Electrically Induced Atrial Tachycardia with Block

A Therapeutic Application of Permanent Radiofrequency Atrial Pacing

By Robert M. Davidson, M.D., Andrew G. Wallace, M.D.,
Will C. Sealy, M.D., and Michael S. Gordon, M.D.

SUMMARY
Atrial tachycardia with block was induced by rapid atrial pacing in a patient with refractory supraventricular tachycardia in an effort to control the ventricular rate. It was initially decided to induce atrial fibrillation as necessary with the use of a permanent radiofrequency-controlled atrial pacemaker. Several days after implantation of the pacemaker, however, rapid atrial pacing produced atrial tachycardia with atrioventricular block rather than atrial fibrillation. Despite this apparent failure, the ventricular rate has been adequately controlled by rapid atrial pacing for the past 9 months. Intermittent induction of atrial tachycardia with block with a permanent radiofrequency atrial pacemaker may represent a useful approach to the treatment of patients with refractory supraventricular tachycardia.

Additional Indexing Words:
Supraventricular tachycardia  Artificial pacemaker  Arrhythmia

Paroxysmal supraventricular tachycardia in the absence of significant cardiac disease is usually managed with relative ease. Attacks can be prevented either by eliminating agents which obviously incite the arrhythmia or by using conventional antiarrhythmic therapy. Our patient was unusual in that paroxysmal tachycardia resulted in virtual incapacitation and could not be prevented even with large and toxic amounts of drugs. The problem was finally managed successfully with the intermittent use of a permanently implanted radiofrequency atrial pacemaker to induce a more rapid atrial tachycardia with block.

Report of Case
M.R., a 20-year-old college student, was admitted to Duke University Medical Center in July 1970 for evaluation of long-standing supraventricular tachycardia. He had been in normal health until 1967 at which time he developed a respiratory infection with signs of bronchitis. After 2 weeks of persistent fever and cough which were unresponsive to penicillin, he was hospitalized and a diagnosis of myocarditis was entertained because of the additional finding of a supraventricular tachycardia at a rate of approximately 150 beats/min and with inverted P waves in leads II and III. The arrhythmia was present almost continually and, although it could be terminated by a Valsalva maneuver, sinus rhythm would persist for only a few minutes before the abrupt return of the tachycardia.

One year after the onset of symptoms, the patient was transferred to another hospital where the arrhythmia was first successfully converted to

From the Divisions of Cardiology and Thoracic Surgery, Duke University Medical Center, Durham, North Carolina, and the Department of Medicine, University of Miami, Miami, Florida.


Address for reprints: Andrew G. Wallace, M.D., Chief of Cardiology, Duke University Medical Center, Durham, North Carolina 27706.

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a sinus mechanism in response to medication. This medication was propranolol administered intravenously. Despite the success achieved with intravenous administration of propranolol, oral treatment with propranolol in doses as high as 280 mg every 3 hours, alone or in combination with digoxin in doses up to 0.5 mg every 8 hours or quinidine in doses up to 1.0 g every 6 hours, failed to control or prevent the tachycardia.

During the 2-year interval between 1968 and his admission to Duke University Medical Center the arrhythmia persisted in spite of numerous efforts by several physicians. In the 6 months which preceded hospitalization at Duke, he had a number of presyncopal spells precipitated by exertion, during which his pulse rate was observed to be in excess of 200 beats/min.

Physical examination at the time of admission to our hospital revealed a well-developed, well-nourished muscular white male in no apparent distress. The pulse rate was regular at 136 beats/min and the blood pressure was 122/78 mm Hg. With vigorous exercise his pulse rate would increase to 200 beats/min. Examination of the heart revealed that the point of maximum impulse was 9 cm to the left of the midsternal line in the fifth intercostal space, and its quality was unremarkable. There were no murmurs or rubs, but there was a prominent fourth heart sound. The remainder of the physical examination was unremarkable. Routine laboratory studies revealed normal hemoglobin and white blood cell count. The urinalysis and blood chemistries, including tests of thyroid function, were unremarkable. The chest X-rays were normal.

The tracings presented in figure 1 illustrate the 12-lead electrocardiogram during an episode of tachycardia. At rest, the heart rate was approximately 120 beats/min, and P waves were inverted in leads II, III, aVf, and V4–6. The P-R interval measured 0.16 sec and the QRS complex was of normal duration. During sinus rhythm as illustrated in figure 2, the heart rate was approximately 60 beats/min, and the P waves were upright in leads II and aVf and in all of the V leads. The QRS complex was of normal duration and the P-R interval measured 0.14 sec.

On the third hospital day, the patient was taken to the cardiac catheterization laboratory for electrophysiologic studies. The tracings shown in figure 3 were obtained during the spontaneous onset of the tachycardia. The upper tracing is a

![Figure 1](12-lead electrocardiogram taken at rest during an episode of spontaneous tachycardia. This particular tracing was taken 1 week after surgery. Changes in ST-T waves were not present on the preoperative tracing.)
Figure 2
A 12-lead electrocardiogram taken at rest and during a period of normal sinus rhythm. Changes in the ST-T waves were an early postoperative change.

Figure 3
Electrograms during the onset of the tachycardia. RA = right atrium; His = bundle of His electrogram; L-II = a lead-II surface electrocardiogram. See text for description.
bipolar electrogram recorded from within the right atrium near the junction between the superior vena cava and the atrial appendage. The second tracing is a record from a catheter located within the right atrium and adjacent to the coronary sinus. The third tracing is from a bipolar electrode catheter which was positioned adjacent to the region of the bundle of His. In the three beats preceding the onset of the tachycardia, the initial activity was recorded from high in the right atrium just before the onset of the P wave. The atrial electrograms recorded from low in the right atrium and from the catheter over the bundle of His showed essentially simultaneous activity. The tachycardia was initiated by an atrial premature depolarization in which the P wave was inverted in the surface electrocardiogram. The interval between atrial activity and the His bundle complex was prolonged on the first and subsequent beats of the tachycardia. During episodes of tachycardia the P-R interval on the surface ECG and the P-H interval on the His bundle electrogram were always longer than in sinus rhythm. Single spontaneous atrial premature depolarizations were never detected in this patient, and the ability to induce episodes of tachycardia with a single premature atrial stimulus was not examined. However, brief periods of atrial pacing with an interval between stimuli of 440 msec consistently induced episodes of tachycardia. In each instance the stimulus which initiated the arrhythmia elicited a premature atrial depolarization which fell within the functional refractory period of the atrioventricular node (i.e., a prolonged P-H interval). During episodes of tachycardia, the earliest atrial activity was recorded from electrodes near the coronary sinus and atrioventricular node and activity high in the right atrium was recorded substantially later.

The tracings presented in figure 4 were obtained during the termination of the arrhythmia which occurred after the release of a Valsalva maneuver. The five beats to the left of the arrow were recorded during the tachycardia, after release of the Valsalva maneuver, and during a presumed increase of vagal nerve activity to the heart. The interval between atrial activity and the complex recorded from the bundle of His increased on each of the three beats prior to termination of the tachycardia. There was an associated increase of atrial cycle length. Following the last ventricular complex of the tachycardia, there was an atrial depolarization which

![Figure 4](image)

**Figure 4**

Electrograms during termination of the tachycardia and return to normal sinus rhythm. P = P wave; H = His bundle electrogram; V = ventricular electrogram. The tachycardia terminated approximately 8 sec after release of a Valsalva maneuver.

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by a pause after which sinus rhythm resumed. This pattern was characteristic of many episodes in which the arrhythmia was terminated by release of a Valsalva maneuver. It was of considerable interest that, when the tachycardia slowed prior to its termination, the R-P interval remained constant despite sometimes marked lengthening of the P-R interval. Furthermore, cessation of the tachycardia always occurred when an atrial depolarization closely coupled to the preceding R wave failed to propagate antegrade to the bundle of His and ventricle. Although this relation between atrial and ventricular events at the onset and termination of the arrhythmia may have been fortuitous, initiation by a premature atrial depolarization which fell within the functional refractory period of the A-V node and termination with an atrial depolarization which was coupled to the last R wave (an atrial echo) and which failed to propagate antegrade to the His bundle are most compatible with a mechanism which involves reentry within the A-V node.\textsuperscript{1,2}

Further evidence that reentry within the A-V node was the probable mechanism for the arrhythmia may be obtained from consideration of two other observations in this patient. First, as shown in the upper panel of figure 5, a premature junctional beat with retrograde activation of the atrium initiated the tachycardia. Finally, it was possible to terminate the tachycardia with a single premature ventricular beat which failed to propagate to the atrium but presumably interrupted the reentry path within the A-V node by concealed conduction.

This patient was referred to us for possible definitive surgical correction of his disturbance of cardiac rhythm. One possibility which we entertained was division of the bundle of His and implantation of a permanent ventricular pacemaker. This approach was discarded because of the potential complications of inducing complete heart block and the potential morbidity associated with the lifelong requirement for a cardiac pacemaker. A second possibility we entertained was to implant a fixed rate atrial or ventricular pacemaker, in the hope that single beats initiated by the pacemaker would terminate episodes of tachycardia. This approach was discarded because the patient could terminate the tachycardia at will with the Valsalva maneuver, but it would return immediately, and what seemed to be needed was some mechanism to hold him at a normal rate at rest and to prevent the excessive increase of rate which occurred with exercise or other physical activity.

In view of an observation that atrial fibrillation could be induced electrically with a substantial reduction in ventricular rate, it was our initial plan to implant an atrial pacemaker which could be used to induce atrial fibrillation. The feasibility of this was established preoperatively by placing a pacing catheter in the right atrium for several

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**Figure 5**

Electrically induced atrial fibrillation on second postoperative day. (Upper panel) Sinus rhythm with an episode of spontaneous tachycardia (rate 120) induced by a premature junctional beat with aberration. (Lower panel) The effect of atrial pacing at 750/min (presumed atrial fibrillation) which led to a reduction of ventricular rate to 85 beats/min. Monitor lead approximates a lead III.
INDUCED ATRIAL TACHYCARDIA

days, maintaining the patient in atrial fibrillation and demonstrating that the ventricular rate could be controlled with digitalis.

Accordingly, the patient was taken to the operating room and the chest was opened through a right thoracotomy. There were no obvious lesions of the right atrium and there was no evidence of old pericarditis. Wires were implanted on the right atrial appendage and connected to a radiofrequency impulse generator* which was implanted in the abdominal wall. The chest was closed, and the patient was returned to the recovery room. During the first three postoperative days, it was easy to induce atrial fibrillation with atrial pacing at a stimulation frequency between 600 and 800 beats/min (fig. 5). However, on approximately the third postoperative day, during the course of digitalization, we first noted that it was no longer possible to induce atrial fibrillation, although the atrium could easily be captured by the pacemaker at rates up to 250 beats/min.

During the second postoperative week, the patient became ambulatory and the functions of the pacemaker were evaluated. In the absence of any medication other than a maintenance dose of digoxin, 0.25 mg/day, the atrium could be stimulated at a rate of approximately 200 beats/min with 2:1 A-V block at rest. During exercise, however, the degree of block decreased, and an excessively rapid ventricular response resulted. Accordingly, he was given an additional 0.75 mg of digoxin and 80 mg of propranolol every 8 hours. With these medications, there was an increase in the degree of atrioventricular block such that, when the atrium was paced at a frequency of 220 beats/min, there was second-degree A-V block which varied from 3:1 to 5:1 at rest (fig. 6, upper panel). During exercise there was 2:1 block with a ventricular rate of 110 beats/min (fig. 6, lower panel). This appeared to be a satisfactory mechanism since, at rest, the ventricular rate was approximately 60 beats/min and, during exercise, it increased to 110 beats/min but no faster. The patient was discharged with the pacemaker implanted and with instructions to turn it on in the event that the tachycardia occurred, or in situations in which he could anticipate that exertion would bring on the tachycardia. Medications which were to be continued after discharge included digoxin, 0.50 mg/day, and propranolol, 80 mg every 8 hours. Figure 7 shows a 12-lead ECG during atrial pacing at the time of discharge.

Discussion

This case is unusual both in the clinical course of a usually benign type of arrhythmia and in the final treatment of this problem. The etiology of the patient's arrhythmia remains obscure to us. It was felt that his febrile illness was probably associated with myocarditis and that the arrhythmia was a consequence of this

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*Specially designed and provided by Medtronic Corporation.

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Figure 6

Atrial pacing at 220/min postoperatively. (Upper panel) At rest. (Lower panel) During exercise (jogging). Lead V1.

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A 21-lead electrocardiogram at rest, while pacing the atrium at 240/min, on the day of discharge.

Figure 7

Atrial pacing has been used successfully in the treatment and prevention of atrial tachycardias. Atrial flutter and other reentrant types of tachycardia have been easily terminated with atrial pacing. Paired atrial pacing has also been used in the past to slow the ventricular rate in sinus tachycardia. With this latter technic, a premature atrial depolarization is delivered during the refractory period of the A-V node, causing resetting of the sinus node and thus slowing of the ventricular rate. Another recent approach to the treatment of refractory supraventricular tachycardia has been the intentional induction of atrial fibrillation with atrial pacing. In one case, a temporary transvenous catheter and external pacemaker were used to induce atrial fibrillation in a 61-year-old man with rheumatic heart disease and refractory atrial tachycardia. The catheter was left in place for 6 days, after which the patient remained in predominantly sinus rhythm with the use of conventional drug therapy. In another recent report, paired atrial pacing from the coronary sinus was employed in two patients to convert refractory supraventricular tachycardia to atrial flutter, which was then changed to atrial fibrillation with the use of digitalis. It was
our intention in this patient to induce atrial fibrillation, as necessary, with rapid atrial pacing with a radiofrequency controlled permanent pacemaker. Permanent atrial pacemakers set at frequencies above the spontaneous sinus rate have been used in the past to prevent atrial and ventricular tachyarrhythmias. Recently, a permanent atrial pacemaker which could be activated by the patient was used in the treatment of paroxysmal tachycardia associated with the Wolff-Parkinson-White syndrome. We are not aware of the previous use of a permanent atrial pacemaker to induce either atrial fibrillation or atrial tachycardia with block. In our patient, the radiofrequency technic was selected because of anticipated intermittent need for pacing and because the battery life of an implanted unit set at these frequencies would be prohibitively short.

Although the original intention was to use the pacemaker to induce atrial fibrillation, it became evident a few days after surgery that atrial fibrillation could no longer be induced but, rather, that rapid atrial pacing resulted in atrial tachycardia with varying degrees of atrioventricular block. The reason for the fact that atrial fibrillation could be induced with ease on the first few postoperative days, but not thereafter, was not clear to us. It may have been explained either by postoperative changes around the electrodes or an effect of digitalis. In a recent study, digitalis intoxication prevented the induction of atrial fibrillation with alternating-current stimulation of the atrium. This suggested that digitalis might produce a type of pacemaker exit block.

Fortunately, it was no longer necessary to produce atrial fibrillation to control our patient's tachycardia since the production of atrial tachycardia with block was adequate for this purpose. In summary, intermittent induction of atrial fibrillation or atrial tachycardia with block, with a permanent radiofrequency atrial pacemaker, appears to have adequately controlled an otherwise refractory supraventricular tachycardia in this young man, and may be considered as one of several alternatives for the treatment of refractory supraventricular tachycardias in other patients.

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ROBERT M. DAVIDSON, ANDREW G. WALLACE, WILL C. SEALY and MICHAEL S. GORDON

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