Radiofrequency Ablation Therapy in Concealed Left Free Wall Accessory Pathway With Decremental Conduction*

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An electrophysiologic study followed by transcatheter radiofrequency ablation therapy was performed in two adult patients with a permanent form of junctional tachycardia. Both patients had no structural heart disease and exhibited a normal resting ECG. The P wave during tachycardia was negative in leads I, 3, and aVF, biphasic over V6, and positive in V1 and aVL in both patients, while the P-R/R-P interval ratio during tachycardia was 0.82 and 0.36, respectively, in both patients. Both patients displayed an eccentric atrial activation sequence with the earliest atrial activation occurring at the distal coronary sinus and a decremental retrograde conduction property during incremental ventricular pacing, suggesting the presence of a concealed slowly conducting left free wall accessory pathway. The tachycardia used the normal atrioventricular pathway for anterograde conduction and the concealed slow left accessory pathway for retrograde conduction. It was terminated following adenosine administration in both patients; termination of tachycardia was due to a block in the retrograde accessory pathway in one patient and due to a block in the atrioventricular node in the other patient. Radiofrequency ablation was performed by the retrograde transaortic approach. The radiofrequency current was delivered to the site of the earliest atrial activation during tachycardia at the ventricular aspect of the mitral annulus. The successful ablation site had a ventriculoatrial (VA) interval of 120 and 130 ms, respectively, and was located at the posterolateral and lateral aspects of the mitral annulus. Following ablation, there was no VA conduction; however, conduction through the normal atrioventricular pathway was noted during isoproterenol infusion in both patients. There was no induction of tachycardia. This study demonstrates that the permanent form of junctional tachycardia in adults can incorporate a concealed left free wall accessory pathway with a decremental property. Radiofrequency ablation therapy is effective and safe in this form of arrhythmia. (Chest 1995; 107:4145)

VA=ventriculoatrial

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In 1967, Coumel et al1 described a permanent form of junctional tachycardia characterized by a retrograde P wave and a R-P interval that is longer than the P-R interval. This form of tachycardia occurs primarily in infants and children, and it is frequently incessant and resistant to antiarrhythmic drugs. Two mechanisms have been demonstrated to be responsible for this tachycardia. The first mechanism involves reentry using a fast atrioventricular nodal pathway for anterograde conduction and a slow atrioventricular nodal pathway for retrograde conduction.2 The second mechanism involves reentry using the normal pathway for anterograde conduction and a concealed slowly conducting accessory pathway for retrograde conduction.3-5 Previous studies have consistently shown that the slowly conducting accessory pathway in the permanent form of junctional tachycardia is located in the parasepal area at the posterior interatrial septum. However, Okumura et al,6 in 1986, reported two patients with the permanent form of junctional tachycardia who had a left lateral location of a concealed slowly conducting accessory pathway. Recently, Ticho et al7 reported variable locations of the concealed slow accessory pathway that conducts only in retrograde direction in eight young patients with the permanent form of junctional tachycardia. In three patients the accessory pathway was located in the ostium of the coronary sinus, in one it was located in the right posteroseptal region just outside the ostium of the coronary sinus, in two it was located in the right atrial free wall, in one it was located in the right anterior septum, and in one it was located in the left posterior region just outside of the septal region.

Transcatheter ablation therapy is the current preferred therapy in many forms of supraventricular and ventricular tachycardias. It is also effective in the treatment of the permanent form of junctional tachycardia.7,8 In this study, two patients were reported with atrioventricular reentry tachycardia.
incorporating a concealed left free wall accessory pathway with a decremental conduction property, which was successfully ablated by application of the radiofrequency current.

**Materials and Methods**

Included in this study were two patients with a permanent form of junctional tachycardia, which utilized the normal pathway for anterograde conduction and a left free wall concealed accessory pathway with a decremental property for retrograde conduction. The study protocol was reviewed and approved by the institutional review board and was in accordance with local ethical standards. Both patients underwent complete anterograde and retrograde electrophysiologic studies in the drug-free state after discontinuation of cardiac drugs for three half-lives. Multiple electrode catheters with 1-cm interelectrode spacing were positioned in the high right atrium, in the coronary sinus, across the tricuspid valve, and in the right ventricular apex for recording of intracardiac electrogram and pacing. Surface and intracardiac electrograms were simultaneously displayed and recorded on a multichannel oscilloscopic recorder (Electronics for Medicine, VR-16, White Plains, NY) at a paper speed of 100 or 150 mm/s. The pacing stimuli were approximately twice the diastolic threshold in strength, 2 ms in duration, and were provided by a programmable digital stimulator (Bloom and Associates, DTU-200, Reading, Pa.). Conduction intervals and refractory periods were measured and defined according to the previously described criteria. Diagnosis of atrioventricular reentry tachycardia was made as previously described.3

Radiofrequency ablation therapy was performed at the completion of the electrophysiologic studies. A 7F quadrripolar steerable electrode catheter with a 4-mm distal electrode and a 2-mm interelectrode spacing of the distal 2 electrodes (Mansfield-Webster, Watertown, Mass.) was introduced percutaneously into the right femoral artery and advanced to the ventricular aspect of the mitral annulus at the site of the accessory pathway insertion. The radiofrequency current was delivered as a continuous, unmodulated sine-wave energy at a frequency of 500 kHz from a commercially available electrocautery unit (Radionics RFG-3C, Burlington, Mass.). The current was delivered between the tip electrode and a cutaneous dispersive pad that was applied to the left posterior chest. It was delivered to a site where the ventriculoatrial (VA) interval was the shortest and where discrete atrial and ventricular electrograms with an appropriate ratio were present. It was delivered under continuous digital monitoring of the power level and the impedance. Ventricular pacing was performed immediately after each application to determine if the ablation was successful. If the ablation was successful, a booster dose was administered one or two additional times at the same site. A complete electrophysiologic study was performed with and without isoproterenol infusion to ensure the success of ablation. The patient was then observed in the hospital for 3 days with measurements of serum creatine phosphokinase, a two-dimensional echocardiographic examination, and a 24-h Holter monitor recording. They were then discharged to the clinic for follow-up examinations. A repeat electrophysiologic study was scheduled 2 to 3 months later.

**Case Reports**

**Case 1**

A 37-year-old woman with a 10-year history of paroxysmal supraventricular tachycardia was referred to this institution for radiofrequency ablation therapy because of increasing frequency and severity of tachycardia attacks in the past year. Examinations revealed an absence of structural heart disease. A resting ECG showed normal P wave, P-R interval, and QRS complex duration. An ECG during tachycardia displayed a rate of 155 beats per minute, with a narrow QRS complex. The P wave was negative in leads 3 and aVF, biphasic (initially positive and then negative) in leads 1, 2, and V6, and positive in leads aVL and V1 through V3. It occurred before the QRS complex and had a P-R/R-P interval ratio of 0.82.

An electrophysiologic study revealed a sinus cycle length of 840 ms, an A-H interval of 80 ms, and an H-V interval of 40 ms. The longest atrial paced cycle length that caused second-degree atrioventricular nodal block was 360 ms. Measurement of the anterograde effective refractory period was limited by the atrial functional refractory period of 260 ms. A retrograde study revealed an eccentric atrial activation sequence suggesting the presence of a left-sided accessory pathway; the earliest atrial activation was registered from the posterolateral aspect of the mitral annulus at the distal coronary sinus. Decremental conduction with a maximum increment in VA interval of 60 ms was demonstrated during incremental ventricular pacing and ventricular extrastimulus testing. The longest ventricular paced cycle length that induced retrograde Wenckebach block in the accessory pathway was 280 ms. The retrograde effective refractory period of the accessory pathway was 380 ms. Nonsustained atrioventricular reentry tachycardia lasting for three beats was inducible with atrial or ventricular extrastimulus. Sustained atrioventricular reentry tachycardia was inducible after intravenous infusion of isoproterenol (2 μg/min). At this time, the cycle length of the tachycardia was 360 ms; the A-H interval, 125 ms; and the H-V interval, 235 ms (Fig 1, A). The earliest atrial activation was registered from the posterolateral aspect of the mitral annulus. The longest atrial paced cycle length that caused second-degree atrioventricular nodal block shortened to 240 ms, and the longest ventricular paced cycle length that induced Wenckebach block in the accessory pathway shortened to 270 ms. An intravenous bolus of 10 mg of adenosine triphosphate reproducibly terminated the tachycardia within 2.5 s due to a block in the accessory pathway. A progressive lengthening of the VA interval from 145 to 170 ms was noted before termination.

A radiofrequency current of 29 W for 15 s was delivered to the site of earliest atrial activation during tachycardia at the ventricular aspect of the posterolateral mitral annulus (Fig 1, B). It resulted in immediate termination of the tachycardia. The successful ablation site exhibited a VA interval of 120 ms (Fig 1, A). After this, two additional booster applications of the current at the same site were given. The fluoroscopic exposure time was 13 min and the total procedure time was 100 mins. A ventricular pacing study showed no VA conduction after ablation (Fig 1, C). With isoproterenol infusion, retrograde conduction through the normal atrioventricular pathway was noted; the longest ventricular paced cycle length that caused second-degree AV block was 360 ms. A single fast-slow form atrioventricular nodal reentry echo was inducible with ventricular extrastimulus. However, there was no induction of tachycardia. A repeat electrophysiologic study performed 74 days later revealed 1:1 ventricular-atrial conduction through the normal pathway at a paced cycle length of 620 ms. There was no evidence of retrograde accessory pathway conduction and no induction of tachycardia. A follow-up observation over a period of 17 months showed no recurrence of arrhythmia.

**Case 2**

A 33-year-old man with a 5-year history of paroxysmal supraventricular tachycardia was referred to this institution for radiofrequency ablation therapy because of frequent attacks of tachycardia. Examinations revealed an absence of structural heart disease. A resting ECG showed normal P wave, P-R interval, and QRS complex duration. An ECG during tachycardia displayed a rate of 205 per minute with a narrow QRS complex. The P wave,
which was superimposed on the T wave, was negative in leads 2, S, and aVF, birefringent in lead V6, and positive in leads aVL and V1; the P-R/R-P-P interval ratio was 0.36.

An electrophysiologic study revealed a sinus cycle length of 860 ms, an A-H interval of 80 ms, and an H-V interval of 35 ms. The longest atrial paced cycle length that produced second-degree atrioventricular nodal block was 860 ms, and the anterograde effective refractory period of the atrioventricular node was 340 ms. A retrograde study revealed an eccentric atrial activation sequence suggesting the presence of a left-sided accessory pathway. The earliest atrial activation was registered from the distal coronary sinus. Decremental conduction with a maximum increment in VA interval of 90 ms was noted in the accessory pathway during incremental ventricular pacing and ventricular extrastimulus testing. The longest ventricular paced cycle length that produced Wenckebach block in the accessory pathway was 380 ms. The retrograde effective refractory period of the accessory pathway was 340 ms. Atrioventricular reentry tachycardia was inducible with rapid ventricular pacing and with ventricular extrastimulus testing. The tachycardia had a cycle length of 380 ms, an A-H interval of 125 ms, an H-V interval of 40 ms, and a VA interval of 215 ms (Fig 2, A). The VA interval of the beat with a left bundle branch block morphology during tachycardia was 70 ms longer than that of the beat with a normal QRS complex morphology. The earliest atrial activation during tachycardia was registered from the lateral aspect of the mitral annulus at the distal coronary sinus similar to that during ventricular pacing (Fig 2, A). An intravenous bolus of 5 to 10 mg of adenosine triphosphate reproducibly terminated the tachycardia due to a block in the atrioventricular node.

The radiofrequency current was delivered to the earliest atrial activation site during tachycardia at the ventricular aspect of the lateral mitral annulus. The first two attempts were unsuccessful. The third attempt with a power strength of 26 W and a duration of 15 s terminated the tachycardia immediately (Fig 2, B). The successful ablation site exhibited a VA interval of 130 ms (Fig 2, A). One additional booster current was applied at the same site after successful application. The fluoroscopic exposure time was 12 mins and the total procedure time was 165 mins. There was no VA conduction after ablation (Fig 2, C). However, retrograde conduction through the normal atrioventricular pathway was noted during isoproterenol infusion (2 µg/min). The longest ventricular paced cycle length that produced a block in the normal pathway was 360 ms. There was no induction of tachycardia. A repeat electrophysiologic study performed 82 days later revealed 1:1 retrograde conduction through the normal pathway at a ventricular paced cycle length of 700 ms. There was no evidence of accessory pathway conduction and no induction of tachycardia. A follow-up observation over a period of 7 months showed no recurrence of arrhythmia.

**DISCUSSION**

**Accessory Pathway With Decremental Conduction**

The accessory pathway may occasionally manifest with a decremental property resembling atrioventricular nodal conduction.3-5,9,10 When this node-like accessory pathway conducts in an anterograde fashion, it usually presents with paroxysmal tachycardia with a left bundle branch block morphology.10 This type of accessory pathway is typically located at the anterior medial aspect of the tricuspid ring connecting the right atrium and the right ventricular myocardium or the distal right bundle branch.11 However, other locations around the tricuspid ring have been shown.12 In contrast, the concealed accessory
pathway that conducts in retrograde fashion and with a decremental property usually is located at the paraseptal region near the posterior interatrial septum. It presents with a special form of paroxysmal tachycardia with a long R-P interval and a short P-R interval. The presence of a retrograde left free wall accessory pathway with node-like property was first suggested by Denes et al in 1979. They reported a patient with two atrioventricular reentry tachycardias using the normal atrioventricular pathway for anterograde conduction and two concealed left free wall accessory pathways, a fast and a slow pathway with identical atrial activation sequence, for retrograde conduction. Longitudinal dissociation of a single node-like accessory pathway was proposed for this patient. Subsequently, Okumura et al described two patients with the permanent form of atrioventricular reentry tachycardia using the normal pathway for anterograde conduction and a slow left free wall accessory pathway for retrograde conduction. Lerman et al noted two patients with the permanent form of junctional tachycardia having a slowly conducting retrograde accessory pathway located at the left free wall. Ticho et al noted that in five of the eight pediatric patients with the permanent form of junctional tachycardia, the slowly conducting retrograde accessory pathway was located in sites other than at the usual posteroanterior area. It was in the right free wall in two patients, in the right anterior septum in one patient, and in the left free wall in one patient. The pharmacologic responses of the slowly conducting concealed retrograde accessory pathway are unclear. In the two patients reported by Okumura et al, verapamil prolonged the conduction time of the accessory pathway and prevented induction of tachycardia in one of the two patients. Lerman et al found that adenosine caused a block in the retrograde accessory pathway and terminated the permanent form of junctional tachycardia in five patients while verapamil caused a block in the retrograde accessory pathway and terminated the tachycardia in only two of the five patients. They suggested that there are two types of retrograde concealed slow accessory pathways, one composed of depressed fast-sodium channel tissue and the other consisting of node-like tissue. The response to adenosine in one of our two patients is consistent with the finding of Lerman et al.

**Catheter Ablation in Accessory Pathway With Decremental Conduction**

Transcatheter ablation is the preferred therapy in many forms of supraventricular and ventricular tachycardias. Experience with catheter ablation in patients with atrioventricular reentry tachycardia incorporating a concealed retrograde slow accessory pathway is limited. Using direct current shocks, Gang et al successfully ablated a retrograde slow accessory pathway with delivery of the current to the ostium of the coronary sinus in one patient. However, Smith et al successfully ablated the retrograde slow accessory pathway in only one of their four patients with the permanent form of junctional tachycardia when the direct current shocks were delivered to the coronary sinus electrode that recorded the earliest atrial activation. In contrast, Chien et al successfully ablated the retrograde slow accessory pathway in five of their six patients with the permanent form of junctional tachycardia when direct current shocks...
were delivered to the electrode positioned just outside the ostium of the coronary sinus; the remaining patient had a complication swing to complete heart block. Using radiofrequency current, Ticho et al \(^7\) successfully ablated the retrograde slow accessory pathway at various locations that included the typical posteroseptal region and other locations around the mitral and tricuspid rings in eight patients with the permanent form of junctional tachycardia. The successful ablation of the retrograde slow pathway in the two patients in the present study is complimentary to the study of Ticho et al. \(^7\) Both studies showed that the retrograde concealed slow accessory pathway can be ablated effectively and safely by application of a radiofrequency current. This study, as well as the studies of Okumura et al, \(^6\) Lerman et al, \(^13\) and Ticho et al, \(^7\) also indicates that the atypical insertion site in retrograde concealed slow accessory pathway, other than the typical paraseptal site, is not uncommon in patients with the permanent form of junctional tachycardia, and this possibility should be considered in conducting radiofrequency ablation in these patients.

REFERENCES