with electrical storm had an increased risk of nonsudden cardiac death (risk ratio, 2.4). In the MADIT-II substudy, patients with electrical storm had a 7.4-fold higher risk of death than patients without electrical storm.[4] Both studies showed that the risk of death was highest within the first 3 months after a storm. The prognosis remained poor for patients who survived the initial period of electrical instability—many sustained recurrent electrical storms and refractory heart failure. It is unclear whether electrical storm contributes directly to a poor outcome or is simply an epiphenomenon of advanced structural heart disease.[6,7] Recurrent VT or VF and ICD shocks may cause left ventricular (LV) systolic dysfunction and myocardial injury,[8,9] which can lead to adrenergic neurohormonal activation and exacerbate heart failure.[10,11]

Initial Evaluation of Electrical Storm

Caring for patients with electrical storm begins by accurately diagnosing the clinical arrhythmia. In patients who have bundle branch block, ventricular preexcitation (Wolff-Parkinson-White syndrome), or a rate-related aberrancy, supraventricular tachycardia (SVT) can resemble VT. The differentiation of VT from SVT with aberrant conduction has been well described by Wellens,[12] Kindwall,[13] and Brugada[14] and their respective associates. It is strongly emphasized that the patient's hemodynamic status is not helpful in this distinction. Patients with VT may have minimal symptoms that prompt the erroneous diagnosis of SVT with aberrant conduction. For this reason, an ambiguous wide-complex tachycardia should be presumed to be VT, especially in patients who have structural heart disease. If this rule is followed, the diagnosis of electrical storm will be accurate in 80% of all patients with tachycardia and in 95% who have had a previous MI.[15]

Furthermore, treating VT as though it were SVT (by using calcium-channel blockers or adenosine) can precipitate cardiac arrest, whereas SVT might resolve with treatment aimed at VT. If cardiac arrest results from VT-induced electrical storm, it is important to incorporate all aspects of critical care in this acute setting. These elements include prompt management of a compromised airway, post-shock bradycardia, hypotension, and ischemia, and defibrillation of symptomatic or hemodynamically unstable patients. Simultaneous therapies are usually necessary. Patients who have poor systolic function or rapid VT might require multiple electrical cardioversions or defibrillations. When hemodynamic status is stable, antiarrhythmic medication can be given as a trial. If medical therapy is unsuccessful, cardioversion under sedation is indicated.

Patients who present with refractory VT or VF often have underlying structural heart disease and chronic renal failure.[16] Other risk factors for electrical storm include advanced age, male sex, a low LV ejection fraction (LVEF), and New York Heart Association functional class III or IV heart failure.[6] Antiarrhythmic agents can precipitate electrical storm.[2] Of note, ICD recipients with diabetes mellitus and patients who are taking lipid-lowering drugs reportedly have a lower incidence of electrical storm.[17]

An important step in evaluating this condition is to identify and reverse the causative factors of electrical storm. Specific precipitants include acute ischemia, worsening heart failure, hypokalemia, hypomagnesemia, arrhythmogenic drug therapy, hyperthyroidism, and infection or fever.[1] Active ischemia, decompensated heart failure, and electrolyte imbalances should be remediated aggressively. Patients often have severe anxiety and increased catecholamine levels, which can amplify the severity and perpetuate electrical storm. Multiple predisposing factors can be present, and the complex interactions that culminate in an electrical storm are poorly understood.

Clinical Syndromes of Electrical Storm

Electrical storm develops when a vulnerable anatomic substrate (such as that from structural heart disease or scarring after an MI) is affected by a triggering event, such as premature ventricular contractions (PVCs) or an electrolyte imbalance. (In the absence of vulnerable substrate, these other events might be of little clinical consequence.) Determining the cause of electrical storm is essential, because treatment must target the underlying mechanism.

Electrical storm can initially be classified on the basis of 3 gross electrocardiographic (ECG) surface morphologies: monomorphic VT, polymorphic VT, or VF (Fig. 1).

Monomorphic Ventricular Tachycardia

In most cases, electrical storm presents as sustained monomorphic VT that is associated with structural heart disease. Monomorphic VT occurs when the ventricular activation sequence is the same without any variation in the QRS complexes. Most monomorphic VT is due to electrical wavefront reentry around a fixed

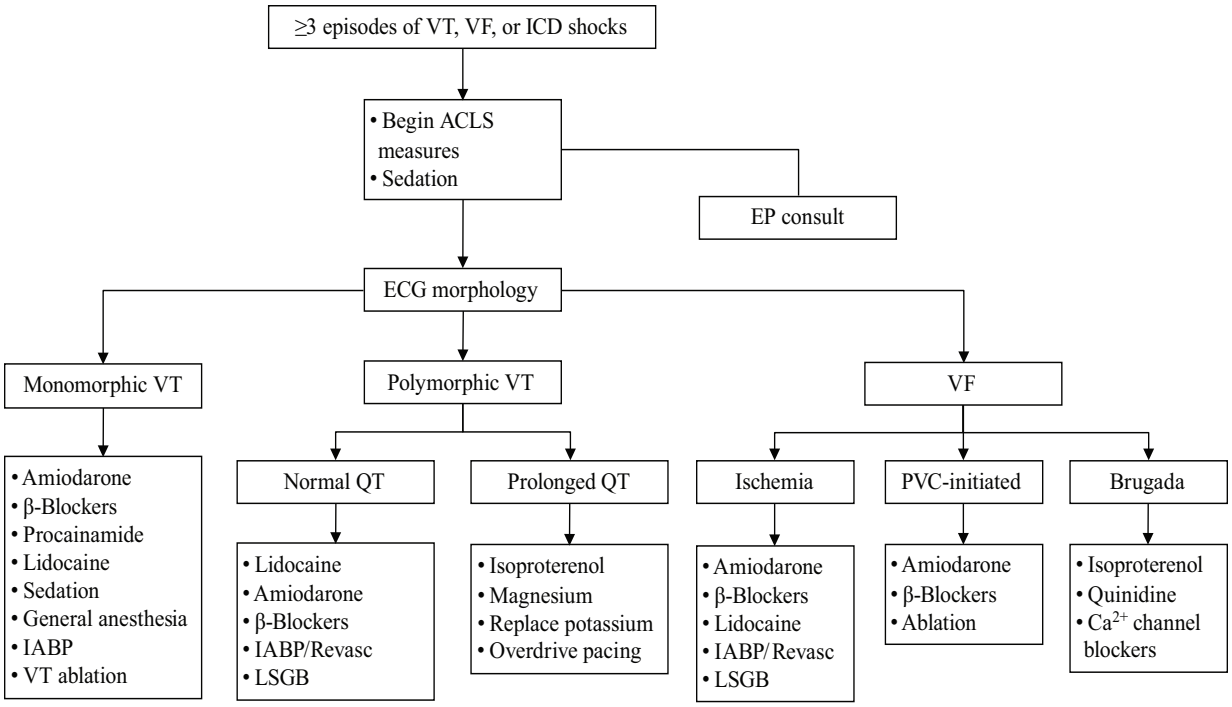

Fig. 1 *Management of electrical storm.*

ACLS = advanced cardiac life support; ECG = electrocardiographic; EP = electrophysiology; IABP = intra-aortic balloon pump; ICD = implantable cardioverter-defibrillator; LSGB = left stellate ganglion blockade; PVC = premature ventricular contraction; Revasc = revascularization; VF = ventricular fibrillation; VT = ventricular tachycardia

anatomic barrier, most commonly scar tissue after MI. Monomorphic VT due to wavefront reentry does not require active ischemia as a trigger, and it is uncommon in patients who are having an acute MI.

In ischemic or nonischemic cardiomyopathy, the vulnerable substrate for reentry lies within heterogeneous areas of scarred myocardium. After an acute MI, or as nonischemic cardiomyopathy progresses, the heart undergoes structural changes. Fibrosis leads to scar formation, which creates areas of conduction block. However, bundles of myofibrils can survive, particularly around the border of a scar. Slow conduction through these regions provides a pathway for electrically stable reentry. Then, an otherwise harmless trigger, such as premature ventricular depolarization, is all that is required to initiate monomorphic VT.

During monomorphic VT, the surface ECG morphology depends upon the location of the scar and the exit site into the ventricle. The VT may occur early or late after MI. The burden of ventricular arrhythmias is higher when inadequate reperfusion or large areas of infarction are present. Monomorphic VT can be asymptomatic or can present as cardiac arrest. The degree of hemodynamic compromise depends upon the ventricular rate, LV function, the presence of heart failure, any loss of atrioventricular synchrony, and the pattern of ventricular activation.[18] Amiodarone and β-blockers are preferred for pharmacologic management.

Polymorphic Ventricular Tachycardia

Polymorphic VT occurs when the ventricular activation sequence on surface telemetry or ECG consists of beat-to-beat variations in the QRS complexes. Polymorphic and monomorphic VT have fundamentally different mechanisms. For polymorphic complexes to appear on the surface ECG, multiple wavefronts must propagate throughout the heart or appear simultaneously in several parts of the heart.[19] Polymorphic VT can be associated with a normal or a prolonged QT interval in sinus rhythm. Although polymorphic VT as an ECG pattern is most often associated with acute ischemic syndromes, it is also seen in the absence of organic heart disease.[20] Patients who have acute myocarditis or hypertrophic cardiomyopathy may also present with polymorphic VT. Therapy for polymorphic VT and VF varies, depending upon the mode of initiation and the underlying QT interval in sinus rhythm.

Electrical storm is often the initial manifestation of ischemia. In contrast, monomorphic VT is unusual during the first 72 hours of infarction unless the patient has previously infarcted myocardium that serves as a substrate for reentry. The specific arrhythmia that arises from acute myocardial ischemia is almost always polymorphic VT.[21] In these cases, the baseline QT interval may be normal. In acute MI, polymorphic VT can be due to ischemia, altered membrane potential, triggered activity, necrosis, or scar formation. Ischemia

may result in dispersion of electrical refractory periods between the endocardium and epicardium, which is a requirement for multiple waves of reentry.[22] Ischemia increases Purkinje cell automaticity, and the spontaneous firing of these fibers triggers polymorphic VT or VF.

Patients may experience intense electrical storms of polymorphic VT during episodes of ischemia. The most effective treatment is to reverse the ischemia with emergency coronary revascularization or with anti-ischemic, antiplatelet, or thrombolytic agents. Amiodarone and β-blockers are the most effective antiarrhythmic agents. Initially, lidocaine was thought to be the optimal therapy for VT in the presence of ischemia, but randomized trials have not confirmed that it is superior to other antiarrhythmic medications. Magnesium therapy is unlikely to be effective in polymorphic VT that is associated with normal QT intervals.

Patients with recurrent polymorphic VT should have their baseline (sinus-rhythm) ECG carefully evaluated for a prolonged QT interval, because this entity requires a unique clinical approach. For example, torsades de pointes is pause-dependent polymorphic VT with a long QT interval, often in the presence of bradycardia. Close inspection of the QT interval in sinus rhythm may reveal marked QT prolongation. U waves are usually present but might be difficult to distinguish from abnormal T waves.[21] Risk factors for torsades de pointes include female sex, bradycardia, heart block, QT-prolonging drugs, hypokalemia, and inherited long QT syndrome.

The initial evaluation of polymorphic VT with a long QT interval requires consideration of inherited and acquired causes. Inherited long QT syndromes are associated with sudden cardiac death, but they rarely present as an electrical storm. These syndromes have been reviewed elsewhere.[23] Using catecholamines, including isoproterenol, should be avoided in these patients. Polymorphic VT with a long QT interval should prompt a search for acquired causes, including electrolyte imbalances (hypokalemia, hypocalcemia, or hypomagnesemia), hypothyroidism, and the use of medications that are known to prolong the QT interval, including sotalol, haloperidol, methadone, and erythromycin. In instances of bradycardia or heart block, torsades de pointes should be managed with isoproterenol therapy or temporary pacing, followed by the implantation of a permanent pacemaker in refractory cases. Intravenous magnesium administration is reasonable therapy for patients with polymorphic VT and a long QT interval. In all cases, potassium repletion to a serum level above 4.5 mmol/L is recommended.[1]

Ventricular Fibrillation

Ventricular fibrillation, or chaotic (fibrillatory) activation on surface telemetry or ECG, is usually fatal if it is not treated promptly. Even with defibrillation, VF may recur repeatedly and present as electrical storm. When this happens, mortality rates are between 85% and 97%.[24,25] Ischemia, which is the primary mechanism of VF storm, should be the focus of treatment.

Patients who have a normal heart may develop a VF storm that is triggered by closely coupled monomorphic PVCs. This syndrome is characterized by identical PVCs (in terms of morphology and coupling intervals relative to the preceding QRS complexes) during sinus rhythm that lead to VF.[26,27] A similar presentation has been observed late after MI.[28,29] The PVC is the trigger and often originates in the distal Purkinje system. Radiofrequency (RF) catheter ablation at these sites can eliminate future VF episodes.

Brugada syndrome, an inherited arrhythmic condition caused by a defective cardiac sodium channel gene, manifests itself in adulthood with recurrent VF and a characteristic ECG pattern of right bundle branch block with ST-segment elevation in leads V_1 through V_3. Brugada syndrome can present as electrical storm.[30] The prevalence of malignant arrhythmias ranges from 5% in patients without previous arrhythmias to 40% in those with a history of cardiac arrest. Hypokalemia, a high vagal tone, bradycardia, and fever are predisposing factors for electrical storm. However, after evaluating patients with Brugada syndrome who had a history of electrical storm versus those without a history, Ohgo and colleagues[31] could not identify any predictive clinical, laboratory, ECG, or electrophysiologic characteristics. In that study, continuous isoproterenol infusion completely normalized ST-segment elevation and suppressed electrical storm. Oral antiarrhythmic therapy may be required, because attempts to wean patients from isoproterenol can result in recurrent VF. Because class I antiarrhythmic agents are potent sodium-channel blockers, most are contraindicated in patients who have Brugada syndrome. However, quinidine has prevented ventricular arrhythmias in these patients by blocking the transient outward potassium channel that is responsible for phase 1 of the action potential. Quinidine is recommended therapy for refractory cases of electrical storm caused by Brugada syndrome; however, further studies are required before routine use can be recommended.[32]

Pharmacologic Therapy for Electrical Storm

Adrenergic Blockade

Electrical storm activates the sympathetic nervous system. Although extremely high levels of endogenous catecholamines have been documented during cardiac arrest,[33] the current guidelines for advanced cardiac life support state that epinephrine or vasopressin should be used in cases of pulseless VT or VF. Epinephrine

induces intense vasoconstriction by stimulating the α-adrenergic receptor and redirecting blood flow to the central circulation, thereby increasing coronary perfusion. Studies have shown increased rates of spontaneous circulation, coronary blood flow, and short-term survival after the administration of epinephrine.[34,35] However, catecholamines are proarrhythmic and may exacerbate ventricular arrhythmias. Epinephrine makes the patient more susceptible to VF, contributes to myocardial dysfunction, and increases myocardial oxygen demand by stimulating the β-adrenergic receptor.[36] The beneficial α-adrenergic effects of catecholamines on coronary perfusion pressure may be outweighed by the detrimental effects of the β-adrenergic receptor on VF susceptibility and by the increased demand for myocardial oxygen.[37]

β-Blockers play a key role in the management of electrical storm. Their effects were discovered in the 1970s, when they were studied as therapy for acute MI. Propranolol consistently decreases the incidences of fatal VF during acute MI and sudden cardiac death after MI.[38] Although several β-blockers decrease susceptibility to VF, most of the studies have focused on propranolol. In a canine study,[39] β-blockers increased the fibrillation threshold (that is, made the animals less susceptible to fibrillation) 6-fold under ischemic and nonischemic conditions. The improvement was greater with the use of more potent β-blockers and those that antagonized both the β_1 and β_2 receptors. In patients with congestive heart failure, propranolol decreases sympathetic outflow more than does metoprolol, perhaps because β_2 receptors prevail in failing hearts.[40] The lipophilic nature of propranolol enables active penetration of the central nervous system and the blockade of central and prejunctional receptors in addition to peripheral β receptors.[41,42]

Propranolol may effectively suppress an electrical storm even when metoprolol has failed.[38] Therefore, propranolol is the preferred β-blocker, pending further clinical studies. Nademanee and colleagues[43] investigated the efficacy of sympathetic blockade in electrical storm by comparing propranolol, esmolol, and left stellate ganglionic blockade to combined lidocaine, procainamide, and bretylium therapy. Their patients had experienced a recent MI and more than 20 episodes of VT within 24 hours or more than 4 episodes per hour. Although the trial was nonrandomized, sympathetic blockade provided a marked survival advantage (78% vs 18% at 1 wk, and 67% vs 5% at 1 yr). Despite the high doses of propranolol, heart failure was not exacerbated. These authors and others[38] have suggested that the combination of amiodarone and propranolol improves survival rates and should be the mainstay of therapy in managing electrical storm. Because propranolol can exacerbate heart failure in patients with poor systolic function, its use in these patients should be carefully monitored.

Amiodarone

Amiodarone is widely used in the treatment of electrical storm.[1] In acute amiodarone therapy, rapid intravenous administration blocks fast sodium channels in a use-dependent fashion (producing more channel blockade at faster heart rates), inhibits norepinephrine release, and blocks L-type calcium channels but does not prolong ventricular refractoriness. Conversely, in oral amiodarone therapy, prolonged ventricular refractory periods are seen over periods ranging from days to weeks.[44,45] Amiodarone has few negative inotropic effects and is safe in patients who have depressed systolic function. Moreover, the incidence of torsades de pointes is low in such patients despite the potential for significant prolongation of the QT interval. Amiodarone has resolved electrical storm at conversion rates of approximately 60%. When compared with placebo in the ARREST trial, amiodarone improved survival-to-hospital admission rates in patients who had an electrical storm that involved VF or pulseless VT.[46] The trial lacked the statistical power to detect differences in rates of survival to hospital discharge.

Amiodarone can be effective even when other agents have been ineffective. Levine and colleagues[47] examined 273 hospitalized patients who had electrical storm that was refractory to lidocaine, procainamide, and bretylium therapy. When amiodarone was given, 46% of the patients survived for 24 hours without another episode of VT, and another 12% responded after taking amiodarone plus another agent. In short-term use of the drug, side effects were rare. Amiodarone is also effective as adjunctive therapy to prevent recurrent ICD shocks.[48] Although long-term amiodarone therapy is usually successful, substantial side effects include pulmonary fibrosis, hypothyroidism, liver toxicity, and corneal deposits. In addition, amiodarone may increase the energy required for successful defibrillation, so patients with ICDs should undergo repeat defibrillation-threshold testing. Patients who have episodes of electrical storm despite amiodarone therapy may be candidates for RF ablation.

Class I Antiarrhythmic (Sodium Channel-Blocking) Agents

Lidocaine binds to fast sodium channels in a use-dependent fashion. Binding increases under cellular conditions that are common in ischemic VT, such as a reduced pH, a faster stimulation rate, and a reduced membrane potential.[49] However, outside the setting of ischemia, lidocaine has relatively weak antiarrhythmic properties: conversion rates from VT to sinus rhythm range from 8% to 30%. In 1 study of 347 patients who had out-of-hospital, shock-resistant VT or VF, only 12% who were randomized to receive lidocaine survived to hospital admission, versus 23% who received amiodarone. On the basis of this and other findings,

amiodarone has replaced lidocaine as 1st-line therapy for refractory VT and VF.[50]

The 2006 American College of Cardiology/American Heart Association guidelines for treating ventricular arrhythmias[1] gave a IIb recommendation ("usefulness is less well established") for intravenous lidocaine only in the treatment of polymorphic VT that is associated with ischemia. If lidocaine is used, it should be administered as an intravenous bolus of 0.5 to 0.75 mg/kg that is repeated every 5 to 10 min as needed. A continuous intravenous infusion of 1 to 4 mg/min maintains therapeutic levels. The maximum total dose is 3 mg/kg over 1 hr.

Procainamide blocks fast sodium channels in a use-dependent fashion. However, the active metabolite of procainamide, N-acetylprocainamide, blocks potassium channels and accounts for much of the antiarrhythmic effect in vivo. Procainamide prolongs the QT interval and therefore could cause torsades de pointes. Its use is contraindicated in patients with impaired renal function, because N-acetylprocainamide is excreted by the kidneys. When given as a loading dose of 100 mg over 5 min, procainamide is a reasonable choice for terminating monomorphic VT. In patients with depressed systolic function, procainamide can cause hypotension or prolong the width of the QRS complex by more than 50%, which would necessitate discontinuation of the drug.

Anesthetic Agents

The physical and emotional stress that patients experience in association with electrical storm and multiple electrical cardioversions often perpetuates arrhythmias. All patients who have electrical storm should be sedated. Short-acting anesthetics such as propofol, benzodiazepines, and some agents of general anesthesia have been associated with the conversion and suppression of VT.[51] Left stellate ganglion blockade and thoracic epidural anesthesia have also reportedly suppressed electrical storms that were refractory to multiple antiarrhythmic agents and β blockade.[43,52] These therapeutic approaches directly target nerve fibers that innervate the myocardium, and a reduced adrenergic tone is most likely responsible for the reported efficacy.[53] Further study is needed to determine whether sedative and anesthetic agents have direct antiarrhythmic effects.

Nonpharmacologic Therapy

The suppression of malignant arrhythmias is an accepted indication for placing an intra-aortic balloon pump or percutaneous LV assist device.[1,24] These devices increase coronary perfusion pressure and can dramatically relieve the ischemic substrate. The mechanical effects of balloon counterpulsation might be directly antiarrhythmic, because this therapy has been effective in treating electrical storm outside the presence of ischemia.[54] The mechanism may involve reductions in afterload, LV size, and wall tension.[55] Extracorporeal life support has been used to terminate refractory ventricular arrhythmias.[56] If life support is implemented, its deployment early during electrical storm is important for achieving successful outcomes, preventing secondary organ damage, maintaining sufficient cardiac unloading, and avoiding complications.

Intracardiac mapping and RF ablation can alter the myocardial substrate for reentry. Ablating multiple and unstable VTs is challenging, and electroanatomic or noncontact mapping is frequently performed. Percutaneous LV assist devices provide hemodynamic support and enable the mapping and ablation of unstable VT.[57] In the past, RF ablation to resolve electrical storm or to halt frequent ICD shocks was considered only after therapy with multiple antiarrhythmic drugs had failed. However, in a multicenter trial, the RF ablation of VT effectively reduced appropriate ICD shocks in patients who had presented with multiple VTs.[58] When frequent ICD therapy is the indication for RF ablation, the cycle length of the clinical VT can be obtained from stored intracardiac electrograms.

Prophylactic RF ablation at the time of ICD implantation is beneficial. In a study of patients with unstable VT, cardiac arrest, or syncope with inducible VT, patients who underwent prophylactic VT ablation plus ICD implantation received fewer ICD shocks than did those who underwent ICD implantation only.[59] In a multicenter trial, patients with stable VT, a history of MI, and low LVEF underwent prophylactic RF ablation plus ICD implantation and had longer times to recurrence of VT than did patients who received an ICD without ablation.[60] These findings support the early use of RF ablation in patients with VT who receive an ICD and remain at high risk of VT.

In regard to acute management, emergency RF ablation completely suppressed drug-refractory electrical storm in all 95 patients in 1 series.[61] Many were hypotensive and required hemodynamic support. Long-term suppression of electrical storm was achieved in 92%, and 66% were free of VT at 22-month follow-up examination. Of note, the endpoint for ablation was the noninducibility of all clinical VTs. Of the 10 patients who continued to have inducible VT, 8 had recurrent electrical storm, and 4 died despite appropriate ICD therapy.

Radiofrequency ablation is also indicated in recurrent polymorphic VT or VF when specific triggers (such as monomorphic PVCs) can be targeted. In this clinical setting, electrical storm has been durably suppressed in patients with ischemic and nonischemic cardiomyopathy.[26,28] Pending further study, early intervention for electrical storm with RF ablation appears to be feasible. The Heart Rhythm Society and the European Heart Rhythm Association support the use of ablation early in the management of recurrent VT.[62]

Electrical Storm in ICD Patients

Implantable cardioverter-defibrillators are commonly used in patients who are at high risk of sudden cardiac death. However, these devices do not prevent arrhythmias, and implanting an ICD is contraindicated in the acute phase of electrical storm. Before ICDs, many patients would have died of the initial malignant arrhythmia; now, ICD recipients may survive the arrhythmia only to experience multiple recurrences and shocks over time. Intravenous analgesics and sedatives should be given early and aggressively to patients who sustain multiple ICD shocks.[63] If an ICD fails to convert a life-threatening rhythm, external defibrillation pads should be ready for use.

An ICD storm may result from appropriate therapy (antitachycardia pacing, cardioversion, or defibrillation), inappropriate therapy (shocks without evidence of an arrhythmia), or phantom shocks (Fig. 2). (These last 2 conditions are not considered to be true electrical storm.) In cases of ongoing arrhythmia with hemodynamic compromise, the arrhythmia should be corrected immediately. Interrogating the device helps to distinguish appropriate from inappropriate therapy. If the device reveals appropriate termination of VT or VF, a search should begin for ischemia, electrolyte imbalances, worsening heart failure, and other causes. Transient ST-segment changes and mildly elevated cardiac troponin levels are common after multiple shocks. Shocks without evidence of an arrhythmia indicate device malfunction, such as the sensing of electrical noise from a fractured lead. In such cases, the patient should be hospitalized and observed by means of telemetry with the ICD programmed to "off." (The nursing staff should be apprised that the ICD is turned off.) Rapid SVT or atrial fibrillation may result in inappropriate shocks, in which case a magnet can be placed over the ICD to inhibit sensing and treatment of the arrhythmia. If the patient develops a ventricular arrhythmia, removing the magnet enables the delivery of therapy. Applying a magnet does not alter the pacing ability of the ICD.

Shocks from ICDs have adverse effects. Among patients with heart failure who receive an ICD for primary prevention, those who receive shocks for arrhythmia have a higher mortality rate than do patients who receive no shocks.[64] Not only are the shocks painful and

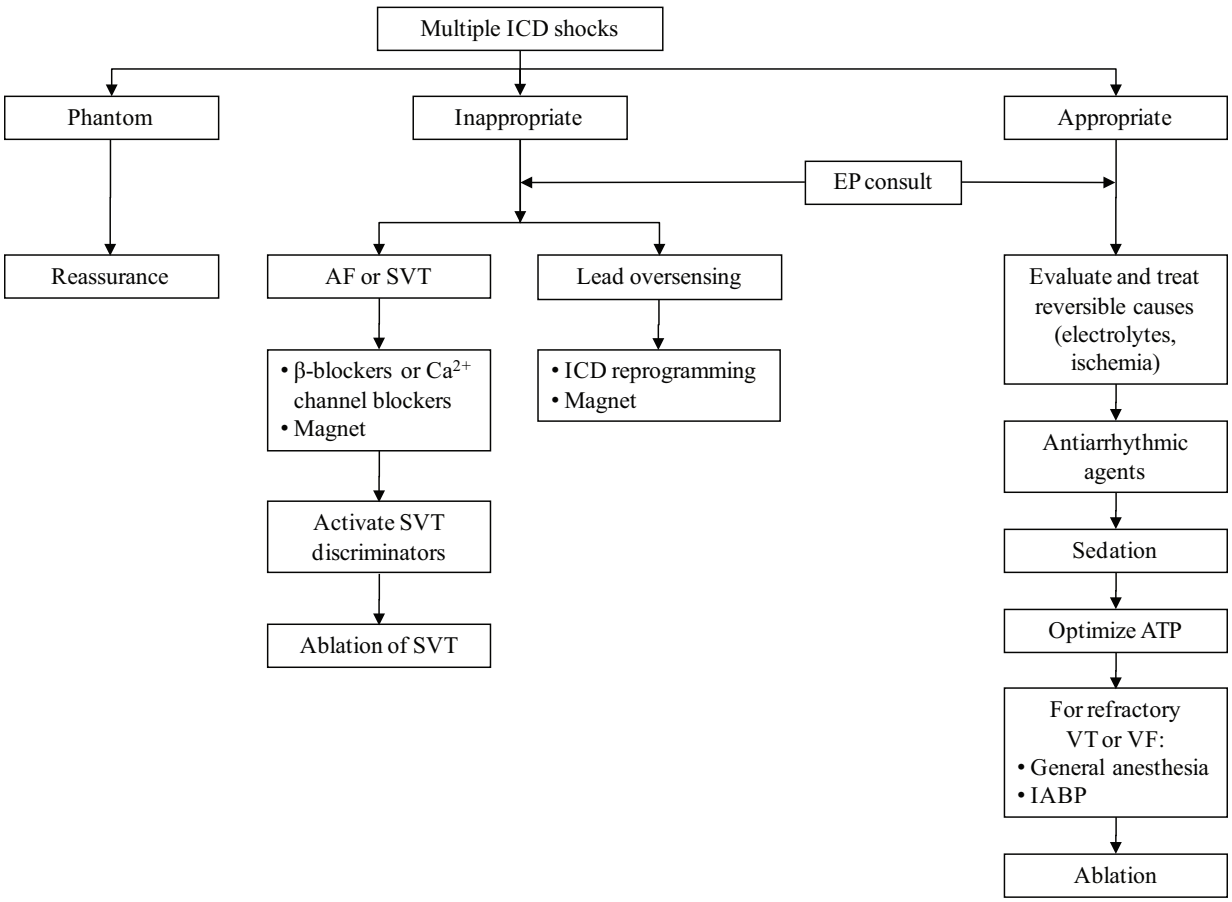

Fig. 2 *Treatment of multiple implantable cardioverter-defibrillator (ICD) shocks.*

AF = atrial fibrillation; ATP = antitachycardia pacing; EP = electrophysiology; IABP = intra-aortic balloon pump; SVT = supraventricular tachycardia; VF = ventricular fibrillation; VT = ventricular tachycardia

distressing to patients, but repeated shocks can cause depression and posttraumatic stress syndrome, and they have been associated with phantom shocks.[65] Patients require reassurance if they report a shock but interrogation of the device reveals that no therapy was delivered.

Antiarrhythmic medications can reduce the frequency of ICD shocks. In 2 studies, racemic sotalol reduced the incidence of recurrent sustained VT and lowered the risks of death and ICD shock.[66,67] The novel class III antiarrhythmic drug azimilide significantly reduced appropriate ICD therapies in the Shock Inhibition Evaluation with Azimilide study.[68] In the multicenter Optimal Pharmacological Therapy in Cardioverter-Defibrillator Patients trial,[69] patients with ICDs were assigned to receive a β-blocker alone, amiodarone plus a β-blocker, or sotalol. At 1-year follow-up evaluation, amiodarone plus a β-blocker had most effectively reduced the number of shocks. The shock rate was 10.3% in the amiodarone plus β-blocker group, 24.3% in the sotalol group, and 38.55% in the β-blocker group.

Coordination with an electrophysiologist is important for ICD patients who experience electrical storm. Programming ICDs to deliver antitachycardia pacing for fast VT (a rhythm in which the rate exceeds the programmed detection criteria) can reduce the need for shocks. Rapid pacing often terminates VT. In the PainFree Rx II trial, antitachycardia pacing very effectively treated fast VT (range, 188–250 beats/min).[70] This resulted in 70% fewer shocks than did normal ICD programming and improved the patients' quality of life. In hopes of avoiding repeated shocks in patients with nonsustained VT, the PREPARE investigators evaluated the effect of extending the VT detection intervals that were needed to trigger ICD shocks.[71] Spontaneous episodes that were treated with shocks were significantly reduced in 700 patients who were undergoing primary prevention.

It is difficult to predict which ICD recipients who have single episodes of VT will develop electrical storm. Progressive heart failure has been a predictor of electrical storm, in several studies.[4,72] Cardiac resynchronization therapy (CRT) may reduce the incidence of electrical storm. Nordbeck and associates[73] retrospectively analyzed the incidence of electrical storm in 561 ICD patients and 168 consecutive patients who had a CRT device and defibrillator (CRT-D). The mean LVEF was 0.22 in the CRT-D group and 0.35 in the ICD group. One CRT-D patient and 39 ICD patients experienced an electrical storm (0.6% vs 7%; $P < 0.01$). The percentage of patients who had isolated episodes of VT or VF and appropriate therapy did not differ between the 2 groups.

The well-documented hemodynamic benefits of CRT include improvements in heart-failure symptoms, exercise capacity, LVEF, and LV volume. In addition, CRT-related reverse remodeling is sustained over the long term in patients with ischemic and nonischemic cardiomyopathy.[74] In CRT recipients, all-cause death is reduced by 40%, heart-failure death by 45%, and sudden death by 46%.[75] The reduction in electrical storm might indicate a CRT-induced improvement in the underlying cardiac gene expression, myocardial substrate, and hemodynamic characteristics; however, further study is warranted.[76]

Conclusion

Electrical storm, an increasingly common and life-threatening emergency, is characterized by 3 or more sustained VT or VF episodes or appropriate ICD shocks within 24 hours. Patients with an electrical storm typically have a poor outcome. The presence or absence of structural heart disease and the ECG morphology of the presenting arrhythmia provide important diagnostic clues to the mechanism of electrical storm. Initial management involves identifying and correcting the underlying ischemia, electrolyte imbalances, or other inciting factors. Amiodarone and β-blockers, especially propranolol, form the cornerstone of antiarrhythmic therapy in most patients.

Nonpharmacologic treatment, including RF catheter ablation, may be implemented in drug-refractory patients. Patients who have ICDs can present with multiple shocks and may require drug therapy and device reprogramming. After the acute phase of an electrical storm, the focus should shift to the maximization of heart-failure therapy, to possible revascularization, and to the prevention of future ventricular arrhythmias.

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