Paradoxical Use of a Demand Pacemaker in Treatment of Supraventricular Tachycardia Due to the Wolff-Parkinson-White Syndrome

Observation on Termination of Reciprocal Rhythm

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SUMMARY
This paper describes a new technique for terminating attacks of supraventricular tachycardia in a patient with Wolff-Parkinson-White syndrome by using a demand pacemaker. The circuitry of this demand pacemaker generator is designed so that a magnet held near the generator converts the unit from demand to fixed mode at a pre-set discharge rate of 72/min. During an attack of tachycardia, a magnet held near the generator pocket activates a fixed rate discharge and produces competitive pacing. Retrograde atrial depolarization occurs with resultant reversion to sinus rhythm.

Additional Indexing Words: Retrograde atrial depolarization Ventricular premature beat Sinus rhythm Lactate production

Despite the rather precise description of both the anatomic and electrical pathways of the impulses causing the tachycardia in the Wolff-Parkinson-White (WPW) syndrome, treatment in some cases remains perplexing and difficult. Although in most patients the arrhythmias are well tolerated, in others they result in severe symptoms and occasionally in sudden death. Quinidine and procainamide in combination with digitalis and propranolol have been recommended as the treatment of choice to abolish and prevent tachycardias. Direct current cardioversion has also been used in the treatment of supraventricular tachycardia (SVT). Recently direct surgical interruption of the A-V bundle with implantation of a pacemaker has been reported as a treatment for refractory SVT.

Attacks of SVT in the WPW syndrome can be both initiated and terminated by appropriately timed pacemaker-induced atrial or ventricular premature beats. The purpose of this paper is to report a new technique for the treatment of SVT in a patient with the WPW syndrome who experienced frequent and debilitating attacks of SVT. This was accomplished by using a permanent demand pacemaker to produce a ventricular premature beat and retrograde atrial depolarization.

Report of Case
M.L., a 52-year-old white married female with known WPW syndrome, has experienced recurrent attacks of SVT of increasing severity and frequency for the past 15 years. These attacks are associated with weakness, diaphoresis, lightheadedness, and anterior chest pressure and pain with extension into her left arm. In the last year, she has had approximately 20 emergency room visits and 11 hospital admissions.

Attempts to prevent these attacks with procainamide (4.0 g daily), diphenylhydantoin (400
mg daily), quinidine (1.6 g daily), digoxin (0.5 mg daily), and propranolol (240 mg daily) were all unsuccessful. During the attacks, intravenous injection of lidocaine, edrophonium hydrochloride, propranolol, phynylephrine hydrochloride, and metaraminol were all used on numerous occasions with variable results. The attacks usually lasted 12 to 18 hours and occasionally reverted spontaneously after failure of various therapeutic maneuvers.

The patient has been known to have diabetes mellitus for many years and has been taking acetoheaxamide (250 mg daily) for the last 5 years. She has also been moderately hypertensive with blood pressures averaging 180/90 mm Hg.

She was admitted to the Rochester General Hospital in March 1968, with an attack of unusual severity that failed to respond to any of these methods of treatment. Shortly after admission, the attack terminated spontaneously. Physical examination was unremarkable except for blood pressure of 170/90 mm Hg. The cardiac examination revealed a prominent left ventricular impulse. No murmurs or gallop sounds were audible. Roentgenograms of the chest showed moderate left ventricular hypertrophy.

An electrocardiogram taken during rest demonstrated a pattern consistent with those of type A WPW syndrome. Cardiac catheterization and selective coronary angiography were performed to evaluate her anginal symptoms. Transmyocardial lactate studies were performed by simultaneous sampling of the coronary sinus and brachial artery blood at rest and after 20 min of atrial pacing at 105 beats/min. These demonstrated lactate production during atrial pacing at which time the patient experienced recurrence of her anginal pain (fig. 1). Selective coronary angiography and left ventriculography revealed normal coronary vessels with good ventricular contractions.

Following discharge from the hospital, multiple episodes of tachycardia continued to recur and became more severe. On May 8, 1968, an episode of unusual severity associated with a systolic pressure of 80 mm Hg occurred and could not be reverted by any of the drugs used previously.

To generate a ventricular premature contraction and record the electrical events, two no. 5 bipolar pacing catheters (U. S. Catheter and Instrument Corporation) were passed percutaneously by way of the femoral veins through two large bore needles. One catheter was placed in the right ventricle for stimulation and one in the right atrium for intracardiac electrocardiographic recording. A ventricular premature contraction mechanically produced with the catheter in the right ventricle caused the attack to revert promptly to sinus rhythm.

Fifteen days later after having had numerous attacks of short duration at home, the patient entered the emergency room with another severe episode of SVT again accompanied by diaphoresis, hypotension, and severe chest pain. Once again, two transvenous bipolar pacemaker catheters were passed by the same technique one to the right atrium and one to the right ventricle. A continuous pacemaker stimulus of 5 ma was delivered at a rate of 75/min via the right ventricular endocardial catheter using a Medtronic no. 5800 external pacemaker generator. Competitive ventricular pacing was induced with retrograde A-V conduction and reversion of the SVT to sinus rhythm (figs. 2 and 3).

Because of the severity of these attacks and the difficulty experienced in reverting them with standard techniques, a permanent endocardial electrode catheter, of demand type, was inserted into the right ventricular apex. Pacing threshold was 1.3 ma. This particular demand pacemaker (American Optical Company) has as part of its circuitry, the capability to be changed from demand to fixed rate pacing by the external application of a magnet over the generator. With the patient in sinus rhythm at a rate exceeding the pre-set rate of the demand pacemaker or during the SVT, the demand pacemaker is nonfunctioning. During an attack of SVT application of the magnet diagonally over the generator will switch the unit to fixed rate discharge.

Since the implantation of the pacemaker, SVT has recurred on numerous occasions. Application of a magnet over the generator pocket, however, has produced competitive pacing with fixed rate discharge and has restored sinus rhythm.

![LACTATE and HEMODYNAMIC RESPONSE to PACING](image)

**Figure 1**

*See case report for discussion.*

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Figure 2
Simultaneous lead I, intra-atrial electrocardiogram (IAECG), and brachial artery pressure recording illustrate reversion of supraventricular tachycardia induced by the sixth pacemaker stimulus (S). R-R' time of this beat is 280 msec with an R'-P' time of 170 msec. The first pacemaker stimulus with an R-R' time of 320 msec does not terminate the tachycardia.

Figure 3
Intra-atrial electrocardiogram (IAECG) and standard ECG lead I with pacemaker stimulus (S) inducing a VPB (R') at 280 msec from the previous QRS that caused early retrograde activation of the atria (R'-P' time of 170 msec). In this case the antegrade pathway following P' was refractory and the tachycardia terminated.

Discussion
Supraventricular tachycardias in the WPW syndrome represent a serious threat to life because of their hemodynamic consequences and are frequently refractory to pharmacological therapy. Kimball and Burch reviewed the literature to 1947 and found eight deaths, and Okel in a more recent review found a total of 22 cases of sudden death due to the WPW syndrome. As pointed out by Saunders...
and Ord,15 cerebral insufficiency, faintness, and extreme weakness can occur with the tachycardia. Although Ferrer and associates25 found no change in blood pressure in one patient and minimal change in another, the blood pressure change associated with termination of an attack of SVT in our patient is illustrated in figure 2. Prompt remission of chest pain and dyspnea has always occurred with termination of an attack.

The development of hypotension and angina in our patient emphasized the danger of prolonged attacks of SVT. Although angina has been reported to occur in the presence of normal coronary arteries by Likoff and coworkers,26 lactate production is thought to occur only when significant coronary disease is present.27 The production of lactate during artificial pacing at accelerated rates is a good index of anaerobic metabolism of the heart. The development of lactate production with angina in our patient with normal coronary arteries suggests that during SVT coronary blood flow is decreased in relation to increased myocardial oxygen requirements.

Anti-arrhythmic drugs may be ineffective in the treatment and prevention of SVT associated with WPW.8,18 Burchell and associates24 reported a case of WPW syndrome and recurrent tachycardias in which surgical interruption of the Kent bundle was attempted, but following surgery episodes of tachycardias continued to recur. The surgical production of complete heart block in the treatment of SVT with23 and without WPW syndrome28 has been successfully carried out using cardiopulmonary bypass by interruption of the conduction bundle just below the atrioventricular node followed by the implantation of a synchronous pacemaker. We had contemplated such a procedure in the patient presented, but after the successful demonstration in our laboratory of the feasibility of terminating the attacks of tachycardia by the method described, we decided that implantation of a permanent transvenous pacemaker system was preferable, because of the much lower morbidity associated with transvenous pacemaker insertion.

Since the original description of the Wolff-Parkinson-White syndrome in 1930,29 anatomic1-4 and electrophysiological studies5-13 have led to the description of the anomalous pathways causing accelerated conduction from the atria to the ventricles through the bundle of Kent or bypass fibers of James and Mahaim either separately or in combination. The genesis of tachycardias in the WPW syndrome is still not clearly defined, but they are thought to occur when a supraventricular impulse is conducted antegrade over the normal pathway and re-enters in a retrograde direction via the accessory pathway causing atrial depolarization. The atrial impulse is then discharged in an antegrade direction and finding the anomalous pathway refractory (unidirectional block) is conducted again via the normal pathway with some degree of A-V block, thus establishing a reciprocal rhythm.30-34

Moe and associates35 were able to show in the dog that an appropriately timed atrial impulse could initiate a paroxysm of A-V nodal tachycardia and also that a more critically timed impulse would terminate an attack by interrupting the reciprocal rhythm within the A-V node.

Massumi and associates,5 utilizing paired atrial stimuli in much the same manner as Moe and associates, were able on numerous occasions to terminate a reciprocating tachycardia in a 10-year-old girl recovering from rheumatic carditis. Their work confirmed the application of this technique to patients.

Durrer and associates6 initiated and terminated attacks of SVT in four patients with WPW syndrome and agreed with the hypothesis that the basic mechanism for the genesis of the tachycardia was retrograde activation of the atrium through the accelerated bypass pathway. Their studies demonstrated that during SVT premature atrial depolarization, either by direct atrial stimulation or ventricular stimulation with retrograde atrial depolarization, terminated the SVT by interrupting the re-entry pathway. A pacemaker stimulus, applied to the right ventricle during the
tachycardia 260 to 290 msec after the preceding QRS, would induce a ventricular premature contraction that terminated the tachycardia. In contrast, a stimulus applied later in the cardiac cycle would induce a ventricular premature beat (VPB) but would not alter the SVT, the pause between the VPB and the next ventricular complex being fully compensatory.

The interruption of the SVT by premature atrial and ventricular depolarization appears to occur by different mechanisms. Premature atrial stimulation as demonstrated by Durrer and associates rendered the atria refractory to retrograde depolarization by the impulse from the accelerated pathway. Premature ventricular stimulation generates early retrograde activation of the atria with identical conduction (R-P) time as the ectopic rhythm. Then when the impulse from this atrial depolarization travels in the antegrade direction through the normal A-V conduction system, the junctional tissue is refractory and the reciprocal rhythm is blocked. Thus, if the ventricular premature beat does not occur early enough, its resultant atrial antegrade transmission is able to penetrate the junctional tissue with a longer conduction time because of a partial refractory state. We were able to demonstrate in our patient that a stimulus applied at 280 msec after the QRS induced a VPB that caused early retrograde activation of the atria and stopped the tachycardia (fig. 3). A stimulus occurring later than 290 msec

![IAECG and standard ECG lead I with pacemaker stimulus (S) inducing a VPB with R-R' interval of 300 msec. The P-R time is 210 msec and the R-P time is 170 msec. Following the VPB, retrograde atrial depolarization (P') occurs via the Kent bundle at the usual R-P time of 170 msec. The antegrade conduction following P' finds the A-V junctional pathway partially refractory; and it does penetrate the A-V junction but with a longer P-R time. (P'-R duration = 290 msec.)](http://circ.ahajournals.org/)

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induced a VPB that would not break the tachycardia (fig. 4). This retrograde beat changed the P-P interval of the SVT by initiating the P-R delay in the next beat due to concealed antegrade conduction of the previous atrial beat. The point in the cycle at which retrograde activation of the atria takes place is, therefore, of critical importance in interrupting the tachycardia. From our studies, it appears that the R-R interval required to break the tachycardia was between 260 and 290 msec with the R-P time of SVT and VPB being the same (170 msec).

The danger of eliciting repetitive ventricular discharge by our method of treatment requires critical evaluation of the problem of repetitive beats induced by an artificial pacemaker stimulus. In multiple episodes of initiating and terminating SVT in four patients with WPW syndrome, Durrer and associates failed to induce ventricular tachycardia or fibrillation. A number of factors may influence the development of repetitive beats in the normal heart, and these same factors can be expected to apply to the patient with WPW syndrome. These include an impulse duration of over 5 msec, myocardial ischemia, and excess sympathomimetic activity. The production of lactate indicative of anaerobic myocardial metabolism and therefore, ischemia, suggests that one of these factors is present in our patient. It is felt, nevertheless, that the morbidity and risk of mortality in an attack of SVT justified this method of treatment in our patient. To date since the pacemaker was implanted, all of our reversion episodes have been electrocardiographically monitored and have not been associated with a single repetitive beat. Propranolol has been continued because of its possible beneficial effect in reducing the likelihood of repetitive beats, and also because the attacks of SVT have been less frequent since the inception of propranolol therapy.

This method of terminating SVT in the WPW syndrome seems established as a safe and effective method when the basic atrial rhythm during an episode of SVT is coupled with the reciprocal rhythm within the A-V node. However, when atrial fibrillation occurs in conjunction with an episode of SVT, termination of the SVT by the method described may not be possible. Further investigation of patients in whom atrial fibrillation or flutter occurs in conjunction with the pre-excitation syndrome is necessary before this method of treatment can be extended to include all types of SVT in the WPW syndrome.

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References


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