Cryosurgical treatment of atrioventricular node reentrant tachycardia*

JAMES L. COX, M.D., WILLIAM L. HOLMAN, M.D., AND MICHAEL E. CAIN, M.D.

ABSTRACT  Paroxysmal supraventricular tachycardia most commonly arises from reentry within the atrioventricular (AV) node. Although ablation of the His bundle has gained popularity for treating patients with AV node reentrant tachycardia refractory to medical therapy, undesirable sequelae include complete heart block and the necessity for a permanent pacemaker. To obviate this limitation, we have developed a discrete cryosurgical procedure that interrupts the reentrant circuit responsible for AV node reentrant tachycardia without blocking AV conduction. After first characterizing the salutary effects of this approach in experimental animals, we performed this procedure in eight patients with AV node reentrant tachycardia. Preoperative, intraoperative, and postoperative electrophysiologic studies were performed in each patient. Under conditions of normothermic cardiopulmonary bypass and during atrial pacing at a constant rate with continuous monitoring of AV conduction, nine separate 3 mm cryolesions (−60°C for 2 min) were placed at predetermined sites around the triangle of Koch in the lower right atrial septum. Postoperatively, each patient had a single AV node conduction curve. No patient had AV node reentrant tachycardia induced or has experienced AV node reentrant tachycardia clinically during a follow-up of up to 5 years. The cryosurgical procedure had no detrimental effects on the AH or HV interval or on the paced cycle length at which AV node Wenckebach occurred. Based on these results, this curative operation offers promise for patients with AV node reentrant tachycardia that is refractory to medical treatment. Circulation 76, No. 6, 1329–1336, 1987.

PAROXYSMAL supraventricular tachycardia most commonly arises from reentry within the atrioventricular (AV) node.1–4 Before 1981, the only effective surgical treatment for medically refractory AV node reentrant tachycardia was surgical ablation of the His bundle.5 In 1982, Scheinman et al.6 demonstrated the feasibility of ablating the His bundle by passing an electric shock through a standard His bundle catheter. Although catheter ablation of the His bundle has gained popularity because it precludes the need for an open heart operation, undesirable side effects of the procedure are complete heart block and the necessity for a permanent pacemaker system.

Because ablation of the His bundle replaces one problem (tachycardia) with another (heart block), we developed and tested a surgical technique capable of interrupting the reentrant circuit responsible for AV node reentry tachycardia without blocking normal AV conduction. In 1982, we reported that multiple discrete (3 mm) cryolesions placed around the triangle of Koch were capable of altering the input pathways of the AV node, resulting in permanent prolongation of AV conduction in experimental animals.7 Subsequent studies documented that in the presence of dual AV node conduction pathways, this discrete cryosurgical procedure was capable of selectively ablating only one of the pathways of conduction, thereby leaving normal AV conduction intact while interrupting the anatomic-electrophysiologic substrate responsible for AV node reentrant tachycardia.8–10 After first developing and characterizing the salutary effects of this approach in experimental animals, we applied the procedure in patients with AV node reentrant tachycardia. This communication describes the clinical characteristics of this group of patients, the surgical technique used for the treatment of AV node reentrant tachycardia, and the results of surgery.
Methods

Patient characteristics. Between August 1982 and July 1987, eight patients with documented AV nodal reentrant tachycardia underwent selective cryosurgical therapy for control of their arrhythmias. There were seven women and one man, ranging in age from 16 to 46 years (mean, 30 years). Concomitant cardiac abnormalities included the Wolff-Parkinson-White (WPW) syndrome in three patients (one of whom had a forme fruste of Ebstein’s anomaly) and atrial flutter/fibrillation in one. In seven of the eight patients, including two with concomitant WPW syndrome, AV node reentrant tachycardia was documented to occur spontaneously. In the remaining patient with WPW syndrome, dual AV node pathways were demonstrated and AV node reentrant tachycardia was induced during the preoperative electrophysiologic study. The three patients with WPW syndrome also suffered from orthodromic supraventricular tachycardia. Pertinent clinical characteristics of the patients are summarized in table 1. For each patient, the indication of surgical intervention was failure of medical therapy because of ineffectiveness or intolerable side effects of antiarrhythmic drugs.

Electrophysiologic studies

Preoperative study. Electrophysiologic studies were performed in all patients while they were in the postsurgical, nonseated state after informed written consent was obtained. All antiarrhythmic drugs were discontinued at least 48 hr before the study. Multielectrode catheters were positioned at the high right atrium, in the coronary sinus, across the tricuspid valve in the area of the His bundle, and at the right ventricular apex. Surface electrocardiographic leads I, aVF, and V1 were recorded simultaneously with the intracardiac electrograms (Electronics for Medicine VR-16) and printed on a 16-channel inkjet reader (Siemens mingograph) at paper speeds of 100 to 250 mm/sec. Programmed electrical stimulation was performed with a programmable stimulator (Bloom Associates, Reading, PA).

Standard criteria for the diagnosis of AV node reentrant tachycardia were used and included: (1) demonstration of disparate AV node conduction curves in response to programmed atrial extrastimuli, (2) initiation of supraventricular tachycardia dependent on a critical AH delay, (3) termination of the tachycardia by AV nodal block, (4) normal caudal-to-cephalad retrograde atrial activation pattern during the tachycardia with earliest retrograde atrial activation recorded from the His bundle catheter.11

<table>
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<th>Patient No.</th>
<th>Age (yr)</th>
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<th>Diagnosis</th>
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<td>AVN reentry</td>
<td>8–12/month</td>
<td>7/9/87</td>
</tr>
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</table>

AVN = atrioventricular node; LFW = left free wall; PS = posterior septal.

During the introduction of programmed atrial extrastimuli, the A1A2 coupling interval was consecutively decreased by 10 msec. During the incremental atrial and ventricular pacing studies, pacing was begun at a cycle length slightly faster than sinus rhythm and then decreased by 50 msec decrements until anterograde and retrograde block were observed or until the reproducible initiation of supraventricular tachycardia precluded further evaluation. Dual AV node conduction curves were demonstrated and AV node reentrant tachycardia was initiated in each patient. In the three patients with concomitant WPW syndrome, orthodromic supraventricular tachycardia utilizing the accessory pathway retrograde was also induced. The locations of the accessory pathways in these three patients with left free wall in one, posterior septal in one, and both left free wall and posterior septal in one.

Intraoperative study. Studies were performed with the use of a specially designed operating room cart equipped with our computerized mapping system.12,13 Quadripolar epicardial plaque electrodes were sutured to the right atrium and right ventricle for pacing and recording reference atrial and ventricular electrograms. A bipolar hand-held probe (1.5 mm interelectrode distance) was used to identify the His bundle. During application of cryothermia, atrial pacing was instituted at a constant cycle length that resulted in stable AV conduction. The pacing spike was set to trigger the sweep of a standard storage oscilloscope (Tektronix). This timing dictates that the reference ventricular electrogram will be positioned at the same site on the oscilloscope screen during each cardiac cycle as long as the AV interval remains constant. After completion of cryothermia but before disengagement from cardiopulmonary bypass, incremental atrial and ventricular pacing and programmed atrial stimulation were performed to assess AV conduction and ventricular-atrial conduction, to generate AV node refractory curves, and to attempt to initiate AV node reentrant tachycardia. Intraoperative mapping was not required to guide the surgical approach since the surgical procedure was performed in the same manner in all patients.

Postoperative study. Electrophysiologic studies were performed in all patients 7 to 10 days postoperatively and before hospital discharge. Temporary atrial and ventricular transhilar pacing wires inserted at the time of surgery were used for programmed electrical stimulation and for the recording of atrial and ventricular electrograms. A No. 7F tripolar catheter was inserted for the purpose of recording the His bundle deflection in seven of the eight patients. Patient 6 did not consent to catheter insertion postoperatively. Programmed atrial and ventricular stimulation were repeated and the effects of cryosurgery on AV conduction and on retrograde ventricular-atrial conduction were assessed.

Surgical technique

Patients with AV node reentrant tachycardia only. The heart was exposed through either a median sternotomy or a right anterior thoracotomy in the fourth intercostal space. The latter incision was preferred by four of the female patients for cosmetic reasons. The aorta and both venae cavae were cannulated for cardiopulmonary bypass and epicardial plaque electrodes were sutured to the right atrium and right ventricle. After incremental atrial pacing and induction and termination of AV node reentrant tachycardia, normothermic cardiopulmonary bypass was instituted and a right atriotomy was performed. A hand-held probe was used to confirm that the His bundle was in its normal position at the apex of the triangle of Koch (figure 1). Atrial pacing was then instituted and the AV interval was monitored as described previously (see intraoperative study). A nitrous oxide cryoprobe (Cryomedics, Inc.) with a 3 mm diameter tip was then placed over the tendon of Tadaro at the upper edge of the os of the coronary sinus. Cryothermia was applied at a
temperature of $-60^\circ$ C for 2 min, during which time the AV interval was monitored on a beat-to-beat basis. Three more cryolesions were placed along the tendon of Tadaro, moving sequentially toward the apex of the triangle of Koch near the His bundle (sites 2, 3, and 4 in figure 2). Cryothermia was applied at each site for a period of 2 min or until transient heart block occurred. In our experience, the placement of these first four cryolesions did not result in significant prolongation of the AV interval.

Cryolesions were then placed along the anulus of the tricuspid valve beginning just beneath the os of the coronary sinus (sites 5, 6, 7, and 8 in figure 2). Prolongation of the AV interval usually occurred first during application of cryothermia at sites 7 or 8 (figure 3). It is important to apply cryothermia to each of these sites for the full 2 min if possible, since permanent tissue injury cannot be assured otherwise. Fortunately, as demonstrated in figure 3, the AV interval prolongs in a linear fashion during application of cryothermia, allowing the electrophysiologist to notify the surgeon of the degree of AV interval prolongation with each succeeding beat. As the AV interval prolongs by approximately 200 to 300 msec, complete AV block can be expected to occur within the next few beats. Cryothermia was terminated instantly on the development of complete AV block and the tip of the cryoprobe was irrigated immediately with copious amounts of warm saline. AV conduction invariably resumed within 2 or 3 beats and the AV interval returned to its control value during the ensuing 10 to 15 beats. The cryoprobe was then moved slightly more peripherally until cryothermia could be applied for the full 2 min to a given site without causing heart block. In this manner, the cryoprobe serves as a “reversible knife” and permanent AV block is precluded since the cryothermia would have to be applied for a more protracted interval to result in permanent conduction block.

After placement of cryolesions at sites 1 to 9 (figure 2), thus encircling the AV node, cryolesions were also placed at as many sites within the triangle of Koch as possible without creating permanent AV block, with use of the same end point of temporary block described above. However, having placed the first nine cryolesions, it was usually impossible to apply cryothermia to additional sites within the triangle of Koch for a full 2 min without causing temporary block. In essence, the objective of this operation is to cryoablate as much of the perinodal tissue as possible without causing permanent AV conduction block. This approach is feasible only because of the unique nature of cryosurgery, which allows a definitive end point (complete heart block) to be reached but only on a temporary, reversible basis.

Patients with AV node reentrant tachycardia and the WPW syndrome. A median sternotomy was used for all patients undergoing surgery for the WPW syndrome. Otherwise, the exposure

FIGURE 1. The right atrial septum viewed through a longitudinal right atriotomy. The patient's head is to the left and feet are to the right. The boundaries of the triangle of Koch are the tendon of Tadaro, the tricuspid valve anulus, and a line connecting the two at the level of the os of the coronary sinus. Within the triangle of Koch resides the AV node and proximal portion of the His bundle, which enters the ventricular septum immediately posterior to the membranous portion of the interatrial septum.
and cannulation of the heart for cardiopulmonary bypass were the same as described previously. In patients with AV node reentrant tachycardia and concomitant WPW syndrome, the latter problem must be surgically corrected first before any attempt is made to treat the AV node reentry. Correction of the WPW syndrome initially is essential because the discrete cryosurgical procedure for AV node reentry depends on the ability to monitor exclusive conduction through the AV node–His bundle complex on a beat-to-beat basis. If the patient has a functioning accessory pathway that conducts in the anterograde direction, it is impossible to monitor the effects of cryosurgical modification of normal AV conduction during atrial pacing since the atrial impulse travels preferentially across the accessory pathway to the ventricles. Therefore, in the three patients in the present series with concomitant AV node reentry and the WPW syndrome, the accessory AV connection was first divided under cardioplegic arrest as previously described and after adequate cardiac rewarming, the discrete cryosurgical procedure for AV node reentry was performed.

Two of the three patients with the WPW syndrome had posterior septal accessory pathways and therefore, a right atriotomy had been performed before beginning treatment of the AV node reentry. The supraannular incision above the tricuspid valve at the base of the triangle of Koch that was used to gain access to the posterior septal accessory pathway was closed and the discrete cryosurgical procedure was then performed without regard for the suture line.

Statistical analysis. Data are presented as the mean ± SD. Statistical analysis was performed by Student’s t test for paired data. A p value < .05 was considered to indicate a statistically significant difference.

Results

Application of the discrete cryosurgical procedure resulted in the selective ablation of only one of the two AV node conduction pathways present in each patient and effected a permanent cure of the AV node reentrant tachycardia in all eight patients. There were no operative deaths. Results of the immediate and late postoperative electrophysiologic studies demonstrated smooth AV node conduction curves (figure 4) and no inducible AV node reentrant tachycardia (figure 5). Moreover, all patients maintained normal conduction through the AV node–His bundle complex postoperatively.

None of the patients have experienced a recurrence of AV node reentrant tachycardia during a follow-up interval that has ranged from 1 month to 5 years. In addition, no recurrence of the WPW syndrome has occurred in the three patients who underwent concom-

FIGURE 3. This photograph was taken directly from the screen of the oscilloscope used intraoperatively to monitor AV node conduction during application of the discrete cryolesions. Leads I, aVF, and V,R are displayed along with the electrograms recorded from the right atrium and right ventricle and time lines. The sweep of the oscilloscope is timed to the S-1 stimulus, so that the right ventricular bipolar electrogram appears at the same spot on the oscilloscope on each beat as long as the AV interval is stable. During application of the cryothermia to site 7 and 8, prolongation of the AV interval occurs on each succeeding beat. This prolongation is seen on the oscilloscope as a progressively later occurrence of the QRS complex and of the right ventricular electrogram with each succeeding beat. If complete AV block develops during the application of the cryolesions, the cryothermia is immediately stopped and the area is irrigated with copious amounts of warm saline. Within 3 to 5 beats, AV conduction invariably returns and within 15 to 30 sec, the AV interval gradually decreases to the range of its preoperative value.
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itant correction of that abnormality. Six of the eight patients have not required antiarrhythmic medications postoperatively and none have required drugs for AV node reentrant tachycardia. Postoperatively, patient 1 continued to require antiarrhythmic medications for paroxysmal atrial fibrillation that had been present preoperatively. She underwent electrophysiologic studies 12 days, 5 months, and 2 years postoperatively, none of which demonstrated evidence of dual AV node conduction pathways or AV node reentrant tachycardia. Four years postoperatively, she suffered an acute myocardial infarction complicated by a cardiac arrest from which she was resuscitated successfully. She underwent emergency thrombolytic therapy followed by an unsuccessful coronary angioplasty that resulted in total occlusion of the right coronary artery and a completed myocardial infarction. She was being treated at that time for drug abuse, and 4 months later (December 23, 1986), was found dead of unknown causes. Patient 7 developed temporary atrial fibrillation postoperatively and received short-term treatment (6 weeks) with procainamide and digoxin. The other six patients are alive and well on no medications.

FIGURE 4. Comparison of preoperative (open circles) and postoperative (closed triangles) AV node conduction curves obtained during an atrial paced cycle length (PCL) of 400 msec. Postoperatively, dual AV node conduction pathways were no longer demonstrated.

FIGURE 5. Comparison of the preoperative (left) and postoperative (right) response to programmed atrial extrastimuli. Each panel is organized from top to bottom with electrocardiographic leads I, aVF, V1, and intracardiac recordings from the high right atrium (HRA) or right ventricular apex (RVA) and His bundle region (HBE). During the preoperative study, dual AV node conduction pathways were demonstrated in response to a programmed atrial extrastimulus (S2) introduced at an S1S2 coupling interval of 530 msec (middle). At an S1S2 coupling interval of 440 msec (bottom), critical conduction delay occurred (AH 320 msec) and sustained AV node reentrant tachycardia (SVT) was initiated. At postoperative study, dual AV node conduction pathways were not demonstrated and AV node reentrant tachycardia could not be induced.
Effects of cryosurgery on AV node–His bundle function.

The electrophysiologic effects of the discrete cryosurgical procedure on AV node function are summarized in table 2. There were no significant differences between values obtained preoperatively and those obtained during the late postoperative study for the AH interval (73 ± 11 vs 80 ± 19 msec, p = NS), the paced cycle length at which AV node Wenckebach first occurred (360 ± 32 vs 362 ± 29 msec, p = NS), or the anterograde effective refractory period (312 ± 62 vs 310 ± 32 msec, p = NS). Thus, the cryosurgical procedure had no apparent detrimental effect on AV node conduction, despite the fact that evidence for dual AV node conduction pathways was abolished (figures 4 and 5).

Postoperatively, the electrocardiograms from three patients (Nos. 1, 4, and 8) demonstrated a right bundle branch block pattern. Values for the HV interval obtained preoperatively and postoperatively from each patient were normal (table 2). The occurrence of right bundle branch block may have been due to extension of the cryolesions to the right bundle branch itself or to damage of fibers in the AV node–His bundle region destined to comprise the right bundle branch.

The effects of the discrete cryosurgical procedure on ventricular-atrial conduction are also summarized in table 2. Immediately after completing the application of all cryolesions intraoperatively, ventricular-atrial conduction was absent in all patients. However, complete ventricular-atrial conduction block persisted in only two patients at the time of the late postoperative electrophysiologic study. In the other six patients, the paced cycle length at which retrograde Wenckebach block was first observed ranged from 250 to 350 msec. Thus, permanent block of retrograde conduction through the AV node region was not a prerequisite for successful interruption of the electrophysiologic substrate responsible for AV node reentrant tachycardia.

Discussion

Although the precise anatomic pathways responsible for the AV node reentrant tachycardia have not been delineated, the underlying functional derangement appears to be the presence of two physiologically distinct conduction pathways in or near the AV node.11 Previous studies have attempted to determine if the atrium or lower atrial septum is an essential portion of the reentrant circuit or if the reentrant pathways are confined to the AV node.15–25 Because of the inability to map the AV node region precisely in patients, progress in the development of a satisfactory surgical method for treating patients with AV node reentrant tachycardia has been slow. Thus, the only surgical option in patients initially was to divide or cryoablate the His bundle and insert a permanent pacemaker system.5 However, a fortuitous occurrence in 1979 encouraged us to pursue a more promising course surgically. During the routine dissection of the AV node–His bundle region in a patient with incessant AV node reentrant tachycardia in an attempt to induce permanent heart block, the tachycardia abruptly terminated and could not be reinduced intraoperatively. The dissection was discontinued in the hope that the reentrant circuit had been interrupted inadvertently without creating heart block. On subsequent follow-up, the patient developed no further episodes of

<table>
<thead>
<tr>
<th>TABLE 2</th>
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<td><strong>Effects of cryosurgery on AV node–His bundle function</strong></td>
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<tr>
<td><strong>Preoperative findings</strong></td>
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<tr>
<td>Patient No.</td>
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<td><strong>Postoperative findings</strong></td>
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<td>92</td>
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PCL = paced cycle length; AVWB = atrioventricular Wenckebach; AVN = atrioventricular node; ERP = effective refractory period; VA = ventricular-atrial; Pre-ex = preexcitation; SVT = supraventricular tachycardia.

*HV intervals measured during orthodromic SVT.*
tachycardia and maintained normal AV conduction.26

Although this experience could not be repeated in subsequent patients, it demonstrated the feasibility of developing a surgical procedure specifically designed to interrupt the reentrant circuit responsible for AV node reentrant tachycardia without creating permanent heart block. The major difficulty in developing such a procedure was the frequent and unpredictable occurrence of sudden heart block during surgical dissection in the region of the AV node. Since surgical division of conducting tissues cannot be “retracted,” if the heart block occurred during the dissection of suspected reentrant pathways, the procedure became an immediate failure. It is also noteworthy that in some patients undergoing attempted catheter ablation of the His bundle for AV node reentrant tachycardia, the tachycardia is inadvertently ablated without accomplishing complete heart block.27 Unfortunately, this result of catheter ablation is unpredictable and is therefore not reproducible. Accordingly, we decided that the most logical course would be to use a type of “reversible knife,” i.e., a cryoprobe. This approach would allow the ablation of as much tissue as possible in and around the AV node without creating permanent heart block because at the instant the latter developed, the target tissue could be rewarmed rapidly and conduction would resume.

Once the decision to use cryosurgery had been made, the problem of developing an animal preparation of AV node reentrant tachycardia was addressed. The only known model had been the isolated rabbit atrium preparation described by Mendez and Moe15 and Janse et al.17 Because of the great disparity in size between the rabbit and human atrial septa, we did not use this preparation for fear that the experimental findings could not be extrapolated to the clinical situation. Instead, we used a canine preparation in vivo in which the end points of the cryosurgical procedure were (1) permanent prolongation of the AH interval, (2) ablation of all retrograde (ventricular-to-atrial) conduction through the AV node, and (3) preservation of AV conduction.7-10 These were not arbitrary end points since they represented the three “side effects” noted in the one patient described in which surgical dissection had inadvertently cured AV node reentrant tachycardia.26

During the course of these studies in experimental animals, a second fortuitous development occurred. Unexpectedly, three dogs were found to have dual AV node conduction pathways. Although AV node reentrant tachycardia could not be induced in these animals, the discrete cryosurgical procedure was applied in each of them and one of the two conduction pathways was selectively ablated in each dog.8,9 Thus, results of these experimental studies suggested that the electrophysiologic substrate responsible for AV node reentrant tachycardia could be interrupted permanently by discrete cryosurgery and that this goal could be achieved in a reproducible and safe manner with preservation of normal AV node function.

As a result of these experimental studies, we performed the first such procedure on a human on August 13, 1982. Results in the eight patients studied to date demonstrate that the approach developed ablates AV node reentrant tachycardia and preserves AV conduction. The specific effects of the discrete cryosurgical procedure on anterograde AV conduction in this series of patients differ somewhat with our previous experimental observations. The short-term effects observed experimentally included an increase in the anterograde Wenckebach point and lengthening of the AV nodal effective and functional refractory periods.7-9 With time, these changes in AV node function returned to normal, but prolongation in the AH interval persisted. In patients, values for the AH interval, paced cycle length at which AV node Wenckebach occurred, and effective refractory period obtained postoperatively did not differ significantly from those measured preoperatively.

Experimentally, the electrophysiologic consequences of the discrete cryosurgical procedure on retrograde conduction were analogous to its effects on anterograde conduction. During incremental ventricular pacing, both the ventricular-atrial interval and the paced cycle length at which ventricular-atrial conduction block first occurred increased in the acute postoperative period, whereas 14 weeks later both of these variables had returned to preoperative values.10 These experimental observations are consistent with our clinical findings of initial ventricular-atrial conduction block in all patients, although total ventricular-atrial block persisted postoperatively in only two patients.

Data derived from this study are not sufficient to determine definitively the components of the reentrant circuit or the mechanism by which the discrete cryosurgical procedure prevents AV node reentrant tachycardia. Analysis of the response to programmed stimulation demonstrates that after cryosurgery, existence of slow-pathway conduction was no longer apparent, despite preservation of normal AV conduction. Elucidation of whether this observation reflects direct ablation of the slow pathway or alteration in perinodal tissue that influences the ability to recognize conduction in a slow pathway will require further study. It is of interest that Ross et al.28 began to perform surgical
dissection in the perinodal region for the same purpose in 1983 and have reported similar results. Our findings and those of Ross do suggest that perinodal tissue may be critical for AV node reentrant tachycardia.

Overall, results of this study demonstrate that the discrete cryosurgical procedure developed ablates AV node reentrant tachycardia and preserves AV conduction in a predictable and reproducible manner. Accordingly, this approach offers promise for the treatment of patients with medically refractory AV node reentrant tachycardia. Furthermore, dual AV node conduction pathways have been identified in up to 12% of patients with WPW syndrome. If patients with both WPW syndrome and AV node reentry undergo surgical correction of the former while the latter is ignored, the patients may still experience tachycardia due to AV node reentry postoperatively. For this reason, we now routinely perform the discrete cryosurgical procedure in patients undergoing surgical correction of WPW syndrome if they have dual AV node conduction pathways and spontaneous or inducible AV node reentrant tachycardia. This approach precludes the necessity for antiarrhythmic medications postoperatively for prophylaxis or treatment of AV node reentrant tachycardia.

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