

24 - 263 Electrical Storm and Incessant Ventricular Tachycardia

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■ ■ FURTHER READING Callans DJ: Josephson's Clinical Cardiac Electrophysiology: Techniques

and Interpretations, 7th ed. Philadelphia, Wolters Kluwer, 2024. Jalife J, Stevenson W (eds): Zipes and Jalife's Cardiac Electrophysiology: From Cell to Bedside, 8th ed. Philadelphia, Elsevier, 2022. Zeppenfeld K et al: 2022 ESC Guidelines for the management of PART 6 Disorders of the Cardiovascular System patients with ventricular arrhythmias and the prevention of sudden cardiac death: Developed by the task force for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death of the European Society of Cardiology (ESC) Endorsed by the Association for European Paediatric and Congenital Cardiology (AEPC). Eur Heart J 43:3997, 2022. William H. Sauer, Usha B. Tedrow

Electrical Storm and

Incessant Ventricular

Tachycardia **ELECTRICAL STORM** Electrical storm or ventricular tachycardia (VT) storm refers to the occurrence of three or more episodes of VT or ventricular fibrillation (VF) within 24 h requiring intervention for termination. Although this disorder is uncommon in the general population, it is of great concern to internists because of its rapid course leading to patient death in the absence of treatment. Therefore, prompt recognition and therapeutic intervention are required. Electrical storms occur in 4% of patients with a primary prevention implantable cardioverter-defibrillator (ICD) but in as many as 20% of patients with a history of known VT or resuscitated sudden death. Catheter ablation, antiarrhythmic drug therapy, and other adjunctive therapies in electrical storms can be life-saving. **INCESSANT VT** VT is designated incessant when VT continues to recur shortly after electrical, pharmacologic, or spontaneous conversion to sinus rhythm (Fig. 263-1). Typically, VT is monomorphic. Rarely, a slow incessant monomorphic VT will fail detection by an ICD because it falls outside of the programmed detection parameters. If the arrhythmia is hemodynamically

stable acutely, patients can present with symptoms of gradual cardiac decompensation. VT may become incessant due to the proarrhythmic effect of an antiarrhythmic drug such as amiodarone or a sodium channel blocker such as flecainide. Hemodynamic support may be required acutely until the precipitating factors can be corrected. Urgent catheter ablation is often warranted.

MANAGEMENT OF PATIENTS PRESENTING WITH ICD SHOCKS A substantial number of patients who receive an ICD can be expected to have an arrhythmia that is terminated by the ICD, either by a shock or antitachycardia pacing. Although this is an expected event, it can VT Antitachycardia pacing terminates VT Spontaneous recurrence of VT II FIGURE 263-1 Example of incessant monomorphic ventricular tachycardia (VT). In the initial portion of this electrocardiogram tracing, monomorphic VT is present. A train of antitachycardia pacing (area bracketed by arrows) that is initiated at the fourth VT complex results in ventricular capture with fusion by the eighth beat and termination of VT at cessation of pacing. The patient has underlying atrial fibrillation. Multifocal premature ventricular contractions are present. VT similar in morphology to the initial VT restarts spontaneously toward the latter part of the trace (arrow).

be a sign of impending instability, deterioration of cardiac function, or emergence of a new arrhythmia and therefore requires evaluation. Interrogation of the ICD is crucial after a patient reports a shock or symptoms of arrhythmia to confirm that the therapy was indeed delivered for a ventricular arrhythmia and not for lead malfunction or an atrial arrhythmia. After a shock and in the absence of other symptoms to suggest arrhythmia or ischemia, patients have the option of waiting until the next working day or using remote monitoring to transmit device interrogation data to their physician. However, occurrence of multiple ICD shocks constitutes a medical emergency and warrants immediate medical attention by activating the emergency medical system. Patients should never drive to the hospital themselves after receiving a shock from their ICD. Spontaneous arrhythmias, particularly those that are converted with a shock, are associated with a subsequent increased risk of death and hospitalization in patients with depressed ventricular function. The occurrence of an arrhythmia, therefore, warrants a reevaluation for possible decline in cardiac function, emergence of ischemia, or intercurrent illness. If the ICD therapy is appropriate for VT or VF, consideration is given to whether therapy is warranted to reduce further episodes with either antiarrhythmic drug therapy or catheter ablation. Patients who have a rare episode of VT that is appropriately terminated and who have no other evidence of instability may not need any additional therapy, particularly if the VT is terminated by antitachycardia pacing rather than a shock. Shocks reduce quality of life and can lead to posttraumatic stress disorder. In many patients, the possibility of a shock can be reduced with appropriate ICD programming. Studies have shown that antitachycardia pacing effectively terminates >70% of VT episodes, even when VT is very rapid. Most ICDs can be programmed to attempt overdrive pace termination during capacitor charge. If the arrhythmia then terminates, the shock is aborted. Appropriate programming of antitachycardia pacing is therefore critical for reducing shocks. For patients implanted with ICDs as primary prevention, programming of VF detection zones >220 beats/min significantly reduces unnecessary and inappropriate shocks. Long detection times will also help avoid unnecessary therapies for VT episodes liable to terminate spontaneously. Recurrent symptomatic episodes of VT or VF (Fig. 263-2) warrant specific therapy with antiarrhythmic drugs or ablation as discussed for the specific arrhythmia. The beta blockers sotalol and amiodarone are the most common pharmacologic options. Amiodarone combined with beta blockers is more effective than sotalol or beta blockers alone. It is important to recognize that although VT/VF episodes may represent a deterioration of clinical status in these patients, interventions to control the arrhythmia itself may

FIGURE 263-4 Management algorithm for electrical storm. Shown is a suggested strategy for managing electrical storm based on the underlying rhythm and substrate. CCB, calcium channel blocker; DHP, dihydropyridine; ECMO, extracorporeal membrane oxygenation; IABP, intra-aortic balloon pump; MMVT, monomorphic ventricular tachycardia; PMVT, polymorphic ventricular tachycardia; PVC, premature ventricular contraction; SGB, stellate ganglion block; VF, ventricular fibrillation; VT, ventricular tachycardia.

■ ■VT/VF IN THE SETTING OF MYOCARDIAL ISCHEMIA Ischemia should be considered especially if polymorphic VT or VF is identified as the primary arrhythmia. If electrical storm is occurring in the setting of an acute coronary syndrome, emergent revascularization and alleviation of anginal symptoms should be attempted. Within the infarcted myocardium, surviving Purkinje cells can exhibit triggered automaticity and lead to recurrent episodes of polymorphic VT/VF requiring frequent cardioversions before and after revascularization. Catheter ablation of premature ventricular contractions (PVCs) that are observed to repeatedly initiate the arrhythmia can be effective (Fig. 263-5). • Beta blockers • Sedation and intubation • Anxiolytics • Stellate ganglion block (SGB) • Cardiac surgical sympathetic denervation ■ ■PVC-INITIATED POLYMORPHIC VT/VF

Similar to the post-myocardial infarction electrical storm, patients without myocardial infarction or ischemia can have PVC-initiated polymorphic VT/VF storm. This idiopathic form of VF is usually caused by triggering PVCs originating from fascicular tissue or papillary muscles. Often, the ventricular ectopy is from scarred myocardial tissue detected on cardiac magnetic resonance imaging. Catheter ablation is indicated for this condition when antiarrhythmic medication is ineffective. ■ ■ACQUIRED OR CONGENITAL LONG QT SYNDROME If QT prolongation causing torsades des pointes (TdP) is possible, intra venous magnesium should be administered for its immediate effect on repolarization. In addition, electrolyte repletion, especially potassium, should be aggressively pursued. Increasing the heart rate can some times normalize the QT interval, and thus, pharmacologic or pacing support should be considered. Isoproterenol can be used to increase a patient's sinus rate, but there is the possibility of increased ectopy with high doses of isoproterenol possibly exacerbating ventricular Beta blockade Amiodarone Lidocaine SGB Sedation Catheter ablation Quinidine Non-DHP CCB Isoproterenol Catheter ablation Steroid pulse Amiodarone SGB

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