Automatic Burst Extrastimulus Pacemaker to Treat Recurrent Ventricular Tachycardia in a Patient With Mitral Valve Prolapse: More Than 2,000 Documented Successful Tachycardia Terminations*

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An automatic burst pacemaker (Cybertach-60) was used to treat drug-resistant recurrent ventricular tachycardia in a patient with mitral valve prolapse. The arrhythmia was associated with multiple syncopal episodes and aborted sudden death. During a 23 month follow-up period the patient has remained asymptomatic and more than 2,000 episodes of ventricular tachycardia have been successfully terminated by the pacemaker.

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Various permanent pacemaking methods have been used in the management of patients with recurrent ventricular tachycardia (1–8). Three general types of antitachycardia devices are available: 1) underdrive, 2) scanning, and 3) burst pacemakers. Underdrive pacemakers deliver random stimuli, and thus have limited value in treating hemodynamically significant tachyarrhythmias. We, along with other investigators (9–12), have successfully used permanent scanning pacemakers to treat sustained supraventricular and ventricular tachycardias. Burst pacemakers have also been therapeutically useful, but acceleration of ventricular tachycardia or induction of ventricular fibrillation, or both, has been frequently reported (1,3,5-8,13).

This report outlines the successful use of an automatic burst pacemaker (Cybertach-60, Intermedics, Inc) to treat recurrent symptomatic ventricular tachycardia in a patient with mitral valve prolapse whose condition had failed to improve with the use of conventional and investigational medications. At 23 months after implantation, the patient remains asymptomatic and ambulatory electrocardiographic (Holter) recordings have demonstrated more than 2,000 successful terminations of ventricular tachycardia.

Case Report

History. A 36 year old woman was diagnosed as having mitral valve prolapse in October 1979 after complaining of episodes of palpitation. In February 1980 she had a syncopal episode and an electrocardiogram revealed wide complex tachycardia at 180 beats/min (Fig. 1). Over the next several months, adverse side effects necessitated discontinuation of quinidine, disopyramide and propranolol. In January 1981, a second syncopal episode occurred while she was taking metroprolol, and procainamide was added. Several episodes of near syncope subsequently occurred, resulting in hospitalization in June 1981. During hospitalization, while taking an increased procainamide dosage, the patient had monitored wide complex tachycardia at 230 beats/min, hemodynamic collapse and subsequent cardiopulmonary resuscitation, requiring ventilatory support for several days. In August 1981, mexiletine therapy was started. For the next 2 1/2 years the patient had mild episodes of palpitation, and 20 ambulatory electrocardiographic (Holter) recordings revealed frequent ventricular premature depolarizations, including ventricular couplets and triplets but no higher repetitive forms. In March 1984, multiple near syncopal episodes resulted in another hospitalization at our institution.

Physical examination and laboratory evaluation. The cardiac examination was normal except for a loud midsystolic click without murmur. Blood chemistry values, chest X-ray film and the baseline electrocardiogram were normal. Echocardiography demonstrated mitral valve prolapse but normal chamber size and function. Three separate exercise stress tests (including one thallium stress test) using the standard Bruce protocol showed no electrocardiographic changes, angina, arrhythmias or perfusion abnormalities.

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Figure 1. Twelve lead electrocardiogram obtained in February 1980 during the patient's emergency room visit for syncope. The ventricular tachycardia is slightly irregular at a rate of 175/min. This same 12 lead configuration for ventricular tachycardia was recorded spontaneously several times and was induced more than 300 times in the electrophysiology laboratory before pacemaker implantation. Standard electrocardiographic leads are labeled.

Holter ambulatory electrocardiographic recordings demonstrated multiple episodes of sustained ventricular tachycardia (Fig. 2), each associated with near syncope. Electrocardiographic 12 lead recordings during symptoms were identical to that in Figure 1. All medications were discontinued but symptoms and tachyarrhythmias continued. The patient refused multiple experimental medications including amiodarone. Because she wished to consider antitachycardia pacemaker therapy, an electrophysiologic evaluation was performed.

Electrophysiologic testing. An electrophysiologic study was performed utilizing standard techniques. Sinus node and atrioventricular (AV) node function were normal, and neither atrial overdrive pacing nor programmed stimulation induced atrial supraventricular arrhythmia. Monomorphic sustained ventricular tachycardia was repeatedly induced by double extrastimuli at the right ventricular apex. The induced ventricular tachycardia was identical in configuration to that observed clinically. Single or double stimuli, or both, delivered at the right ventricular apex were unsuccessful in terminating ventricular tachycardia. However, timed burst pacing with six stimuli at 250 ms terminated 100 consecutive episodes of ventricular tachycardia without rate acceleration (Fig. 3).

Pacemaker implantation. After the electrophysiologic test, the patient again refused therapy with several investigational antiarrhythmic medications, but preferred an implantable antitachycardia pacemaker. Institutional approval for implantation of the Cybertach-60 was obtained. The patient returned to the electrophysiology laboratory where ventricular tachycardia was reproducibly induced utilizing coupled extrastimuli at the right ventricular apex. After 100 consecutive episodes of ventricular tachycardia were successfully terminated utilizing a burst rate of 240 pulses/min for 1.3 seconds, the pacemaker was implanted and an additional 50 episodes of ventricular tachycardia were induced

and successfully terminated. The patient had an uneventful recovery from pacemaker implantation.

Cybertach-60 pacemaker. The Cybertach-60 pacemaker is one of the first automatic implantable pacemakers available for antitachycardia pacing in which both tachycardia identification criteria and terminating responses are programmable after implantation. The tachycardia recognition algorithm is simple, recognizing a heart rate exceeding the programmed rate. Thus, any eight consecutive cycles at a rate greater than the programmed tachycardia criteria will result in burst pacing output from the pacemaker. The

Figure 2. Segment of a full disclosure Holter electrocardiographic recording demonstrating three self-terminating episodes of sustained ventricular tachycardia, each associated with presyncope. The tachycardia rate is approximately 165/min. Each line of recording corresponds to approximately 25 seconds.





Figure 3. Electrophysiologic study. A, Recording showing ventricular tachycardia after induction by double extrastimuli. Atrioventricular dissociation is present. The first of six paced stimuli (p) is synchronized to the last ventricular complex without appreciable change in the surface electrocardiographic configuration. **B**, Continuation of the tracing from **A**. Pacing stimuli (p) continue at 250 ms intervals until six are given. The first pacing stimulus in this panel changes the surface electrocardiographic configuration, and when pacing is discontinued, sinus rhythm resumes. More than 250 successful terminations were documented in the electrophysiology laboratory. HRA = high right atrial intracardiac lead; RV = right ventricular intracardiac lead.

choice of tachycardia criteria is limited to two, although different rate criteria can be manufactured by request. Terminating responses are programmed by burst rate and duration. Burst rates from 180 to 1,440 pulses/min (eight different settings) and burst duration from 0.33 to 5.3 seconds (five steps) are available. Programmability of pulse width, sensitivity, bradycardia stimulation rate and telemetry are also available.

Follow-up. The patient has been observed for 23 months since pacemaker implantation. She has remained asymptomatic without dizziness, near syncope or syncope and has undergone thirty 24 hour Holter monitor recordings, confirming 2,014 successful pacemaker terminations of ventricular tachycardia (Fig. 4A and B). She is taking nadolol, 40 mg/day, which maintains her sinus rate below the programmed tachycardia criteria.

Discussion

Antitachycardia pacemakers. Continuous "overdrive pacing" was reported in 1964 (14) as efficacious for suppressing ventricular arrhythmias. Fixed rate "underdrive





Figure 4. A, Segment of a full disclosure Holter electrocardiographic recording after implantation of the Cybertach-60 pacemaker. Four episodes of ventricular tachycardia termination (arrows) are shown. As in Figure 2, each line of recording corresponds to 30 seconds. B, Dual channel recording of one episode of ventricular tachycardia (VT) from A. The ninth beat of ventricular tachycardia (eight cycle lengths) starts a sequence of six paced beats (pace) at 250 ms intervals, resulting in termination of ventricular tachycardia and resumption of sinus rhythm. (The first stimulus occurs at the onset of the QRS complex of the last tachycardia beat.) More than 2,000 terminations have been recorded on Holter recordings.

pacing" has been applied in supraventricular and ventricular arrhythmias (15). This pacing mode is useful for arrhythmias that are not hemodynamically compromising and can be terminated by single premature stimuli. Its success rate can be increased by concomitant use of antiarrhythmic medications that slow conduction or lengthen the functional refractory period of the reentrant circuit (6).

Scanning pacemakers have been shown to terminate ventricular tachycardia by delivering timed stimuli (8–13). Radio frequency externally activated scanning pacemakers were followed by automatically activated devices. We recently reported (11) the successful use of a programmable automatic scanning pacemaker to treat ventricular tachycardia. The use of scanning single or double extrastimuli for terminating tachyarrhythmias appears to be safe, rarely causing rate acceleration or induction of ventricular fibrillation.

Burst stimuli have been reported to be useful in longterm therapy of ventricular tachycardia (1-7,13). This mode of antitachycardia pacing can be activated manually, by radio frequency signal or automatically. A burst rate at least 30% greater than the tachycardia rate is generally needed to terminate the arrhythmia (1-16,17). The mechanism whereby burst stimuli terminate ventricular tachycardia has not been established, although several mechanisms have been proposed. The use of this method of treating ventricular tachycardia has been limited by reports of acceleration of tachycardia in more than 40% of patients studied in electrophysiology laboratories (1,18). Thus, investigators have required at least 100 successful arrhythmia terminations without rate acceleration before pacemaker implantation (1-5).

Therapeutic considerations. Our patient had recurrent, hemodynamically significant ventricular tachycardia. Adverse side effects or ineffectiveness, or both, limited therapy with conventional antiarrhythmic agents alone or in combination with a beta-adrenergic blocking agent. While taking procainamide the patient had an in-hospital cardiac arrest, probably secondary to drug-induced heart rate acceleration, although the QT interval was normal and torsade de pointes was not noted (single channel lead). The experimental agent mexiletine was successful in eliminating symptoms and Holter-recorded sustained ventricular tachycardia for more than 2 years, but symptomatic ventricular tachycardia eventually recurred. The patient refused several other investigational antiarrhythmic medications including amiodarone, and an electrophysiologic study was performed to test the efficacy of antitachycardia pacing modalities.

Electrophysiologic studies utilizing double extrastimuli at the right ventricular apex reproducibly induced ventricular tachycardia. Single and double pacing stimuli delivered from this site at multiple different coupling intervals were successful in terminating 50% of induced ventricular tachycardias. Synchronizing the first of six pacing spikes delivered at 240 pulses/min with the last sensed ventricular complex, more than 250 successful terminations of ventricular tachycardia were demonstrated before implantation of the Cybertach-60 generator. Combinations of longer or faster burst modes, or both, were also successful, without rate acceleration. It was thought important to test other modes of tachycardia termination so that further reprogramming, if needed, could be performed safely.

Follow-up. Thirty Holter ambulatory electrocardiographic recordings over a 23 month follow-up period have demonstrated 2,014 successful terminations of ventricular tachycardia. No evidence for rate acceleration is present although 5 of the 2,014 terminations required a second burst for termination. Reprogramming of the burst rate or duration has not been needed because the patient remains asymptomatic without dizziness, presyncope or syncope. During maintenance treatment with nadolol, 40 mg/day, multiple exercise tests after pacemaker implantation have shown no abnormality and demonstrate a maximal sinus rate of less than 137 beats/min, the programmed tachycardia recognition criterion.

Although the number of Holter recordings may seem excessive, the investigational protocol as approved by the hospital's internal review board stipulated at least two Holter recordings/month for the first year after pacemaker implantation. In this particular medical care setting, these Holter readings added little expense other than paper costs. Additionally, because the patient was a volunteer at the hospital, time and transportation costs were not overriding considerations.

Conclusions. This report outlines the use of a burst antitachycardia pacemaker in a patient with mitral valve prolapse and symptomatic ventricular tachycardia. Before pacemaker implantation the patient had multiple presyncopal and syncopal episodes, and one episode of aborted sudden death. Conventional medications and one investigational antiarrhythmic medication were therapeutically unsuccessful. After pacemaker implantation, the patient is asymptomatic with more than 2,000 Holter-documented pacemaker terminations of ventricular tachycardia.

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