Paroxysmal Fascicular Tachycardia: Electrophysiologic Characteristics and Treatment by Catheter Ablation

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A 69 year old man with ischemic heart disease underwent electrophysiologic evaluation for paroxysmal wide QRS tachycardia, the configuration of which was identical to that recorded during sinus rhythm, that is, right bundle branch block, left anterior fascicular block and anterior myocardial infarction. Electrocardiographic recordings during tachycardia showed atrioventricular dissociation and His bundle activation occurring 5 ms after the onset of the QRS complex recorded on the surface electrocardiogram, consistent with a left posterior fascicular tachycardia. All traditional therapeutic attempts failed to prevent frequent recurrences of tachycardia, which was finally ablated by three 300 J shocks delivered through an electrode catheter positioned in the posterobasal region of the left ventricular septum.

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Case Report

History. The patient is a 69 year old man who, 7 years before admission to this hospital, sustained an anterior myocardial infarction with aneurysm formation, followed by surgical repair 6 months later. Twelve and 6 months before admission, he was hospitalized elsewhere for electrical cardioversion of paroxysmal tachycardia diagnosed as supraventricular. Digitalis, procainamide and, subsequently, quinidine were administered, but the latter two drugs produced intolerable gastrointestinal side effects, and diltiazem was substituted. Three days before hospital admission, he presented to the emergency room of another hospital with angina pectoris and palpitation caused by recurrent paroxysmal tachycardia at a rate of 220 beats/min, uninterrupted by intravenous lidocaine and verapamil and only briefly interrupted, but not terminated, by cardioversion or pacing techniques. After several synchronized direct current shocks up to 200 J, the tachycardia finally abated and the patient was transferred to this hospital for further evaluation and treatment.

On admission to the hospital, the patient looked chronically ill. His blood pressure was 130/75 mm Hg. He had an old sternotomy scar. The cardiac examination revealed a regular rhythm at 76 beats/min and a widely split S2 and an S4 gallop. There were no signs of cardiac decompensation. The 12 lead electrocardiogram showed sinus rhythm, left axis deviation, right bundle branch block and precordial Q waves characteristic of a large previous anterior myocardial infarction (Fig. 1A). Review of the electrocardiograms obtained during tachycardia showed the same QRS configuration and no clear atrial activity (Fig. 1B). All antiarrhythmic drugs were withheld and a symptom-limited exercise test was performed to the point of fatigue without development of tachycardia or angina. Thallium-201 perfusion imaging showed a small, reversible inferoapical myocardial defect. Postoperative angiograms obtained 4 years before this hospital admission were reviewed and showed complete occlusion of the left anterior descending artery, a single high grade lesion of the distal posterior descending artery and severe, diffuse left ventricular dysfunction. The right coronary artery was small and patent. Because exercise had produced neither tachycardia nor angina pectoris, and

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because thallium-201 imaging showed only a small, peripheral perfusion defect, coronary angiography was not repeated.

**Electrophysiologic findings.** Electrophysiologic testing was performed using standard techniques with electrode catheters placed in the high right atrium, coronary sinus, His bundle region and at the right ventricular apex. All intracardiac signals, along with surface electrocardiographic leads, I, aVF and V1, and femoral arterial pressure were displayed on a multichannel oscilloscope and recorded on a rapid-writing strip chart recorder (Electronics for Medicine VR-16) at speeds between 50 and 250 mm/s. Programmed stimulation was performed with a Bloom DTU-101 unit using stimuli of twice end-diastolic threshold intensity, except during attempts to interrupt the tachycardia by burst pacing, when the output was increased to 5 to 10 times the end-diastolic threshold. During incremental atrial pacing, at a cycle length of 350 ms, the ventricular cycle length suddenly shortened to less than 300 ms, and atrioventricular (AV) dissociation was noted (Fig. 2). The His bundle de-

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**Figure 1.** A 12 lead electrocardiogram during sinus rhythm (A) and during paroxysmal tachycardia (B).

**Figure 2.** Induction of fascicular tachycardia by rapid atrial pacing during electrophysiologic study. The arterial pressure (AP) tracing shows 2:1 mechanical response during atrial pacing. From top to bottom, surface electrocardiographic leads I, aVF, V1; intracardiac recordings: HBE = His bundle electrogram; HRA = high right atrium; PCS = proximal coronary sinus; RV = right ventricular apex; St = stimulus artifact. All intervals are indicated in milliseconds. The arrow shows onset of atrioventricular (AV) dissociation.
flection remained present before each ventricular electrogram; however, the onset of ventricular activation as determined by the QRS configuration on the surface electrocardiogram now preceded the inscription of the His bundle signal, and the HV interval had shortened from 40 to −5 ms. No change in QRS configuration was observed in the three surface electrocardiographic leads. These findings were indicative of a tachycardia originating from the left posterior fascicular area. After a series of cycles of variable length (Fig. 2), the tachycardia became regular at a rate of 200 beats/min. The patient remained conscious, but complained of chest pain and tightness.

For approximately 30 minutes, efforts were made to terminate the rhythm with pacing techniques including ventricular stimulation by burst pacing at cycle lengths as short as 200 ms. Cessation of burst pacing was followed by resumption of tachycardia after one or two cycles of sinus rhythm or, more commonly, after a period of overdrive suppression, the duration of which was inversely related to the cycle length of the burst. On several occasions, the tachycardia resumed with gradual shortening of its cycle length (Fig. 3). After innumerable termination attempts, the rhythm finally ended after triple ventricular extrastimulation. The procedure was terminated and antiarrhythmic treatment was begun.

Drug therapy. During the following 30 days, the patient had 19 episodes of sustained fascicular tachycardia, requiring 27 transthoracic direct current shocks of 50 to 200 J to terminate the tachycardia. In addition, he received approximately 85 intracardiac shocks delivered by a 5350 Medtronic external cardioverter by means of 6880 electrodes inserted on two occasions for a total period of 18 days. At times, the tachycardia could be terminated by intracardiac shocks of 8 J or less, but on most attempts low energy shocks merely suppressed the rhythm briefly, as was observed with pacing techniques. These episodes of tachycardia occurred despite attempts at therapeutic regimens of procainamide, lorcaïnide, mexiletine, phenytoin and amiodarone, and frequently persisted despite the addition of intravenous lidocaine, bretylium tosylate and propranolol. The most tenacious episodes occurred within 24 hours of onset of treatment with lorcaïnide, phenytoin and mexiletine, respectively. Furthermore, 20 days after the beginning of treatment with amiodarone, 1,200 mg daily, incessant ventricular tachycardia developed that could not be interrupted by electrical or by pharmacologic methods. The rate of the tachycardia varied between 160 and 190 beats/min. Thus, it was evident that antiarrhythmic drug treatment not only was ineffective, but that most drug trials had facilitated the arrhythmia. The patient was bedridden and hypotensive unless supported by continuous dopamine infusion, required intensive medical care and had recurrent episodes of angina pectoris.

Catheter ablation. Five days after the onset of incessant ventricular tachycardia, the patient was taken to the electrophysiology laboratory for catheter ablation. This procedure was approved on a one-time basis by the Chairman of the Investigational Review Board of the Jewish Hospital, and informed consent was obtained from the patient. Under local anesthesia, 7F USCI electrode catheters were placed from both femoral areas in the His bundle region, the right ventricular apex and the left ventricle. All signals were displayed and recorded as described earlier. The characteristics of the spontaneous rhythm were identical to those recorded from the induced tachycardia during the first procedure. Under fluoroscopic control, the quadripolar left ventricular electrode was manipulated in search of the site of earliest activation. As expected, this was found in the posterobasal region of the interventricular septum, where the electrogram preceded the onset of the surface QRS complex by 38 ms and the His bundle signal by 45 ms (Fig. 4).

Overdrive pacing from the tip of the electrode at this site produced no change in the surface QRS configuration and minimal changes in the intraventricular activation sequence recorded from the right ventricular and His bundle electrodes (Fig. 5). The catheter could be maintained in a stable position and its most distal electrode was then connected to the cathode of a Lifepack no. 6 external defibrillator. The

Figure 3. Transient overdrive suppression of tachycardia by ventricular burst pacing at a cycle length of 200 ms (300/min). Note 2: 1 V to H and V to A conduction during pacing, and pulsus alternans during spontaneous tachycardia. See text for additional discussion. Abbreviations as in Figure 2.
The anode of the unit was connected to a 12 cm² R2 self-adhesive defibrillating pad that was placed over the mid right anterior hemithorax. The patient was then anesthetized with etomidate, 16 mg intravenously, and a 300 J direct current shock was passed between the electrode catheter and the R2 pad. This was immediately followed by complete infra-Hisian atrioventricular (AV) block and ventricular escape rhythm at a rate of 30 beats/min (Fig. 6), with systemic hypotension that resolved promptly on initiation of ventricular pacing. Within the following 5 minutes, two additional 300 J shocks were given in the same fashion. The patient was allowed to wake up, and was kept for observation in the electrophysiology laboratory for the next 3 hours. In the meantime, AV conduction resumed gradually, first with 2:1 infra-Hisian block at cycle lengths less than 600 ms. Programmed stimulation of the ventricles was also performed, including triple extrastimulation during sinus rhythm and during ventricular paced rhythm at cycle lengths of 600, 500 and 400 ms, and ventricular burst pacing to cycle lengths of 270 ms. These maneuvers all failed to induce any form of ventricular tachyarrhythmia.

**Short-term follow-up.** The patient was returned to the coronary care unit with sustained atrial flutter and 2:1 block that was induced by programmed atrial stimulation at the end of the procedure. All antiarrhythmic agents were withheld; digitalis and anticoagulation therapy with heparin were begun. Approximately 8 hours later, the patient developed sustained ventricular tachycardia at a rate of 200 beats/min with severe hypotension, requiring multiple intracardiac and transthoracic shocks, hemodynamic support with large amounts of vasopressors and intubation with 100% oxygen delivery. Intravenous lidocaine and procainamide were again administered. The QRS configuration in this episode of tachycardia was distinctly different from the QRS configuration recorded during sinus rhythm or atrial flutter. The patient became clinically stable rapidly thereafter and only occasional episodes of nonsustained ventricular tachycardia were noted during continuous monitoring. Over the following 24 hours, total creatine kinase (CK) enzyme peaked to 1,052 U (normal 25 to 160), and the CK-MB fraction was greater than 10 IU/liter. A technetium-99m pyrophosphate scan performed 48 hours after catheter ablation showed an abnormally increased focal uptake in the posterolateral territory of the heart. The 12 lead electrocardiogram was unchanged from that obtained on admission. On day 4 after catheter ablation, procainamide treatment was discontinued and on day 8, the patient left the coronary care unit. Continuous monitoring showed only rare episodes of brief, nonsustained ventricular tachycardia. Fifteen days after tachycardia ablation the patient was taken once again to the...
electrophysiology laboratory, and programmed stimulation was performed with a single electrode catheter placed at the right ventricular apex. Nonsustained ventricular tachycardia was induced by double ventricular extrastimulation (Fig. 7). The QRS configuration of this tachycardia was different from that recorded during previous studies or during sinus rhythm or atrial flutter, but similar to that recorded during pacing from the right ventricular apex. The cycle length before treatment was 310 ms, and increased to 400 ms after procainamide was administered (plasma level 8 mg/liter). None of the induced episodes produced symptoms, and no sustained tachycardia was induced by any method, including triple extrastimulation. Treatment with oral procainamide was started and after 54 days of hospitalization, 21 days after the catheter ablation, the patient was discharged from the hospital.

Long-term follow-up. A few days after hospital discharge, the patient experienced a few episodes of palpitation, prompting his brief readmission to the telemetry unit, where one self-limited episode of sustained tachycardia, lasting a few minutes, was recorded on a single electrocardiographic channel. Procainamide treatment was continued and 6 months later, the patient has remained free of further recurrences of symptomatic tachycardia and angina pectoris.

Discussion
Fascicular rhythms. Sustained fascicular rhythms are difficult to diagnose without intracardiac recordings. Their existence can be suspected when AV dissociation is observed together with a QRS configuration showing less widening and aberrancy than usually seen in ventricular tachycardia. In our patient, the fixed intraventricular conduction abnormalities present during sinus rhythm prevented the recognition of a change in QRS configuration during tachycardia, and the rapid rate and wavy baseline obscured the identification of AV dissociation. Thus, from the surface electrocardiogram, the rhythm was indistinguishable from that of a paroxysmal reentrant tachycardia arising from the AV node or utilizing a concealed bypass tract in the ventriculoatrial direction. Additional possibilities in the differential diagnosis were paroxysmal rhythms arising from the atria, particularly atrial flutter.

Ultimately, the diagnosis of fascicular rhythm rests on the demonstration of a His bundle deflection before each ventricular electrogram and an HQ interval shorter than the interval measured during sinus rhythm (1,3–6). However, this finding, though necessary, is not sufficient because H deflections preceding ventricular electrograms can be re-
corded during ordinary ventricular tachycardia. Thus, other reports of fascicular rhythms have included additional findings; for instance, narrowing of the QRS configuration during ectopic rhythm in the presence of bundle branch block during sinus rhythm (4,7) or, conversely, a moderate degree of aberrancy in escape complexes preceded by His spikes occurring at or near the onset of the QRS complex (1–3,5).

We believe that our case represents an example of paroxysmal fascicular tachycardia for the following reasons: 1) The QRS configuration of complete right bundle branch block, left anterior fascicular block and anterior infarction was identical during sinus rhythm and tachycardia. 2) A His bundle deflection occurring 5 ms after the onset of the QRS complex on the surface electrocardiogram was recorded well ahead of the ventricular electrogram in the His bundle region. Additional supportive findings were the recording of the earliest electrical activity in the posterobasal region of the septum, and the prolonged period of complete infranHisian AV block that resulted from the catheter shock discharged at that site.

Paroxysmal tachycardias originating from the fascicles seem rare. In 1974, Goolsby and Oliva (8) described a case of recurrent tachyarrhythmia thought to arise from the distribution of the anterior fascicle. Their case shared several clinical characteristics with ours, particularly the paroxysmal nature of the arrhythmia and drug refractoriness. In their case, however, the infra-Hisian origin of the rhythm was suggested by marked QRS aberrancy present during tachycardia. Goolsby and Oliva (8) interpreted the rhythm as fascicular tachycardia because of the presence of a distinct His potential preceding the ventricular electrogram in the infranHisian AV block that resulted from the catheter shock discharged at that site.

Most suspected or documented cases of sustained fascicular rhythm were of the nonparoxysmal or accelerated ectopic variety and often occurred in the presence of digitalis toxicity or acute myocardial infarction (2,3,6). Indeed, a digitalis-dependent animal model of this arrhythmia was described by Damato et al. (9). It is thus apparent that abnormal automaticity is the most common mechanism of sustained fascicular rhythms. Our case is no exception to this general finding. Despite its paroxysmal nature, the rhythm observed in our patient had several characteristics suggestive of an automatic tachycardia, such as its refractoriness to electrical treatment and gradual “warm-up” often noted at its onset or after transient suppression.

Catheter ablation. From a therapeutic viewpoint, the case we described is an oddity by current standards. Catheter ablation of tachyarrhythmias has been mostly reserved for the control of refractory supraventricular and AV reentrant tachyarrhythmias (10,11). The application of high energy shocks delivered through electrode catheters for the treatment of paroxysmal ventricular tachyarrhythmia has been described by Hartzler (12) in a few patients whose conditions could not be controlled by traditional methods. Hartzler’s success rate was high according to a recent preliminary report (13). Our case provides additional support both for the application of this technique in the treatment of ventricular tachycardia and for the expansion of animal investigation in this area. For now, however, this technique should be used only as a response to exceptional circumstances. Our patient’s tachycardia was disabling, drug-aggravated, resistant to electrical treatment and occurred in the setting of severe coronary artery disease and severe ventricular dysfunction after surgical repair of a left ventricular aneurysm, making the patient a poor surgical candidate. In addition, given the electrophysiologic characteristics of the rhythm, we were concerned that its induction might not be accomplished under deep anesthesia. Catheter ablation was thus our only option.

Potential complications. The potential serious risks of catheter ablation were illustrated by the period of marked electrical instability that occurred several hours after the procedure. It appears that the shock treatment itself created a new form of ventricular tachycardia which, within the first 24 hours, was a life-threatening complication. This rhythm finally abated only to reappear infrequently in a self-limited form, the form that was induced in the laboratory before the patient’s discharge from the hospital, and which has been easily controlled with oral procainamide. After considerable debate, we chose not to offer this patient a permanent pacemaker, in view of the rapid return after catheter ablation of 1:1 AV conduction during sinus rhythm, and 2:1 conduction during flutter within 2 to 3 hours after the procedure. Furthermore, for 21 days of continuous monitoring thereafter, no instance of failure of AV transmission was observed.

Although this ablation procedure was ultimately successful in removing the original intractable fascicular tachycardia, we do not wish to promote its clinical application except in carefully selected cases refractory to traditional therapeutical methods. Further experimental investigation is manifestly warranted.

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References


