Termination and Inhibition of Recurrent Tachycardias by Implanted Pervenous Pacemakers

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SUMMARY
Three patients are described with recurrent paroxysmal tachycardia who required implanted pervenous demand pacemakers for the control of repetitive tachyarrhythmias. One patient had frequent weekly episodes of life-inhibiting supraventricular tachycardia (SVT) lasting hours to days and refractory to antiarrhythmic drugs. A coronary sinus atrial demand pacemaker at 72 beats per minute was implanted. During the subsequent two months, the patient quickly terminated nine episodes of SVT by activating the generator with an external magnet and competitively pacing the atrium. The second patient had frequent daily episodes of SVT and secondary angina despite vigorous antiarrhythmic medication. Demand ventricular pacing at a rate of 88 beats per minute prevented the recurrence of SVT during the seven month follow-up. A third patient with an old myocardial infarction had frequent biweekly episodes of ventricular tachycardia (VT) refractory to conventional treatment. A ventricular demand pacemaker was implanted, and three episodes of VT were terminated by magnetically activated competitive ventricular pacing at 64 beats per minute. Augmentation of the demand pacemaker rate to 86 beats per minute inhibited the recurrence of VT during a five month follow-up. The rates of the tachycardias were less than 160 beats per minute in all three cases, and evidence suggests a re-entrant or reciprocating mechanism for the tachyarrhythmias. The electrophysiologic rationale for pacemaker therapy in certain types of recurrent SVT and VT is discussed.

Additional Indexing Words:
Supraventricular tachycardia
Coronary sinus pacemaker
Ventricular tachycardia
Re-entrant tachycardia
Therapy of tachyarrhythmias
Reciprocating tachycardia

In the past few years, the underlying mechanisms of certain types of supraventricular and ventricular tachycardias have been clarified by electrophysiologic studies, and the essential role of re-entrant or reciprocating circuits has been demonstrated. Recently, magnetically induced ventricular "demand" pacing has been utilized in the termination of intermittent supraventricular tachycardia (SVT) in patients with and without the Wolff-Parkinson-White syndrome. This method converts SVT to sinus rhythm by retrograde depolarization of the reciprocating circuit within the re-excitation pathway. We utilized this magnetically-induced competitive pacing technique for the termination of recurrent tachycardias in three patients. In the course of the patients' management with demand pacemakers, we observed that the prevention of the recurrent tachycardias was influenced by the demand pacing rate. The successful application of demand atrial or ventricular pacemakers in the antiarrhythmic therapy of SVT and ventricular tachycardia (VT) is the subject of this communication.

Case Reports

Case 1
C.O. (SMH #86-24-91) was a 63-year-old woman with a history of paroxysmal SVT since age 17 years. Over the years, the episodes of SVT had gradually increased in frequency and duration, and since 1972, the patient had been incapacitated by tachycardia episodes occurring two to three times a week lasting from nine hours to three days. The SVT usually occurred at a rate of 150 beats per minute, and the patient developed extreme fatigue and weakness with the prolonged rapid heart action, but no angina or syncope. Repeated clinical evaluations had revealed no evidence of organic heart disease, pre-excitation conduction, or sinus bradycardia. Antiarrhythmic therapy with full doses of digoxin, quinidine, procaine amide, propranolol, and diphenylhydantoin, singly and in various combinations, had been tried, but without beneficial influence on the recurrent SVT.

During an episode of SVT in February 1973, a Goetz pacing electrode catheter was advanced into the right ventricle. The SVT could not be terminated by asynchronous ven-
tricular pacing at rates ranging from 60 to 120 beats per minute. When the electrode was withdrawn to the right atrium, asynchronous atrial pacing immediately terminated the SVT. A permanent pacemaker was not implanted at this time.

Recurrent SVT persisted throughout the next year despite further drug manipulations. The patient was transferred to the Strong Memorial Hospital and on February 5, 1974, a pervenous coronary sinus atrial demand pacemaker was implanted using a Medtronic 5818 bipolar electrode and a 5842 long refractory period generator. An arbitrary atrial pacing rate of 94 beats per minute was selected. The threshold for atrial pacing was high at 5 mA. During the next three days three episodes of recurrent SVT were promptly converted to normal sinus rhythm (NSR) by activating the demand generator into asynchronous mode by an external magnet. Intermittent failure to pace the atrium developed, and on February 10, 1974, the pacemaker generator was disconnected and a high atrial pacing threshold of 10 mA was documented. Asynchronous atrial pacing with an external generator repeatedly induced SVT, and the episodes were quickly terminated by atrial pacing at a rate of 72 beats per minute (fig. 1). A Medtronic high output (18 mA) demand generator (model 1317A) was substituted for the 5842 generator, and the factory set pacing rate of 72 beats per minute was utilized.

During the two months since the new generator was implanted, the patient has terminated nine episodes of SVT by magnetic activation of the demand generator into the asynchronous pacing mode. Each episode was converted to NSR within a minute of application of the magnet over the generator. The patient has not experienced any sustained episodes of SVT in the two months since the pacemaker was implanted — the longest interval in ten years without prolonged tachycardia.

Case 2

A.H. (SMH #45-50-59) was a 72-year-old woman with a 50-year history of well-documented, recurrent SVT. In 1969, she developed an antero-septal myocardial infarction and her subsequent clinical course has been complicated by exertional angina. In the six-month period from March 1973, to August 1973, the patient experienced frequent daily episodes of SVT despite vigorous antiarrhythmic regimens with digoxin, quinidine, procaine amide, and propranolol, singly and in various combinations to the point of toxicity. Numerous hospitalizations were required during this six-month period for termination of arrhythmias and for management of tachycardia-induced myocardial ischemia.

Emergency department evaluation during a typical acute attack on September 10, 1973, revealed a blood pressure of 100/80 mm Hg, regular pulse at 140 beats per minute. Conversion from SVT to sinus rhythm occurred spontaneously and subsequent electrocardiograms revealed sinus rhythm at 60-70 beats per minute, a normal P-R interval of 0.16 seconds (fig. 2, top panel), an old antero-septal myocardial infarction, and ST and T wave changes indicative of acute and chronic ischemia.

On September 13, 1973, a pervenous electrode was advanced to the right atrium, and during catheter manipulation into the coronary vein, an episode of SVT at a rate of 140 beats per minute was induced mechanically (fig. 2, mid-

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**Figure 1**

Onset and offset of SVT in Case 1. Top) sinus rhythm at 78 beats per minute with P-R = 0.16 sec. Middle) asynchronous atrial pacing at 72 beats per minute (dark lines); the fourth atrial pacemaker stimulus initiates an episode of SVT at 130 per minute. Bottom) asynchronous atrial pacing at 72 beats per minute during SVT; the fourth atrial pacemaker stimulus which falls 0.30 sec after the preceding QRS complex terminates the tachycardia and thereafter paces the atrium. Pacemaker stimuli darkened for clarity.

**Figure 2**

Rhythm strip electrocardiograms in Case 2. Top) sinus rhythm at 75 beats per minute with P-R = 0.16 sec. Middle) SVT at 140 beats per minute induced mechanically during catheterization. Bottom) Ventricular paced rhythm (VPR) at a rate of 88 beats per minute.
the first of a series of episodes of VT requiring hospitalization for pharmacologic or electrical cardioversion. At first these episodes occurred twice a year and he appeared to benefit from antiarrhythmic therapy with quinidine and propranolol, and later with procaine amide. Between June and September, 1973, the patient had biweekly episodes of VT necessitating repeated cardioversions and hospitalizations. The VT occurred at various rates which ranged from a maximum of 160 beats per minute to a minimum of 110 beats per minute when he was on large doses of antiarrhythmic agents. During his recurrent admissions with tachycardia, the diagnosis of VT was strongly favored by: 1) the markedly aberrated configuration and the divergent 0.02 sec vector of the QRS complexes during slow tachycardia (fig. 3A) when compared to the pattern in sinus rhythm (fig. 3B); 2) a monophasic upright QRS pattern in V1 during the tachycardia; 3) atrioventricular (A-V) dissociation demonstrated by intra-atrial recordings during the tachycardia (fig. 4); 4) the absence of any response to carotid sinus pressure or edrophonium; 5) the prompt conversion of the tachycardia to sinus rhythm by intravenous lidocaine; and 6) the inability to precipitate the tachycardia by single and paired atrial pacing stimuli which scanned the entire atrial cycle when he was in sinus rhythm.

On September 11, 1973, when the patient was in VT at 115 beats per minute, a pervenous electrode was passed to the right atrium and A-V dissociation documented (fig. 4). Atrial pacing rates ranging from 60 to 150 beats per minute did not terminate the VT. When the electrode was advanced to the right ventricle, mechanical contact with the endocardium immediately terminated the tachycardia. As a result of this experience, a permanent Medtronic pacemaker system was implanted with a 5818 bipolar electrode in the right ventricular apex connected to a 5842 generator set at a demand pacing rate of 64 beats per minute.

The patient was placed on propranolol and quinidine and he remained asymptomatic until October 15, 1973, when he presented in the emergency department with another episode of VT at a rate of 120 beats per minute with a blood pressure of 90/60 mm Hg. Magnetic induction of the demand pacemaker at a rate of 64 beats per minute converted

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**Figure 3**

Twelve lead electrocardiograms in Case 3. A) ventricular tachycardia at a rate of 110 beats per minute. B) sinus rhythm at a rate of 75 per minute with old anterior myocardial infarction.
the VT to sinus rhythm with 15 seconds of pacemaker activation. The paced beat which terminated the VT occurred 0.45 sec after an intrinsic ventricular depolarization. Another episode of VT at 115 beats per minute occurred on October 28, 1973. A single magnet-induced pacemaker beat which fell 0.44 sec after an intrinsic VT beat converted the VT to sinus rhythm (fig. 5). An additional episode of VT at 128 beats per minute occurred on November 11, 1973. Magnetic activation of the pacemaker produced spike potentials at 64 beats per minute which consistently occurred in synchrony with every other QRS depolarization of the intrinsic tachycardia despite numerous “on-and-off” applications of the magnet over the generator. Using a Keith needle, the generator rate was increased to 86 beats per minute and magnetic activation of the pacemaker once again converted VT to sinus rhythm. In the five months since the rate was increased, the patient has not experienced any episodes of recurrent VT. All antiarrhythmic agents have been withdrawn.

**Discussion**

Two of the three cases are unique in that pacing was successful not only in terminating SVT and VT but also in inhibiting the recurrence of these tachyarrhythmias. Although the follow-up periods are relatively short, the change from very frequent episodes of tachycardia prior to pacemaker implantation to the complete absence of all episodes in the seven and five months after demand pacing in cases 2 and 3 is dramatic.

In the two patients with recurrent VT, the tachycardias most probably developed within a reciprocating circuit in the A-V node. Dual fast and slow pathways in the A-V node are thought to be responsible for the re-entrant circuit. In Case 1, competitive atrial pacing terminated the VT, but ventricular pacing did not; the reverse occurred in Case 2. A critical relationship exists between the pacing site, the rate of the tachycardia, and duration of the refractory period in the tissue between the pacing site and the location of the reciprocating circuit. Left atrial pacing from the coronary sinus and right ventricular pacing were required for access to the reciprocating circuits in the A-V node in cases 1 and 2, respectively. Inhibition of recurrent VT by ventricular demand pacing at 88 beats per minute in Case 2 was a serendipitous result.

In the case 3, the diagnosis of the ventricular origin of the tachycardia appears adequately substantiated by the clinical and the electrophysiologic observations which were made. Although junctional tachycardia with A-V dissociation and aberrant QRS cannot be excluded, it appears highly unlikely. The VT most probably originated from an area adjacent to the old anterior myocardial infarction. Two mechanisms can be postulated in the production of VT — either a rapidly discharging focus or a re-entry circuit. Although termination of the VT by a single right ventricular paced beat is consistent with either mechanism, the failure of the VT to recur immediately after termination favors re-entrant mechanism. Recently, electrophysiologic studies of VT have suggested that reciprocating circuits could lie either in the bundle branches, Purkinje fibers with or without adjacent myocardium, infarcted, or fibrotic tissues, or combinations of these. If a

![Figure 4](image1)

**Figure 4**

Atrioventricular dissociation in Case 3. Top) lead II with VT at a rate of 115 beats per minute. Bottom) intra-atrial electrocardiogram (IAE) with atrial rate (sharp negative deflections) 78 beats per minute and ventricular rate (rounded positive deflections) 115 beats per minute. The recordings were obtained sequentially on a single channel recorder and do not represent simultaneous tracings on a multichannel system.

![Figure 5](image2)

**Figure 5**

Sequential but noncontinuous lead II rhythm strips before, during, and after conversion from VT to sinus rhythm in Case 3. Top) VT at 115 beats per minute with apparent retrograde P waves. Middle) a single pacemaker-induced beat (arrow) converts the VT to sinus rhythm. An external magnet was briefly applied to the generator and immediately removed to produce the isolated pacemaker stimulus. Bottom) sinus rhythm at 90 beats per minute.
reciprocating circuit is responsible for VT in case 3, then an appropriately timed ventricular paced beat may enter the re-entrant pathway and render the circuit refractory to further reciprocation, thus terminating the tachycardia. Inhibition of recurrent VT by maintaining a minimum rate of 86 beats per minute with a demand ventricular pacemaker was a fortuitous effect. The demand pacemaker rate was increased from 64 to 86 beats per minute because the slower pacemaker rate, which was exactly half the rate of the manifest tachycardia, did not "scan" the cardiac cycle when the generator was activated by the external magnet. The faster rate quickly terminated the VT and inhibited the recurrence of subsequent VT episodes.

Application of implanted pacemakers for the control of tachycardias is a relatively new field. Ryan et al. described the "paradoxical use of a demand pacemaker" for competitive pacing to terminate recurrent episodes of reciprocating SVT in WPW. More recently, Kitchen et al., Coumel, and Wellens have utilized the same approach for converting intermittent SVT to NSR in patients without pre-excitation. Both Coumel and Wellens believe that the effectiveness of competitive ventricular pacing in terminating paroxysmal tachycardias is inversely related to the rate of the arrhythmia. In our three patients, the supraventricular and ventricular tachycardias were relatively slow, with rates less than 160 beats per minute. At rates below this level, single paced beats are usually able to enter re-entrant conduction pathways thus terminating the tachycardia. At faster rates, a refractory period entrance block may develop around the re-entrant circuit from the very rapid and repetitive depolarization. In such cases, paired stimuli are required to "peel back" the refractory period to permit the second paced beat to enter the re-entrant circuit and terminate the tachycardia. Although competitive ventricular pacing could produce ventricular fibrillation, the latter did not occur in either of our two cases when asynchronous ventricular pacing was applied during the tachycardia. Because of the potential danger of inducing repetitive ventricular arrhythmias with competitive ventricular pacing, this approach should be reserved for only the most refractory and intractable cases, with in-hospital observation during the initial trials with magnetically-induced competitive pacing.

Inhibition of recurrent paroxysmal supraventricular and ventricular tachycardias in cases 2 and 3 by ventricular demand pacing at rates in the mid-80s deserves special comment. Previous investigators have utilized atrial or ventricular tachypacing at rates above 100 beats per minute to prevent rapid life-threatening tachyarrhythmias. In our patients the rates of the paroxysmal tachycardias were not excessively fast, i.e., less than 160 beats per minute, and these slower tachycardia rates may identify a subgroup of patients in whom less rapid pacing rates may be effective in preventing recurrent tachycardic episodes. The demand pacemaker rates of 86 and 88 beats per minute were chosen somewhat arbitrarily. In case 2, a ventricular demand pacing rate of 88 beats per minute prevented the recurrent SVT. In case 3, the initial pacing rate of 64 beats did not prevent recurrent VT, but the faster pacing rate of 86 beats per minute has been dramatic in the elimination of all further episodes of VT. The authors are at a loss to adequately explain why demand pacing rates in the mid-80 per minute range prevented the recurrent tachycardias. Possible electrophysiologic explanations include: 1) shortening of the time interval during which the re-entrant circuit is able to respond to an initiating stimulus; 2) prevention of long cycle lengths which may predispose the re-entrant circuit to reciprocating conduction by a premature beat; 3) reduction in the temporal dispersion of excitability in the reciprocating circuit; and 4) "overdrive suppression" of ectopic premature depolarization which may initiate the tachycardias.

Wellens has pointed out that the site of pacemaker stimulation may be critical in the control of tachycardias. The pacemaker-initiated depolarization must involve the re-entrant circuit for it to be effective in tachyarrhythmia termination and prevention. In the two patients with SVT, competitive atrial pacing was required in one and ventricular pacing in the other to terminate the tachycardias. In case 3, atrial pacing was ineffective in terminating VT, but ventricular pacing was successful. Wellens describes a similar patient with VT that could not be controlled by atrial pacing at 110 beats per minute with 1:1 A-V conduction, yet ventricular pacing at 85 beats per minute completely suppressed the tachycardia.

In summary, many interrelated variables determine the effectiveness of competitive and minimal rate demand pacing in the termination and prevention of recurrent tachycardia. The pertinent variables include the mechanism, anatomic location, and intrinsic rate of the tachycardia relative to the site, rate, and possibly the strength of stimulation by the pacemaker. Appropriate use of pacemakers may have more applicability for the control of refractory reciprocating tachycardias than is presently appreciated.

Acknowledgment

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Further Comments

There are two comments that I should like to make about this paper. First, I would consider the successful outcome in Case 3, assuming this was ventricular tachycardia, as being exceedingly unusual. I would not like the readers of Circulation to get the idea that this is a reasonable way to terminate ventricular tachycardia, as I suspect it would rarely be effective, and of course asynchronous electrical pacing in such a setting could even be dangerous. Second, I believe the literature on the use of pacing such as that reported here to terminate arrhythmias is going to be somewhat slanted in the direction of favorable results. If such measures do not help, then they are not going to be reported! This reviewer knows of two cases in which pacing instituted for tachyarrhythmias similar to those reported in the first two cases of this paper was initially effective, but subsequently totally failed to control episodes of tachycardia. We have tried temporary pacing in at least two other instances without success, and therefore did not institute permanent pacing. I think it would be worthwhile for the authors to indicate whether the three cases presented here represent their total experience, or represent successful results of a group of patients in whom they might have used this approach.

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Authors' Addendum

The three cases presented in this report represent our total experience to date in the use of implanted pacemakers for the treatment of recurrent tachycardias.

After the manuscript was submitted for publication, the report of Wellens et al. (Circulation 49: 467; 1974) appeared which documented the termination of ventricular tachycardia by an appropriately timed electrical stimulus.

References

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