The nonpharmacologic management of the permanent form of junctional reciprocating tachycardia

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ABSTRACT The permanent or recurring form of junctional reciprocating tachycardia (PJRT) is an incessant tachycardia that has characteristically been refractory to medical therapy. Nine patients with PJRT refractory to medical therapy were referred for evaluation and electrophysiologic study. All nine patients had the characteristic clinical and electrophysiologic features of PJRT. Each patient demonstrated near-incessant reciprocating tachycardia with a 1:1 atrioventricular (AV) relationship and with a retrograde P wave (P') occurring closer to the succeeding QRS complexes (i.e., long RP'). With initiation of the tachycardia, there was no prolongation of the PR or AH interval. All patients had evidence of early retrograde atrial activation in their posterior atrial septa and this retrograde limb had properties of decremental conduction. Eight of the nine patients underwent elective surgical ablation of the retrograde limb of tachycardia, and in seven it was successful. Epicardial and endocardial atrial maps recorded during PJRT demonstrated that the site of earliest retrograde activation was in the posterior atrial septum near the coronary sinus orifice. The seven patients in whom surgery was successful left the hospital in sinus rhythm with antegrade conduction, and all are free of tachycardia during the mean follow-up period of 31 months (range 1 to 70 months). In the two remaining patients PJRT was controlled by interruption of the antegrade limb of the tachycardia, the AV node–His bundle. In one patient this was done under direct vision at surgery after an unsuccessful attempt at pathway dissection. The other underwent closed-chest ablation of the AV node with a catheter. Both are well and left the hospital tachycardia free. This study demonstrates that PJRT, which is characteristically refractory to medical therapy, can be successfully managed by surgical ablation of the retrograde limb of the tachycardia.


THE PERMANENT or recurring form of junctional reciprocating tachycardia (PJRT) is so named because of its characteristic incessant nature and refractoriness to medical therapy. Although first reported by Gallavardin and Veil1 over 50 years ago, it was not until 1967 that Coumel et al.2 described the electrocardiographic features of the arrhythmia and named it “permanent” junctional RT. These authors pointed out that the arrhythmia most frequently affects the young, and was often mislabeled ectopic atrial tachycardia. The arrhythmia begins with no increase in the PR interval, and is distinguished by negative P waves (P') in leads II, III, and aVF, which occur closer to the succeeding QRS complex than to the preceding one. Since the report of Coumel et al., several investigators have demonstrated that the arrhythmia is a reciprocating tachycardia with antegrade limb in the atrioventricular (AV) node–His bundle.3–7 The long RP' interval led Coumel et al. to suggest that the retrograde limb had properties of slow conduction. The exact anatomic and physiologic nature of this retrograde limb remains unclear, but most studies have demonstrated that the retrograde limb is a septal ventriculoatrial (VA) structure with decremental functional properties. Gallagher and Sealy3 suggested that the retrograde limb represented an accessory AV node in the posterior septum, while Fárel et al.4 suggested that the limb was a posteroseptal accessory AV pathway with decremental functional properties.

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Most recent reports on PJRT have noted the refractoriness of the rhythm to medical therapy.1-8 Coumel8, 9 suggested a method of near-simultaneous AV pacing for control of the tachycardia, while Gallagher and Sealy3 reported a case in which surgical creation of complete antegrade heart block was used to terminate PJRT. In this study we report on nine patients with PJRT. The intraoperative mapping and results of surgical ablation of the retrograde limb of the tachycardia are reported in eight patients, seven of whom underwent successful dissection. In two of these nine patients tachycardia was abolished by interruption of the antegrade limb, either through direct intraoperative division or catheter ablation of the AV node. In addition, we present new information, derived from the electrophysiologic evaluation of the nine patients with PJRT, on the antegrade limb of the tachycardia.

Methods

The study comprised a total of nine patients (table 1). Patients 1 and 2 did not undergo operative ablation of the retrograde limb of the tachycardia. Patient 1 underwent operative division of the AV node–His bundle and was the subject of an earlier report.3 Patient 2 underwent closed-chest modification of the AV node–His bundle by the catheter ablation technique.10 Patients 3 through 9 underwent surgical ablation of the retrograde limb. Patients 8 and 9 were studied before surgery by Dr. Hein J. J. Wellens who referred the patients for elective surgical ablation of the retrograde limb. Electrophysiologic studies were performed according to techniques previously described.11, 12 A quadripolar catheter was placed in the coronary sinus and both bipolar and unipolar recordings were made. The left AV groove was mapped with the use of this catheter. A quadripolar catheter was placed in the right ventricular apex, while a tripolar catheter was positioned on the tricuspid ring to record His bundle activity. The right atrium was mapped with the use of a modified Brockenbrough catheter. In those patients who underwent surgery both epicardial and right atrial endocardial maps were recorded during reciprocating tachycardia.13, 14 The surgical techniques for dissection of the posterior septum have also been published previously.15, 16

Results

Table 1 lists the clinical characteristics of the patients in this study. The age range was from 5 to 54 years. All patients were referred because conventional medical therapy was unsuccessful in controlling symptoms or because of deteriorating cardiac function in the face of incessant tachycardia. Most patients had incessant tachycardia or tachycardia with short interludes of sinus rhythm. Most patients in the series had evidence of ventricular dysfunction when examined objectively, and in all patients arrhythmia control was poor over several years despite various and intensive antiarrhythmic therapies (table 1). All also displayed the classic electrocardiographic features of PJRT (figure 1), with long RP’ intervals and superiorly directed P’ waves.

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Sex/Age</th>
<th>Duration of tachycardia symptoms (type)</th>
<th>Symptoms</th>
<th>Function(3)</th>
<th>Tachycardia rate (per min)</th>
<th>RP’ RR</th>
<th>Drug history</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M 20</td>
<td>10 yr (incessant)</td>
<td>Class IV</td>
<td>2.8 l/min/m²</td>
<td>144</td>
<td>0.60</td>
<td>Q, D</td>
</tr>
<tr>
<td>2</td>
<td>F 54</td>
<td>&gt; 8 yr (incessant)</td>
<td>Class III–IV</td>
<td>22% EF (MUGA)</td>
<td>133</td>
<td>0.70</td>
<td>D, I, Q, N</td>
</tr>
<tr>
<td>3</td>
<td>F 5</td>
<td>5 yr (incessant)</td>
<td>CHF</td>
<td>18% EF (MUGA)</td>
<td>200</td>
<td>0.57</td>
<td>P, D</td>
</tr>
<tr>
<td>4</td>
<td>F 17</td>
<td>&gt; 6 yr (incessant)</td>
<td>Class III</td>
<td>Poor LV function (echo)</td>
<td>167</td>
<td>0.61</td>
<td>D, Q, D, N</td>
</tr>
<tr>
<td>5</td>
<td>M 6</td>
<td>6 yr (incessant)</td>
<td>CHF</td>
<td>10% EF (RNA)</td>
<td>176</td>
<td>0.74</td>
<td>D, P</td>
</tr>
<tr>
<td>6</td>
<td>F 24</td>
<td>8 yr (paroxysmal)</td>
<td>Syncope, dizziness</td>
<td>76% EF (RNA)</td>
<td>170</td>
<td>0.68</td>
<td>N, I, P, Q</td>
</tr>
<tr>
<td>7</td>
<td>M 19</td>
<td>12 yr (incessant)</td>
<td>Light-headedness (class II)</td>
<td>Normal echo</td>
<td>146</td>
<td>0.50</td>
<td>D, A</td>
</tr>
<tr>
<td>8</td>
<td>F 39</td>
<td>29 yr (paroxysmal)</td>
<td>Fatigue (class II)</td>
<td>EF 58% (RNA)</td>
<td>130</td>
<td>0.69</td>
<td>D, I, N, A</td>
</tr>
<tr>
<td>9</td>
<td>F 50</td>
<td>30 yr (paroxysmal)</td>
<td>Weakness, fatigue</td>
<td>Normal CXR</td>
<td>142</td>
<td>0.72</td>
<td>D, I, N, V</td>
</tr>
</tbody>
</table>

CHF = congestive heart failure; EF = ejection fraction; LV = left ventricular; Q = quinidine; D = digoxin; I = Inderal (propranolol); P = procainamide; A = amiodarone; V = verapamil; CXR = chest x-ray.

Cardiac function was assessed by various means including catheterization (cath), gated blood pool scan (MUGA), first-pass radionuclide angiogram (RNA), or echocardiogram (echo). Class is NYHA.
FIGURE 1. The 12-lead echocardiogram of PJRT. The rate is 150 per minute with an RP'/R-R of 0.75.

The P waves were negative in leads II, III, aVF, and V4-V6 and flat in lead I. This pattern is similar to that previously reported. The ratio of the interval between the R wave and the retrograde P wave (RP') to the cycle length (RR) was between 0.50 and 0.75. The range of the heart rate was between 130 and 200 per minute, with the faster rates occurring in the two children. In those instances in which the tachycardia was reinitiated after sinus rhythm, there was no increase in the PR interval of the first beat (figure 2).

The data from the electrophysiologic studies are listed in table 2. The range of cycle lengths during intermittent sinus rhythm was 280 to 700 msec, while the mean cycle length was $540 \pm 60$(SEM) msec. The AH interval was < 60 msec in all but two patients, both of whom had long tachycardia cycle lengths.

FIGURE 2. Leads I, II, III from patient 2 during interruption of PJRT. The tachycardia could be temporarily terminated by atrial or ventricular overdrive pacing. In this figure the tachycardia was temporarily terminated by esophageal pacing. Note that when the tachycardia resumes there is no increase in the PR interval.
During PJRT, the cycle length was 374 ± 22 msec, with an AH of 49 ± 8 msec. In general, and as demonstrated previously, 5 the AH interval during PJRT was shorter than that during intermittent sinus rhythm. During overdrive pacing at cycle lengths shorter than the tachycardia in patients 1 through 6, the AH interval was similar to that seen during PJRT, which is consistent with the concept that the upper junction of the circuit was formed, in part, by atrial tissue. All patients demonstrated long VA conduction times during PJRT (table 2). The sites of earliest retrograde atrial activation during overdrive pacing were found to be in the coronary sinus orifice and the low septal limb as measured on the His bundle electrogram (figure 3).

In each patient there was evidence of decremental conduction in the retrograde limb of the tachycardia. This was demonstrated as prolongation of the VA times, measured at the coronary sinus orifice during decremental ventricular pacing or, more importantly, by demonstrating increasing VA conduction time with increasing prematureity of either an atrial or ventricular programmed stimulus given during PJRT. Figure 4 illustrates the results of introduction of premature right atrial beats during PJRT with decreasing coupling intervals. As the ventricle was captured by the programmed atrial depolarization at increasingly premature intervals, there was an increase in the subsequent VA conduction time measured at the coronary sinus orifice. This increase in retrograde conduction time demonstrated during tachycardia suggests that decremental functional conduction exists in the retrograde limb of the tachycardia and helps distinguish a bystander phenomena. Also, in each patient the participation of this decrementally conducting pathway in the tachycardia was documented by the ability of right ventricular premature depolarization (VPDs) to preexcite the atrium during tachycardia at a time when the His bundle was refractory (table 2 and figure 3). The sequence of retrograde activation was not disturbed by the VPD, and the tachycardia was advanced. This observation is consistent with the presence of an accessory pathway and with a lower junction distal to the His bundle. 17 In each patient, single VPDs were easily capable of preexciting the atrium.

During intermittent sinus rhythm seven of the nine patients demonstrated AH intervals < 60 msec. This suggested that enhanced AV nodal conduction might be present. 17 High right atrial incremental overdrive pacing was therefore performed in patients 1 through 6. In patient 1, pacing was terminated before AV Wenckebach was observed to avoid atrial fibrillation. All six patients showed 1:1 AV conduction at cycle lengths ≤ 300 msec, with an increase in AH of 47 ± 12 msec over the value in subjects in sinus rhythm. The short AH interval in sinus rhythm, the ability to conduct at cycle lengths ≤ 300 msec, and the small increase in AH interval before AV Wenckebach block suggests that enhanced AV nodal conduction was present in these six patients. 18 However, it should be noted that enhanced AV node conduction is a functional definition, and may represent the response of AV nodal tissue to autonomic input.

The operative epicardial and endocardial maps for the seven patients who underwent surgery are shown in table 3. The site of earliest atrial epicardial activation during PJRT was uniformly found to be over the crux,
near the posterioatrial septum. The VA conduction times were somewhat faster than the minimum VA times recorded before surgery, probably reflecting the effects of general anesthesia. Table 3 demonstrates the endocardial VA times found near the anulus of the tricuspid ring. Listed are the VA times found in the low medial septum (near the His bundle deflection), the coronary sinus orifice, and the lateral right atrium. In all cases the minimum VA time was in the posterior septum near or at the coronary sinus orifice. To eliminate the possibility that the prolonged VA conduction was due to intraventricular conduction delay, the contiguous sections of the ventricular septum and base were mapped. Conduction in these areas demonstrated ventricular activation after the onset of the surface QRS, yielding a local VA interval between 103 and 214 msec. Figure 5 depicts the endocardial and epicardial activation sites in patient 4 during PJRT. This again demonstrates that the site of earliest retrograde activation was in the posterioatrial septum.

Dissection of the pathway was carried out as previously described, and involved standard dissection of the posterioatrial septum. All patients survived surgery. After surgery all patients but patient 1 were in sinus rhythm; in patient 1 the dissection was unsuc-

FIGURE 3. Intracardiac electrograms from patient 4. Displayed are surface leads I, II, III, V₁, and V₆. The bipolar intracardiac electrograms displayed are from the right ventricular apex, the high right atrium, the His bundle electrogram, and both the proximal and distal coronary sinus. The unipolar coronary sinus electrograms are also displayed. The coronary sinus catheter was positioned such that the proximal coronary sinus pole was near the coronary sinus orifice. The electrograms demonstrate the long VA times during PJRT and the early retrograde activation at the coronary sinus orifice. Also shown is the effect of a VPD. The VPD preexcites the atrium by 23 msec at a time when the His bundle is refractory. The sequence of retrograde activation is unchanged and the tachycardia is advanced.

FIGURE 4. The effect of a right atrial appendage premature depolarization (APD) on PJRT. The VA conduction time from the right ventricular apex to the coronary sinus orifice is plotted as a function of the increasing conduction time. The graph suggests decremental conduction.
TABLE 3  
Operative maps in PJRT (intervals in msec)

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>CL</th>
<th>Minimum VA</th>
<th>Local VA^a</th>
<th>Low medial RA</th>
<th>Endocardial</th>
<th>CS orifice</th>
<th>Lateral RA</th>
</tr>
</thead>
<tbody>
<tr>
<td>3</td>
<td>320</td>
<td>Crux — 200</td>
<td>133</td>
<td>208</td>
<td>192</td>
<td>229</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>360</td>
<td>Crux — 216</td>
<td>167</td>
<td>237</td>
<td>216</td>
<td>263</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>320</td>
<td>Crux — 236</td>
<td>159</td>
<td>267</td>
<td>225</td>
<td>258</td>
<td></td>
</tr>
<tr>
<td>6^</td>
<td>285</td>
<td>Crux — 155</td>
<td>103</td>
<td>185</td>
<td>138</td>
<td>195</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>350</td>
<td>Crux — 273</td>
<td>120</td>
<td>299</td>
<td>285</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>480</td>
<td>Crux — 236</td>
<td>—</td>
<td>253</td>
<td>224</td>
<td>242</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>460</td>
<td>Crux — 291</td>
<td>214</td>
<td>274</td>
<td>231</td>
<td>265</td>
<td></td>
</tr>
</tbody>
</table>

RA = right atrium; CS = coronary sinus.  
^aLocal VA (see text). In the area of the retrograde limb, the ventricle was activated shortly after the surface QRS, yielding a short local VA interval. This rules out an intraventricular conduction defect as the source of the conduction delay.  
^bRequired isoproterenol for operative map.

cessful and he later had operative ablation of the AV node. Seven patients demonstrated normal antegrade nodal function and left the hospital tachycardia free. Patient 8 was left with a PR interval of 0.20 sec, but had been treated with amiodarone.

Two patients underwent ablation of the AV node–His bundle for tachycardia control. Patient 1 represented the initial attempt at elective ablation of the retrograde limb, which was unsuccessful. Because of associated cardiomyopathy, he was judged a poor candidate for repeat septal dissection. Consequently, he underwent division of the AV node–His bundle under direct vision. Prompted by the recent report by Critelli et al.,^19 which demonstrated for the first time antegrade conduction over the accessory pathway only after closed-chest modification of the AV node, we reinvestigated our records, looking for evidence of antegrade posteroseptal conduction. We discovered a postoperative electrocardiogram for patient 1 that was recorded after the initial unsuccessful dissection (figure 6). The results were interpreted to indicate ventricular tachycardia but almost certainly represented transient ante-

**FIGURE 5.** The epicardial and endocardial map during PJRT from patient 4. The earliest epicardial activation is over the crux, while the earliest endocardial activation is in the posterior septum near the coronary sinus orifice.
grade posteroseptal conduction; the electrocardiogram is identical to that in figure 7 of Critelli et al. Also, an intraoperative trace recorded before AV node division shows antegrade pathway Wenckebach block in this rhythm (figure 7). A permanent pacemaker was implanted and he is doing well after 70 months with no further evidence for antegrade posteroseptal conduction. Patient 2 was also judged to be a poor operative candidate, and she subsequently underwent closed-chest modification of the AV node–His bundle by a recently introduced catheter technique. This patient required the addition of amiodarone for tachycardia control (the AV node was modified but not ablated), but has now been free of tachycardia for 7 months.

**Discussion**

PJRT is an uncommon and almost continuous form of reciprocating tachycardia characterized by an obligatory 1:1 AV relationship, with P’ waves occurring closer to the succeeding QRS complex than to the preceding one. The relationship of the P wave to the succeeding QRS complex produces a pattern that has been called a long RP’ tachycardia. Differential diagnosis of this tachycardia includes ectopic atrial tachycardia, the atypical form (fast-slow) of AV node reentry, atrial flutter with 2:1 conduction, and Ebstein’s anomaly with an accessory AV pathway.

The nine patients in this study had long RP’ tachycardias that were, in most cases, incessant. All nine patients demonstrated the classic electrocardiographic features of PJRT: rates between 120 and 250 per minute, superiorly directed waves with RP'/RR between 0.50 and 0.75, and no prolongation of the PR interval with initiation of the tachycardia. In this series the age range varied more than that described by Coumel et al., and included two older patients who were 50 and 54 years old. All patients were referred because their arrhythmias were intractable to medical therapy.

Each of the nine patients underwent detailed electrophysiologic studies that revealed the presence of a narrow complex reciprocating tachycardia with a 1:1 AV relationship. The antegrade limb of the tachycardia appeared to be the AV node–His bundle. During tachycardia all patients demonstrated earliest retrograde atrial activation in the posterioatrial septum, as measured near the coronary sinus orifice. In one patient, VA conduction times were equal in the coronary sinus orifice and low septal right atrium. This pattern of activation is consistent with retrograde conduction in the septum, and could be seen with either a septal accessory pathway or reentry in the AV node. However, in each patient right ventricular VPDs during PJRT were easily capable of preexciting the atrium at a time when the His bundle was refractory, without disturbing the pattern of retrograde atrial activation. This suggests that the retrograde limb of the tachycardia inserted distal to the His bundle, and excludes the possibility of AV nodal reentry.

Coumel’s original suggestion that the long retrograde activation time is due to decremental functional properties in the retrograde limb was supported in this study, as it has been in the reports of other investiga-
Decrement was suggested by the gradual prolongation of the VA times with rapid ventricular pacing and by VA prolongation with increasing prematurity of either atrial or ventricular test stimuli.

The initiation of tachycardia without an increase in the PR or AH interval when a critical sinus cycle length is reached is one of the hallmarks of PJRT. Coumel\textsuperscript{8} attributed this to preexisting decremental conduction in the retrograde limb with concealed antegrade conduction into the accessory pathway. In six and perhaps seven of the patients in this study, we found evidence of enhanced AV node conduction.\textsuperscript{19} Patients 1 and 3 through 7 had AH intervals < 60 msec. Patients 1 through 6 were capable of 1:1 AV nodal conduction at cycle lengths \( \leq 300 \text{ msec} \), with little increase in AH interval over that observed when they were in sinus rhythm. Even in patient 2 in whom the AH interval was initially 90 msec, there was 1:1 AV conduction to a cycle length of 280 msec, with an increase in AH of only 92 msec. Other investigators have made similar observations in patients with PJRT.\textsuperscript{4, 6, 7} It is possible that the combination of enhanced conduction in the antegrade limb of the tachycardia, the AV node–His bundle, coupled with slow conduction in the retrograde limb, is an important feature in the maintenance of the reciprocating tachycardia. This type of mechanism would be analogous to the fast-slow or atypical form of AV nodal reentry, which has also been described as incessant.\textsuperscript{20, 22} Recently, Benditt et al.\textsuperscript{23} demonstrated that enhanced AV node conduction may play an important role in reciprocating tachycardia in which a concealed AV pathway is used as the retrograde limb. Certainly though, enhanced AV node conduction may be a continuum of AV nodal physiology responsive to autonomic tone.

The epicardial and endocardial maps recorded at surgery support the concept that the retrograde limb is a posterior septal structure. In each patient, the earliest atrial epicardial activation was in the crux, over the posterior septum. The atrial maps confirmed that the posterior septum was activated before the low medial septum. Furthermore, isolated dissection of the posterior septum successfully terminated the tachycardia in each patient, while leaving antegrade conduction intact. The successful surgical termination of PJRT by this selective dissection is compelling evidence that the retrograde limb of PJRT is an isolated posterior septal structure. This selective dissection, however, does not settle the issue as to the exact physiologic nature of this AV pathway. The combination of enhanced antegrade AV nodal conduction in six and perhaps seven of the patients in this study and decremental conduction in the retrograde limb would be consistent with a congenital defect in the AV node and perhaps, as previously suggested, an accessory AV node.\textsuperscript{5} Certainly, as proposed by Farré et al.,\textsuperscript{4} the retrograde limb could be an accessory AV pathway with decremental functional properties. The anatomic basis of the slow conduction remains unclear.

Two of the patients in this series underwent modification of the antegrade limb of the tachycardia, the AV node–His bundle. In patient 1, surgical ablation of the AV node was done after an unsuccessful attempt at selective pathway division; the ablation was the subject of an earlier report.\textsuperscript{5} The second patient underwent closed-chest catheter ablation\textsuperscript{10} because she was a poor surgical risk. Both methods are available for patients with poorly tolerated tachycardia in whom prolonged cardiopulmonary bypass is thought injudicious. Initial attempts at catheter ablation of the AV node in this entity are interesting. Critelli et al.\textsuperscript{19} recently provided convincing evidence for new antegrade posteroseptal conduction after catheter ablation. Our own experience with catheter ablation has suggested similar phenomena.\textsuperscript{6} The relative difficulty in AV node modification would be consistent with a congenital AV node

malformation as originally proposed by Gallagher and Sealy. The usefulness of closed-chest modification of the AV node for treating PJRT needs to be investigated further. These methods provide an alternative to surgery in those patients whose conditions are refractory to medical therapy and who have associated cardiomypathy.

In this study the electrophysiologic characteristics of the PJRT were reviewed. The rhythm was defined as an almost incessant 1:1 AV reciprocating tachycardia, with prolonged VA conduction during the rhythm and early retrograde atrial activation at the coronary sinus orifice. It was also observed that, during the tachycardia, programmed VPDs could easily preexcite the atrium when the His bundle was refractory. Finally, the retrograde limb showed an increase in VA conduction time when programmed premature atrial or ventricular depolarizations were introduced into PJRT.

This study demonstrates that PJRT that is refractory to medical therapy can be successfully terminated by selective surgical ablation of the accessory pathway. We believe definitive surgery should be offered to individuals with incessant PJRT with medically refractory symptoms or with evidence of deteriorating ventricular function. At the very least, ventricular function should be closely monitored. In this study, seven of eight patients who underwent dissection of the posterior septum, after years of incessant tachycardia, had their arrhythmia terminated, leaving antegrade conduction intact. All seven patients left the hospital tachycardia free and in sinus rhythm and remain so for a mean follow-up period of 31 months. One of the eight surgically treated patients required reoperation for AV node dissection. This patient was one of two who underwent modification of the antegrade limb of the tachycardia and he also remains tachycardia free.

References
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