EDITORIAL COMMENT

Inside or Out? Another Option for Incessant Ventricular Tachycardia*

William G. Stevenson, MD, FACC, Kyoko Soejima, MD
Boston, Massachusetts

Sustained monomorphic ventricular tachycardia (VT) is most commonly due to reentry in a region of prior myocardial infarction or ventricular scar. It may be hemodynamically tolerated, or if rapid, produce hemodynamic collapse with syncope or cardiac arrest. The vast majority of cases are paroxysmal. Following termination of tachycardia by cardioversion or antiarrhythmic drug administration, stable sinus rhythm returns and persists. Recurrences are common, however, with more than a 20% of patients experiencing another episode within two years, even if antiarrhythmic drug therapy is utilized (1,2). An implantable cardioverter-defibrillator (ICD) is an excellent option for many patients, providing effective termination of the arrhythmia, often with a burst of antitachycardia pacing, or failing that, a shock.

See page 2036

Ventricular tachycardia that repeatedly recurs and persists for more than half of a 24-h period despite repeated attempts to terminate the arrhythmia is designated “incessant.” Depending on the rate and associated heart disease, the clinical consequence ranges from asymptomatic elevation in heart rate to death. Rarely, idiopathic VT that is not associated with structural heart disease is incessant (3). The most common type originates from the right ventricular outflow tract, causing a left bundle branch block, inferior axis configuration VT. It may be relatively slow and asymptomatic, or cause palpitations or symptoms of hypotension. Incessant idiopathic VT can cause tachycardia-induced cardiomyopathy if not recognized and treated (4). More commonly, incessant VT is due to structural heart disease, with severe, immediate consequences.

Incessant VT typically takes one of two forms. The most common situation is for VT to be sustained, terminated by external cardioversions, but recurrent. The time between cardioversion and recurrence may be seconds, minutes or more. A second form, common with the idiopathic VTs, manifests as repeated bursts, with runs of VT that spontaneously terminate for a few intervening sinus beats, followed by the next tachycardia burst (3). Cardioversion is futile, but may be periodically required if bursts of VT occasionally degenerate to ventricular fibrillation.

Often incessant VT evolves from an episode of monomorphic VT. Following termination of an episode of VT, an antiarrhythmic drug, commonly intravenous amiodarone, may be administered (5). Repeated episodes of VT after antiarrhythmic drug administration may simply indicate that the drug is ineffective. It is important, however, to recognize the possibility of a drug induced proarrhythmic effect, which may take the form of slowing the VT, but rendering it incessant. Such proarrhythmia was well recognized during therapy with antiarrhythmic drugs that prominently slowed conduction velocity, such as flecaïnid, propafenone, and moricizine (6,7). Treatment is directed at maintaining hemodynamic support until the drug is excreted. This type of proarrhythmia may respond favorably to a beta-adrenergic blocking agent or lidocaine (7). In general administration of additional antiarrhythmic drugs should be avoided, however, because they may exacerbate the arrhythmia or hemodynamic tolerance of the tachycardia.

When incessant VT is polymorphic, drug induced torsade de pointes associated with QT prolongation, or myocardial ischemia are the major concerns (8). Runs of polymorphic VT may even repeatedly initiate monomorphic VT in patients with reentry circuits in regions of scar. Suppression of torsade de pointes with intravenous administration of magnesium sulfate and/or overdrive pacing may restore stability. Coronary angiography and intervention for an acute ischemic syndrome may be required.

Sedation and measures to reduce sympathetic tone are important, and often effective (9). If episodes of arrhythmia are not precipitated by slow heart rates or pauses, administration of beta-adrenergic blockers should be considered. General anesthesia quiets episodes and restores stability in some cases. Intra-aortic balloon counter-pulsation can also be helpful (10). When incessant VT associated with structural heart disease is not due to a reversible cause or when hemodynamic stability cannot be maintained such that the underlying cause can be treated, the outcome is often death.

Catheter ablation is an important option for management of incessant monomorphic VT and can be life-saving (11,12). When hemodynamic stability can be maintained, presence of the tachycardia facilitates mapping to identify the source of the arrhythmia. Ablation typically terminates the arrhythmia, restoring hemodynamic stability. Most scar related reentry circuits in patients with prior myocardial infarction are located on the endocardium and are susceptible to catheter ablation from an intravascular, endocardial approach (13). However, 10% to 20% of patients have reentry circuits that are not successfully ablated. The location of some circuits deep to the endocardium or in the epicardium, are important causes for failure. For patients with incessant VT, remaining options then include arrhythmia surgery, placement of a ventricular assist device, or cardiac transplantation (14).

In 1996, Sosa et al. (15) reported a percutaneous catheter technique for mapping the epicardium from the pericardial space in patients with ventricular tachycardia due to Chagas...
disease. They subsequently demonstrated, consistent with observations made during surgery by Svenson and coworkers, that some VTs due to prior inferior wall infarction can be ablated with this epicardial approach (16,17).

In this issue of the Journal, Brugada et al. (18) report their experience with epicardial catheter ablation for incessant VT. Of all patients undergoing ablation at their center, 19, approximately 7%, had incessant VT. An epicardial approach was attempted in 10 patients, either because endocardial ablation failed or endocardial mapping was precluded by presence of left ventricular thrombus or peripheral vascular disease. Incessant VT was successfully ablated from the epicardium in 8 of 10 patients.

Several caveats should be noted. Catheter ablation of scar related VT remains more challenging than ablation of supraventricular tachycardias, and experience outside of large, experienced centers is limited. Safety in the small reported series of epicardial catheter ablation has been quite good. Hemopericardium requiring drainage has occurred. Transient pain consistent with pericarditis after the procedure is not uncommon. Damage to epicardial coronary arteries, the left phrenic nerve, and adjacent lung are potential concerns that warrant careful consideration during the procedure. Coronary angiography to assess proximity to large epicardial vessels is usually performed. In some patients pericardial fibrosis from prior cardiac surgery may prevent percutaneous access to the pericardial space. A surgical approach to the epicardium may be a reasonable consideration for some of these patients, when expertise in arrhythmia surgery is available.

Questions remain regarding management after ablation. The majority of patients with scar-related VTs have more than one potential reentry circuit and many morphologies of monomorphic VT inducible with electrophysiologic testing (12). After ablation of incessant VT, ablation of other VTs is often considered. The long-term follow-up after ablation is still being defined. In series where ablation targets are stable, “mapable VTs” after myocardial infarction, more than 20% of patients experience a recurrence after initially successful ablation (12,19). Whether recurrences will be diminished with more extensive ablation approaches targeting regions of scar, remains to be assessed during long-term follow-up (13). Although most recurrences are not fatal, the excellent efficacy of ICDs for terminating recurrences with antitachycardia pacing and the potential for avoiding antiarrhythmic drug therapy make device implantation an attractive option. In the present series, only one of the eight patients experienced a late recurrence after initially successful ablation with follow-up ranging from 2 to 46 months.

Catheter ablation can be a lifesaving therapy for patients with incessant VT. Brugada et al. (18) have shown that epicardial ablation can be a life-saving option when endocardial ablation fails or cannot be attempted. Due to the technical challenges of catheter ablation for VT and the relatively low incidence of this problem, this therapy will largely remain in experienced centers. As shown by Brugada et al. (18), application of Dr. Sosa’s pioneering work in percutaneous mapping and ablation from within the pericardial space provides an important option for controlling this potentially fatal arrhythmia.

Reprint requests and correspondence: Dr. William G. Stevenson, The Cardiovascular Division, Brigham and Women’s Hospital, 75 Francis Street, Boston, Massachusetts 02115. E-mail: wstevenson@partners.org.

REFERENCES