Electrophysiological Findings and Long-term Follow-up of Patients With the Permanent Form of Junctional Reciprocating Tachycardia Treated by Catheter Ablation

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Background. The permanent form of junctional reciprocating tachycardia (PJRT) commonly presents as recurrent drug-refractory, narrow-complex tachycardia. We studied the efficacy and safety of catheter ablation in treating these patients.

Methods and Results. Six patients with the diagnosis of PJRT were treated at our institution with direct-current catheter ablation. The study cohort comprised three men and three women with a mean age of 33.8±4.5 years. The mean time from onset of symptoms to ablation was 129±44.7 months. All failed multiple drug therapy (mean number of drugs failed was 5.3±0.5). The left ventricular ejection fractions were calculated by echocardiography and were greater than 60% in all except two patients, whose ejection fractions were 25% and 32%. Symptom duration was significantly longer in those with depressed ejection fraction compared with normal patients (258 versus 64.5 months, p<0.01). Electrophysiological findings revealed evidence of an atrioventricular reciprocating tachycardia involving retrograde decremental conduction over an accessory pathway localized to the posteroseptal area. Five patients received two direct-current shocks (250±16.7 J per shock) via paired electrodes from a catheter positioned just outside the coronary sinus or to a patch placed between the scapulae or on the anterior chest wall. One patient received a single direct-current shock of 300 J. The only complication was the development of complete atrioventricular block in one patient. This patient had previously undergone permanent pacemaker insertion for the sick sinus syndrome. The mean hospital stay after ablation was 2.2 days. Mean peak creatinine phosphokinase after ablation was 352±58.1 units/l and the MB fraction was 12±2%. Follow-up echocardiograms or gated nuclear studies showed improvement of ejection fraction in the two patients who presented with depressed ejection fractions. After a mean follow-up of 35.8±10.3 months, all patients remained free of tachycardia without antiarrhythmic drugs.

Conclusions. We conclude that catheter ablation by using direct current energy appears to be an effective treatment in patients with PJRT. (Circulation 1992;85:1329–1336)

KEY WORDS • tachycardia • ablation • cardiomyopathy • electrophysiology

The permanent form of junctional reciprocating tachycardia (PJRT) commonly presents as a frequently recurring tachycardia in childhood1 and may persist into adulthood.2 The arrhythmia is usually refractory to drug therapy, and these patients are at risk of developing tachycardia-induced cardiomyopathy.3 Therefore, early recognition and surgery have been recommended for those resistant to drug treatment. Catheter ablation has been used to treat a variety of supraventricular4 and ventricular tachycardias5 including those involving an atrioventricular (AV) bypass tract.6,7 In this report, we discuss the electrophysiological findings of this form of arrhythmia and review our long-term experience using a catheter technique for ablation of the accessory pathway in six consecutive patients with the diagnosis of PJRT. We conclude that catheter ablation appears to be relatively safe and effective in this group of patients and offers an excellent alternative to surgery.

Methods

Study Patients

Since 1985, six patients with the diagnosis of PJRT were treated at our institution. The electrocardiographic characteristics of the tachycardia in all patients were consistent with the diagnosis of PJRT. An example shown in Figure 1 reveals a narrow-complex tachycardia at rate of 140 beats per minute. There is a P wave preceding each QRS complex and the PR interval is less than RP interval. The P wave during tachycardia is negative in leads II, III, aVF, and V6 but positive in V1. Patient characteristics are shown in Table 1. The mean age was 33.8±4.5 years. Without medication, all except...
TABLE 1. Patient Characteristics and Follow-up

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age/sex</th>
<th>CHF class</th>
<th>Duration (mo)</th>
<th>Initial EF (%)</th>
<th>Meds failed</th>
<th>Symptoms</th>
<th>Follow-up time (mo)</th>
<th>Follow-up symptoms</th>
<th>Follow-up EF (%)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>45/M</td>
<td>II</td>
<td>312</td>
<td>25</td>
<td>q/d/β/pr  palp/syncope</td>
<td>68</td>
<td>None</td>
<td></td>
<td>33</td>
<td>Had VVI pacer for sss</td>
</tr>
<tr>
<td>2</td>
<td>29/F</td>
<td>I</td>
<td>48</td>
<td>&gt;60</td>
<td>q/β/V/d/n/p/l palp/fatigue</td>
<td>60</td>
<td>None</td>
<td></td>
<td>NA</td>
<td>Had follow-up EP study</td>
</tr>
<tr>
<td>3</td>
<td>25/M</td>
<td>I</td>
<td>204</td>
<td>32</td>
<td>q/n/l/d/p/β palp</td>
<td>44</td>
<td>None</td>
<td></td>
<td>&gt;60</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>23/F</td>
<td>I</td>
<td>42</td>
<td>&gt;60</td>
<td>q/d/β/pr/l/a palp/syncope</td>
<td>22</td>
<td>None</td>
<td></td>
<td>&gt;60</td>
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<tr>
<td>5</td>
<td>50/M</td>
<td>I</td>
<td>120</td>
<td>&gt;60</td>
<td>q/β/d/l/n palp/fatigue</td>
<td>10.5</td>
<td>Skipped beats</td>
<td></td>
<td>&gt;60</td>
<td>Exercise-induced tachycardia</td>
</tr>
<tr>
<td>6</td>
<td>31/F</td>
<td>I</td>
<td>48</td>
<td>&gt;60</td>
<td>β/n/l/d palp/fatigue</td>
<td>10</td>
<td>None</td>
<td></td>
<td>&gt;60</td>
<td></td>
</tr>
</tbody>
</table>

Average (±SEM) 33.8±4.5 129±44.7 5.3±0.5 35.8±10.3

CHF, coronary heart failure; EF, left ventricular ejection fraction; Meds, medications; M, male; F, female; q, quinidine; n, disopyramide; f, flecainide; d, digoxin; p, procainamide; β, β-blocker; pr, propafenone; a, amiodarone; v, verapamil; palp, palpitations; NA, not available; VVI, ventricular-inhibited; sss, sick sinus syndrome; EP, electrophysiology.

one patient had frequent tachycardia of long duration. The remaining patient developed persistent tachycardia with exercise. All had been treated with multiple drugs (mean, 5.3±0.5), including propafenone in two patients, flecainide in five patients, and amiodarone in one patient; they continued to be symptomatic despite maximal medical therapy. All patients had palpitations, and two had syncope. The mean duration of symptoms before referral for catheter ablation was 129 months. Ejection fraction was measured by echocardiography using biplane planimetry during sinus rhythm, a method validated by previous studies from this institution, in all patients except patient 1, who had follow-up gated nuclear studies at the referring hospital. The left ventricular ejection fraction was normal except for two patients who had significantly longer duration of symp-

toms compared with patients with normal ejection fractions (258 versus 64.5 months, p<0.01); they were otherwise healthy except for one patient who had a ventricular-inhibited (VVI) pacemaker implanted for sick sinus syndrome. Three of the six patients were previously presented as part of our overall experience with direct-current (DC) catheter ablation of posteroseptal accessory pathways. In this study, we expand the series and provide electrophysiological findings as well as long-term follow-up for patients with PJRT.

Electrophysiology Study

Patients were studied in the postabsorptive, drug-free state. Quadrupolar electrode catheters were inserted percutaneously and positioned at the high lateral right
atrium, right ventricular apex (RVA), and across the tricuspid valve for His bundle recording. A new quadripolar electrode catheter (1-cm interelectrode distance) with central lumen (Bard Electrophysiology, Tewksbury, Mass.) was placed in the coronary sinus. The coronary sinus os was located by contrast injection into the catheter. During tachycardia, premature ventricular depolarizations (PVDs) were introduced from the RVA in all patients and the right ventricular summit (RVS) in two patients to assess their effects on the subsequent atrial electrogram. Pacing from the RVS was accomplished by pacing from the distal electrode pair recording the largest His bundle deflection. We have previously introduced this technique as a method for distinguishing septal from free wall accessory pathways; it was used in two patients in this study who failed to show advancement of the atrial deflection in response to PVD introduced when the His bundle was refractory during tachycardia. Mapping of the earliest site of retrograde atrial activation during tachycardia was performed using the coronary sinus catheter and a modified Brockenbrough catheter (Elecath, Rahway, N.J.) positioned at various sites around the tricuspid annulus. The study was designed to exclude an atrial tachycardia and to show the presence of a retrogradely conducting accessory pathway that was part of the tachycardia circuit.

**Catheter Ablation**

All ablations were performed in the electrophysiology laboratory immediately after diagnostic studies except for one patient, when it was performed 1 day later. Under general anesthesia, DC shocks were delivered via the paired proximal electrodes on the coronary sinus catheter positioned just outside the coronary sinus os (cathode) and a skin patch (anode) placed between the scapulae in five patients and on the anterior chest wall in one patient. The shocks were delivered from a standard defibrillator (Lifepak 6, Physiocontrol, Redmond, Wash.). In the first three patients, the central lumen coronary sinus catheter was replaced with a new 6F quadripolar electrode catheter (USCI, Billerica, Mass.) for ablation. In the last three patients, the same coronary sinus catheter with central lumen used for mapping was used for ablation. An initial shock of 300 J was delivered because this amount of energy was associated with higher success rate in the ablation of AV junction. When the first shock was successful in eliminating the conduction over the accessory pathway, a second shock was given in the same manner 10–15 minutes later to decrease the possibility of return of accessory pathway function. After the development of complete AV block in patient 1, an initial shock of 200 J was used in the next patient. Since then, the protocol was changed to use a 300-J first shock followed by a 200-J second shock. In patient 6, a second shock was not given because of the slow return of AV conduction.

**Follow-up**

Serial creatinine phosphokininase (CPK) with MB isoenzymes were obtained. Echocardiograms were done the day after catheter ablation and at varying intervals thereafter in five of the six patients. One patient had follow-up gated nuclear study at the referring hospital. Twenty-four-hour Holter monitors were done in asymptomatic patients. All patients were encouraged to return for follow-up electrophysiology study. However, most patients declined because of lack of clinical symptoms, and only patient 2 had repeat electrophysiology study 4 months later.

**Results**

**Electrophysiology Study**

Three patients were in almost continuous tachycardia during the study. One patient had no inducible tachycardia at baseline but had persistent tachycardia with isoproterenol infusion. The electrophysiological findings are summarized in Table 2. There was no ventricular preexcitation with right atrial or coronary sinus pacing. The mean tachycardia cycle length was 431 msec, and intracardiac recordings during tachycardia showed the earliest site of retrograde atrial activation at the coronary sinus os. In all patients, retrograde ventricular conduction time during tachycardia was long (range, 280–350 msec). The importance of inserting critically timed PVDs during tachycardia when the His bundle is refractory has been emphasized by others. A PVD introduced while the His bundle is refractory that abbreviates the atrial cycle length demonstrates the presence of an accessory pathway. In addition, if the atrial activation sequence is identical to that during tachycardia, it is likely that the accessory pathway is part of the tachycardia circuit. This was observed in four of the six patients with pacing from the RVA. In the other two patients, PVDs introduced from

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**Table 2. Electrophysiology Study/Ablation**

<table>
<thead>
<tr>
<th>Patient</th>
<th>SVT CL (msec)</th>
<th>VA (msec)</th>
<th>Energy (J)</th>
<th>Shocks</th>
<th>Preablation AVBCL</th>
<th>Postablation AVBCL</th>
<th>VABCL</th>
<th>CPK units/l (MB%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>400</td>
<td>280</td>
<td>300/300</td>
<td>2</td>
<td>300</td>
<td>Junctional</td>
<td></td>
<td>354 (17%)</td>
</tr>
<tr>
<td>2</td>
<td>460</td>
<td>NA</td>
<td>200/200</td>
<td>2</td>
<td>250</td>
<td>340</td>
<td>N</td>
<td>144 (10%)</td>
</tr>
<tr>
<td>3</td>
<td>400</td>
<td>305</td>
<td>300/200</td>
<td>2</td>
<td>&lt;400</td>
<td>500</td>
<td>N</td>
<td>465 (8%)</td>
</tr>
<tr>
<td>4</td>
<td>520</td>
<td>350</td>
<td>300/200</td>
<td>2</td>
<td>&lt;520</td>
<td>500</td>
<td>N</td>
<td>468 (13%)</td>
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<tr>
<td>5</td>
<td>325A</td>
<td>300</td>
<td>300/200</td>
<td>2</td>
<td>330A</td>
<td>&lt;500A</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>6</td>
<td>480</td>
<td>300</td>
<td>300</td>
<td>1</td>
<td>&lt;480</td>
<td>600</td>
<td>N</td>
<td>338 (10%)</td>
</tr>
</tbody>
</table>

Average (±SEM) 431±29 307±12 352±58 (12±2%)

SVT CL, supraventricular tachycardia cycle length; VA, ventriculoatrial time during tachycardia; AVBCL, atrioventricular block cycle length; VABCL, ventriculoatrial block cycle length; CPK, creatinine phosphokinase; NA, not available; N, no ventriculoatrial conduction; Δ, on isoproterenol infusion.
the RVA failed to shorten the atrial cycle length (Figure 2, top panel). In both of these patients, PVDs were introduced from the RVS because this area was thought to be closer to the ventricular insertion site of the accessory pathway. In both patients, PVDs introduced at the summit while the His bundle was refractory curtailed the atrial cycle length and showed the presence of an accessory pathway (Figure 2, bottom panel). In addition, in one of these patients the presence of a retrogradely conducting accessory pathway was proved in an alternative manner. PVDs delivered from the RVA when the His bundle was refractory resulted in consistent delay in the inscription of the next atrial electrogram (Figure 3). A PVD that delays the next atrial electrogram while the His bundle is refractory shows the presence of an accessory pathway with decremental properties.

In all patients, the earliest area of atrial activation was localized to the os of the coronary sinus. In addition, all showed evidence of decremental accessory pathway conduction either during insertion of PVDs during tachycardia (Figure 3) or with programmed ventricular pacing during ventricular overdrive pacing (Figure 4). In three patients, atrial tachycardia could be excluded by a PVD introduced during tachycardia that terminated the tachycardia without return to the atrium (Figure 5). In two patients, atrial tachycardia was excluded by the delay of the next atrial electrogram by a PVD during tachycardia. In one patient (patient 5) with a history of exercise-induced incessant tachycardia, typical PJRT could only be induced after infusion of isoproterenol. Although AV nodal-blocking drugs, e.g., β-blocker or adenosine, may unmask the antegrade conduction of the pathway, these were not used in our study.

**Catheter Ablation**

Catheter ablation using DC energy was performed in all six patients. Five patients received two DC shocks and one patient received a single 300-J shock (Table 2). Transient AV block and sinus pause were observed in all patients and resolved in less than 10 minutes in five of the six patients. Patient 1 developed chronic complete AV block with a junctional rhythm at 60 beats per minute after catheter ablation. This patient had been previously treated with a VVI pacemaker for sinus node dysfunction. Programmed stimulation was repeated within 30 minutes after the ablative procedure. Isoproterenol infusion was used only in patient 5, who had a history of exercise-induced tachycardia. All the other patients had easily inducible tachycardia before ablation. There was no baseline ventriculoatrial conduction and no inducible tachycardia in any patients after ablation. Patient 5 similarly showed no ventriculoatrial conduction or inducible tachycardia even with isoproterenol infusion. Conceivably, retrograde ventriculoatrial conduction might have been present in the other patients with isoproterenol infusion. In addition, it is conceivable that return of retrograde conduction occurred after the acute barotraumatic effects of the shock.

**Complications**

The only significant complication was the development of third-degree AV block in patient 1. This patient received the highest (stored) energies (300 J twice). No patients developed pericarditis or tamponade. There was a mean CPK rise of 352 units/l (12±2% MB) after ablation. The normal range of CPK for our laboratory is 17–267 units/l (0–6% MB). Echocardiograms performed the next day showed no pericardial effusion and no segmental wall motion abnormalities in any patients.

**Follow-up**

All patients were discharged within 2–3 days after the ablation procedure. After a mean follow-up of 35.8 months, no patients were receiving antiarrhythmic drugs. Follow-up ECGs showed sinus rhythm with normal AV conduction in all patients except patient 1, who had a ventricular paced rhythm. This patient later showed evidence of return of AV conduction with first-degree AV block by Holter monitoring 5½ years after ablation. He also had asymptomatic runs of narrow-complex tachycardia at a rate of 176 beats per minute and AV dissociation suggestive of junctional tachycardia. Patient 5 complained of intermittent “skipped beats” that correlated with premature atrial depolarizations on ambulatory monitoring. For the two patients who presented with depressed ejection fractions, serial echocardiograms or gated nuclear studies demonstrated progressive improvement of ventricular function in one patient (Table 1). In patient 3, the ejection fraction was 45%, 55%, and greater than 60% 1, 2, and 3 years after ablation, respectively. In patient 1, the ejection fraction was 32% and 33% 2 months and 5½ years after ablation, respectively. Patient 2 had repeat electrophysiology study done 4 months after the ablation procedure. During this study, the AV block cycle length was 290 msec and the ventriculoatrial block cycle length was 330 msec. There were no echo beats or inducible tachycardia using programmed stimulation.

**Discussion**

Although our series of patients primarily included patients referred to a tertiary medical center, this study is in agreement of previous reports of patients with PJRT showing arrhythmias that are persistent and often refractory to medical treatment. The electrocardiographic features of the tachycardia in our patients, i.e., RP interval greater than PR interval and a superior P wave axis, are typical for PJRT, but the atypical form of AV node reentry tachycardia or atrial tachycardia can present with similar findings; therefore, careful study is needed to establish the diagnosis. Patient 5 is particularly interesting because during electrophysiology study, there was no ventriculoatrial conduction at baseline, but with isoproterenol infusion, persistent orthodromic tachycardia occurred. This showed that the conduction property of the pathway could be very sensitive to changes in autonomic tone. This may explain why using AV sequential pacing to prevent tachycardia as suggested by Coumel is generally ineffective because of changes in autonomic tone.

**Right Ventricular Summit Stimulation During Tachycardia**

We confirmed that introduction of PVDs during tachycardia is particularly important in establishing a diagnosis of PJRT. However, in two patients, PVDs from RVA alone failed to enter the tachycardia circuit. Patient 6 had
FIGURE 2. Simultaneous recordings of surface leads V₁, I, II, and aVF together with high right atrium (HRA), His bundle electrogram (HBE), distal coronary sinus (CSd), proximal coronary sinus (CSp), and right ventricular apex (RVA). Top panel: A premature ventricular depolarization (PVD) from right ventricular apex when His bundle was refractory had no effect on the subsequent atrial electrogram in patient 5. Numbers denote atrial interval that remained unchanged after the PVD. Paced depolarization from RVA should have a superior axis. PVD shown has an inferior axis because of fusion between tachycardia and paced depolarizations. H, His bundle electrogram. Bottom panel: In the same patient, a PVD from the right ventricular summit (RVS) when the His bundle was refractory was able to advance the next atrial electrogram by 20 msec. Note left bundle branch block/inferior axis morphology of the PVD, which is compatible with stimulation from the RVS.
an electrophysiological study performed at another institution 1 year before referral. The study was interpreted as compatible with the atypical form of AV nodal reentry because of the lack of effect of PVDs introduced from RVA on the subsequent atrial deflection. Patient 6 continued to be symptomatic despite various drug combinations. On repeat study at our institution, RVA stimulation again failed to affect the tachycardia, but with stimulation from the RVS, we showed that a PVD inserted during His bundle refractoriness advanced the next atrial deflection. Similar findings occurred in patient 5 and are shown in Figure 2. Thus, RVS stimulation, because of its proximity to the tachycardia circuit, may enter the circuit compared with RVA stimulation and should be included in the electrophysiology study.

**Catheter Ablation**

As mentioned above, PJRT may be refractory to pharmacological treatment. Flecaenide appears to show promise in treating young patients but was only partially effective in one of the five patients tried on this medication. Cardiac electrosurgery appears to be a suitable treatment alternative for this condition. Both epicardial and endocardial approaches have been reported with good success.

Use of catheter ablation in treating PJRT in pediatric patients has been reported. However, in that report,

**FIGURE 3.** Recordings from patient 3 show the effect of premature ventricular depolarization (PVD) on atrial activation when the His bundle is refractory. Shown are the simultaneous recordings of surface leads V1, I, II, and aVF together with His bundle electrogram (HBE), distal coronary sinus (CSd), proximal coronary sinus (CSp), and blood pressure (BP) recordings. The first three complexes are recorded during tachycardia and associated with a relatively long ventriculoatrial interval. The earliest site of retrograde atrial activation occurred at CSp, which is positioned at the os. A PVD (S) introduced when the His bundle is refractory delayed the next atrial electrogram by 20 msec. Numbers denote atrial intervals in msec. H, His bundle electrogram; A, atrial electrogram.

**FIGURE 4.** Graph of programmed stimulation showing decremental retrograde conduction of the accessory pathway. In patient 5, the right ventricle was paced at constant cycle length of 600 msec and progressively premature ventricular extra-stimuli were introduced (V1V2). This resulted in progressively longer ventriculoatrial conduction time (V2A2), but the coronary sinus os remains the earliest site of retrograde atrial activation.
four of the five patients required permanent pacemaker insertion after ablation, and the remaining patient continued to require two antiarrhythmic medications for control of tachycardia. In adults, two cases have been reported of successfully treating PJRT with DC ablation of the AV junction, and these patients remained free of tachycardia with a follow-up of 2–31 months.2,21 Our prior experience (four patients)10 and the report by Gang et al22 are descriptions of successful ablation of the accessory pathway using DC shock. Using our ablation protocol, PJRT was successfully treated in all six of our patients. Only one patient developed complete AV block, and fortunately, this patient already had a permanent VVI pacemaker in place for treatment of the sick sinus syndrome. There are several possible predisposing factors for AV block in this patient: 1) He received the highest amount of energy delivered, and 2) he was the only patient in this series with anode placed on the anterior chest wall. This configuration is associated with longer duration of transient AV block and has been abandoned.10 In the other patients, although there was an increase in the AV node block cycle length after ablation (Table 2), none of these patients developed AV block on follow-up, which extended up to 60 months in one patient. In the one patient who had repeat electrophysiological study 4 months after ablation, there was improvement in the conduction through the AV node without inducible tachycardia. In the previous report,10 there was one patient who developed hemopericardium after DC shock for ablation of a posteroseptal pathway. This was thought to be due to insulation break in a previously used catheter causing part of the current to be delivered to the distal electrodes inside the coronary sinus. All of the catheters for ablation in our patients were new, and none of our patients developed this complication.

We and others22 have described the safety and effectiveness of radiofrequency ablation for accessory pathways in all locations. Present experience has shown that the radiofrequency approach has superceded use of DC shocks for pathway ablation, and we would currently recommend the use of radiofrequency current as the initial approach for patients with PJRT. To our knowledge, there is only one preliminary report describing the use of radiofrequency current for patients with PJRT.23 Selective ablation of retrograde conduction is compatible with previous suggestions that the retrograde limb of the tachycardia circuit is an extra AV nodal structure in close apposition to the os of the coronary sinus.24 Alternatively, the tachycardia circuit could involve an atrio-Hisian accessory pathway antegradely and AV node retrogradely.25 However, this mechanism was excluded in our patients because we found no evidence of an atrio-Hisian pathway, and ablation was directed well away from the AV junction. As expected, there was a rise of CPK–MB enzymes compatible with some myocardial damage, but echocardiogram afterward did not detect any segmental wall abnormalities or

**FIGURE 5.** Recordings from patient 3 showing termination of tachycardia by a premature ventricular depolarization (PVD) without conduction to the atria; first two are tachycardia beats. A PVD after the stimulus artifact (S) occurred after the atrial (A) electrogram (arrow), which was on time. This PVD terminated the tachycardia without conduction to the atria; this excluded the diagnosis of atrial tachycardia. HBE, H, His bundle electrogram; CSp, distal coronary sinus; Csd, proximal coronary sinus; BP, blood pressure.
damage to the heart valves. All patients were ambulatory the day after the procedure and discharged within 2 or 3 days. All patients continued to do well on a mean follow-up of 38.5 months.

**Tachycardia-Mediated Cardiac Dysfunction**

Persistent tachycardia is associated with the development of cardiac dysfunction. Two of our patients had decreased left ventricular function before ablation. They also had symptoms associated with tachycardia for a longer period of time. In one patient, cure of tachycardia was associated with a gradual improvement of ventricular function. This is in agreement with the reversibility of tachycardia-induced cardiomyopathy after surgical cure in patients with PJRT. In the other patient with the longest duration of symptoms before effective treatment, there was significant residual impairment of the left ventricular function (patient 1). This underscores the importance of recognizing this form of tachycardia and early application of definitive treatment even among patients who have established heart disease.

**Conclusions**

This report showed that 1) catheter ablation using DCC shocked appears to be effective in treating patients with PJRT and because of its low morbidity offers an excellent alternative to surgery. 2) Right ventricular summit stimulation during tachycardia is helpful in establishing diagnosis in certain patients. 3) Tachycardia-mediated cardiac dysfunction can be reversible after catheter ablation. 4) Our findings are compatible with the suggested mechanism that PJRT is an AV reciprocating tachycardia involving retrograde conduction over an extra AV nodal pathway with decremental conduction properties localized to the posteroseptal area in the region of the coronary sinuses.

**References**


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