Percutaneous Catheter Modification of the Atrioventricular Node
A Potential Cure for Atrioventricular Nodal Reentrant Tachycardia

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Our purpose was to describe a technique of atrioventricular (AV) node modification for patients with drug refractory AV nodal reentrant tachycardia (AVNRT). Nine patients (mean age, 45±20; range, 14–82) with recurrent drug refractory AVNRT (n=8) or sudden cardiac death thought to be precipitated by AVNRT (n=1) underwent a percutaneous catheter procedure to modify AV nodal function. The area between the electrode recording the maximal His-bundle electrogram and the ostium of the coronary sinus was divided into three zones. Perinodal direct current shocks of 100–300 J were delivered to one (n=2), two (n=3), or three (n=4) zones without complications. The procedure endpoints were modification of AV conduction (either first degree AV block or complete retrograde ventriculo-atrial [VA] block) and failure to induce AVNRT before or after isoproterenol and/or atropine administration. Six of nine patients (67%) have no inducible or spontaneous AVNRT over a mean follow-up of 12.3±4.1 months (range, 4.5–17). One of the six underwent repeat, successful modification, because AVNRT was inducible at restudy 2 days after the initial procedure. AVNRT recurred in three patients (33%), one early (3 days) and two late (3–4 months). Two of these patients underwent complete ablation of the AV junction and permanent pacemaker placement, whereas one is controlled with drug therapy. Therefore, AV nodal modification resulted in tachycardia control without antiarrhythmic drugs in six of nine (67%) and obviated the need for complete AV junctional ablation in seven of nine patients (78%). Elimination of AVNRT appears to result from either block in the retrograde fast pathway or modification of the antegrade slow pathway, such that AVNRT cannot be sustained. Additional findings suggest that an atrio-Hisian accessory connection may not be involved in AVNRT in some of these patients. Percutaneous catheter AV nodal modification appears to be a promising technique for treatment of refractory AVNRT and may obviate need for complete AV junctional ablation in a substantial number of patients with drug/pacemaker refractory AVNRT. (Circulation 1989;80:757–768)

Atrioventricular nodal reentrant tachycardia (AVNRT) is a very common cause of paroxysmal supraventricular tachycardia1–6 and may be associated with disabling symptoms refractory to medical therapy. Patients may require total disruption of atrioventricular (AV) conduction to achieve tachycardia control. In the most recent report from the worldwide Percutaneous Catheter Mapping and Ablation Registry, approximately 20% of patients with drug refractory supraventricular tachycardia underwent attempted catheter ablation of the AV junction because of AVNRT.7 The obvious disadvantage to this approach is that tachycardia control is achieved at the expense of AV conduction and results in life-long dependency on cardiac pacing.

More recently, surgical techniques have been introduced to control tachycardia in patients with AVNRT but with preservation of AV conduction. Cox et al8,9 demonstrated that cryolesions made around the AV node in canines prevented echo beats but still allowed for normal AV conduction. In recent clinical reports,10–12 both cryodestruction and surgical dissection around the AV node were effective in abolishing AVNRT without producing AV block. These procedures, nevertheless, require

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open heart surgery with its attendant risk and expense.

Prior canine work in our laboratory showed it possible to deliver large direct current (DC) shocks in the region of the AV node without causing complete AV block. Dogs with up to one half of their AV nodes destroyed still maintained normal AV conduction. We, therefore, attempted to combine this experience with dogs and the surgical and catheter experience with humans to devise a procedure of creating lesions around the AV node using a percutaneous approach.

In this report, we explore the use of a catheter technique for the delivery of perinodal DC shocks to modify AV nodal function, such that AVNRT is abolished but AV conduction is preserved.

Methods

Clinical Data

Nine patients (mean age, 45±20 years; range, 14–82 years) with recurrent drug refractory AVNRT, or sudden cardiac death thought to be precipitated by AVNRT, underwent a percutaneous catheter procedure to modify AV nodal function. Clinical data on these eight women and one man are summarized in Table 1.

Eight patients had recurrent symptomatic tachycardia for a mean of 10.6±7.3 years (range, 1–20 years). In two (3 and 7), AVNRT was virtually incessant. These patients were refractory to or intolerant of multiple drugs (mean, 6.5±2.1 drugs; range, 3–10 drugs). Antitachycardia pacemakers failed to improve symptoms in two patients (3 and 7). One patient (8) had sudden cardiac death with normal coronary arteries and left ventricular function. Neither ventricular tachycardia nor ventricular fibrillation could be induced during electrophysiologic testing, but AVNRT at a rapid rate was easily induced.

Baseline Electrophysiologic Study

Before attempted catheter modification, each patient underwent invasive cardiac electrophysiologic studies to determine arrhythmia mechanism. These studies were preformed in the nonsedated, postabsorptive state after cardiac medications were discontinued for at least 72 hours.

Written informed consent was obtained from all patients. Using standard 6F quadrapolar electrode catheters, electrograms were recorded from the right atrium, His bundle, right ventricle, and coronary sinus in all patients.

A standard stimulation protocol including atrial and ventricular incremental pacing and extrastimulation was performed. Intravenous isoproterenol, or atropine, or a combination was given if supraventricular tachycardia could not be induced at baseline. Isoproterenol was given as a continuous intravenous infusion, beginning with 1 µg/min and titrated to achieve an increase in heart rate of 25–50% above baseline. Atropine was given in 0.5-mg intravenous increments to achieve the same heart rate parameters. Although there is no finding diagnostic of AVNRT, the following criteria were sought to rule out other mechanisms of SVT and support the presence of AVNRT; in all cases tachycardia induction was dependent on achievement of a critical atrio-His (AH) interval, atrial activation occurred before or simultaneous with ventricular activation, and premature ventricular stimuli introduced during inscription of the His-bundle deflection did not advance the subsequent atrial electrogram. Other findings suggestive of AVNRT were the presence of dual AV nodal pathways (discontinuous A1A2 and H1H2 curves) (five patients) and tachycardia termination with a ventricular complex that did not result in atrial activation (two patients).

Atrioventricular Nodal Modification Procedure

After obtaining informed written consent, the patients were taken to the catheterization laboratory. Two standard 6F Quadrupolar electrode catheters (USCI) were introduced into the right femoral vein; one was positioned across the tricuspid valve to record the maximal His-bundle potential and the other against the right ventricular apex. A 6F ele-
trode catheter with lumen (Gorlin) was introduced into either the subclavian or internal jugular vein and placed into the coronary sinus. Radiographic contrast was injected into this catheter to locate the ostium of the coronary sinus. The location of the ostium was marked on the fluoroscopic screen for future reference (anteroposterior projection). Arterial pressure was monitored through a short interarterial catheter throughout the procedure. A third 6F Quadrupolar electrode catheter (USCI) was inserted via a femoral vein and was used for mapping and for delivery of DC shocks.

Anatomic studies have shown that the AV node lies within the Triangle of Koch, which comprises the area of the right atrium bounded by the tendon of Todaro, the septal attachment of the tricuspid valve, and the orifice of the coronary sinus. The apex of Koch’s triangle is the central fibrous body. The electrode recording the His-bundle potential was used to approximate the location of the central fibrous body. The area above a line drawn between the ostium of the coronary sinus and the His-bundle recording site was divided into three zones by drawing perpendicular lines to the line connecting these landmarks on the fluoroscopic screen (inset).

The purpose of zoning was to test the hypothesis that delivery of shocks to the site of earliest retrograde atrial activation during tachycardia would result in tachycardia control. In addition, it was thought that this technique would best guide application of perinodal shocks.

Because during AVNRT, atrial and ventricular electrograms occur almost simultaneously, mapping to accurately determine the site of earliest atrial excitation was achieved by introducing single programmed ventricular extrastimuli during tachycardia at 10-msec decrements. In all patients, ventricular premature complexes could be dissociated from the tachycardia, allowing for accurate mapping of the perinodal region (Figure 2).

Catheter position was determined both by use of biplane fluoroscopy (anteroposterior and right anterior oblique projections) and unipolar electrogram characteristics recorded from the mapping catheter. Three electrogram characteristics were required before delivery of the DC shocks (Figure 3): 1) an atrial injury current of >0.5 mV to ensure good tissue contact, 2) nearly equal amplitudes of the atrial and ventricular electrograms (Ruder et al have shown that this finding reliably locates the region above the anulus and, therefore, obviates the possibility of shock delivery to the ventricle.), and 3) absence of a His-bundle deflection. In this technique, we assiduously avoided delivery of shocks to any area in which a His-bundle deflection was inscribed. This is totally different from our previously described protocol for complete AV junctional ablation, in which the largest unipolar His bundle deflection is used to determine the shock site.

Shocks were initially delivered to or near the site of earliest recorded perinodal atrial activation. In one patient (7), the initial shock was inadvertently delivered to zone 2, although the site of earliest retrograde atrial activation was posterior in zone 3. Patients were reevaluated after each shock. If procedure endpoints (see below) were not achieved, additional shocks were delivered to the other zones. If endpoints were achieved in any zone transiently, additional shocks of the same or higher energy were delivered to that zone. The sequence of shocks depended, in part, on our ability to reproducibly achieve the atrial electrogram characteristics desired in a given zone.

Patients were anesthetized with a short-acting intravenous barbiturate, and shocks were synchronized to the R wave on the surface ECG. DC shocks were delivered via a standard defibrillator (Life Pak 6, Redmond, Washington) between the distal electrode of the mapping catheter serving as the cathode and a large-diameter skin electrode (R2 Corp, Skokie, Illinois) on the sternum serving as the anode. In the first two patients, shocks to zone 3 were delivered between the two electrodes of the coronary sinus catheter outside the coronary sinus ostium (cathode) and the skin electrode (anode). Patients received 1–7 shocks of 100–300 J to one (n=2), two (n=3), or three (n=4) areas (Table 2). Procedure endpoints were modification of AV conduction (either first degree AV block or complete retrograde ventriculo-atrial [VA] block) and

**Figure 1.** Schematic representation of the heart with the right atrium opened. Catheters are positioned in the coronary sinus (A), and across the tricuspid valve (B), to record the His-bundle electrogram. The stippled region is a schematic representation of the atrioventricular junction. The area between the coronary sinus ostium and the electrode recording the largest His-bundle deflection was divided into three zones (1, 2, 3) by drawing perpendicular lines to the line connecting these landmarks on the fluoroscopic screen (inset).
failure to induce AVNRT before or during isoproterenol infusion and/or atropine administration. Continuous electrocardiographic monitoring was performed in all patients for 24–48 hours after the procedure. Creatinine phosphokinase levels were determined at 6 hours and daily, thereafter. All patients had an echocardiogram within 3 days following the procedure.

Follow-up

Electrophysiologic studies were repeated 2–4 days and 2–4 months following the procedure when appropriate. Repeat studies were not deemed appropriate in patient 4 who had undergone complete AV junctional ablation, patient 8 who had an early spontaneous recurrence, or patient 7 who was in complete and then second-degree AV block. Anterograde and retrograde conduction were evaluated and inducibility of AVNRT was reassessed. Patients underwent ambulatory electrocardiographic monitoring and received transtelephonic event monitors to evaluate the recurrence of spontaneous arrhythmias.

Results

A summary of the baseline electrophysiologic studies is found in Table 3. It was difficult to evaluate anterograde or retrograde conduction characteristics in some patients because atrial stimulation promptly initiated AVNRT (patients 3, 7, and 9). Dual AV nodal physiology was demonstrated in five of nine patients. AVNRT was of the common variety (anterograde conduction over a slow pathway and retrograde conduction over a faster conduction pathway) in all patients. The mean cycle length of AVNRT in these patients was 290±59 msec (range, 240–380 msec). The site of earliest retrograde atrial activation during AVNRT was localized to the anterior low right atrial septum (zone 1–2) in eight patients and the posterior low right atrial septum (zone 3) in one patient (7).

Findings Immediately Following the Procedure

Immediately after the procedure, AVNRT was not inducible in any patient. Seven patients had first degree AV block, one patient (7) had complete AV block, and one patient (8) had transient complete AV block after DC shock, but ultimately regained normal anterograde and retrograde conduction within 15 minutes. Three patients had retrograde VA block (3, 5, and 9). Peak creatinine phosphokinase (IU/dl) was 438±493 and peak MB fraction was 42±39.
Echocardiography of all patients performed 1–3 days after the procedure showed no pericardial effusion or new valvular or wall-motion abnormalities.

**Findings at Early Restudy**

Repeat electrophysiologic studies of seven patients were performed after 2–4 days. Of the patients restudied, AVNRT was not inducible in six of the seven patients before and after isoproterenol and/or atropine administration. In one patient (9), although the AH required to induce AVNRT was increased from 230 to 310 msec, the induced tachycardia was associated with symptoms, and this patient underwent a repeat modification procedure. Patient 8, who showed normal AV conduction immediately after the procedure, had spontaneous AVNRT 2 days later and was not restudied. She was treated with flecainide and subsequently was found to be noninducible on drug therapy. One patient developed complete AV block associated with a narrow complex escape at a rate of 70 beats/min and was not restudied. A summary of the results of these studies is found in Table 3.

First-degree AV block was still present in four of seven patients (2–5), and retrograde VA block remained in two of three patients (3 and 5). AV nodal reentrant echo beats were induced in three of seven patients (2, 6, and 9). Dual AV nodal physiology was demonstrated in two (6 and 9) of the five patients who showed this before the procedure.

**Findings at Late Restudy**

At 2–4 months, restudy was performed of six patients, including the patient (9) who underwent a repeat modification procedure. The results of the six late restudies are found in Table 3. Five patients were not inducible before or after isoproterenol and/or atropine administration. One patient (1) had inducible AVNRT and atrial tachycardia. First-degree AV block persisted in three patients (3, 5, and 9), and retrograde VA block persisted in two patients (3 and 5).

Of the other three patients not restudied, two (4 and 8) had spontaneous AVNRT and one (7) had type 1 second-degree AV block with spontaneous isolated atrial echoes for 3 months but over the past 9 months has had stable first-degree AV block.

**Mechanism of Tachycardia Control**

The mechanism for tachycardia control varied in the six patients who underwent repeat electrophysiologic study at 2–4 months. In three (3, 5, and 9), AVNRT could not be induced because of retro-

![Figure 3. Unipolar unfiltered electrogram from the mapping (probe) electrode shows a large atrial potential, an atrial injury potential, and no His-bundle deflection. The standard represents 1 mV. HBE, His-bundle electrogram; CS, coronary sinus.](http://circ.ahajournals.org/lookup/doi/10.1161/01.CIR.761.09.816A)

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**Table 3.** Results of Repeat Electrophysiologic Studies

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grade VA block in the fast pathway. In these patients, despite achieving AH intervals equal to or in excess of those achieved in the baseline study (which produced AVNRT), no atrial echoes could be induced. In three patients (1, 2, and 6), tachycardia control was achieved by attenuation of anterograde conduction. In these patients, although echo beats could be readily induced, sustained tachycardia could not be maintained because of anterograde block in the slow pathway. In two patients (1 and 6) with persistent dual AV nodal physiology after modification, the AV nodal refractory curves were shifted upward and to the right (Figure 4), suggesting modification of both fast and slow pathway anterograde refractory.

Observations Relating to Mechanisms of Atrioventricular Nodal Reentrant Tachycardia

In the course of these studies, several observations were made that may have implications relevant to the mechanism of AVNRT. In one patient (6), late restudy showed that critical AH intervals, identical to those observed in the baseline study, initiated a series of echo complexes characterized by inscription of the low septal before the high right atrial electrogram and gradual advancement of the atrial electrogram to precede the His-bundle deflection (Figure 5). The most likely explanation for this phenomenon is antegrade Wenckebach conduction over the slow pathway with relatively constant retrograde conduction over the fast pathway. The

<table>
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<tr>
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*All patients had no inducible atrioventricular nodal reentrant tachycardia at the end of the procedure. †Retrograde ventriculo-atrial block (findings within 5 minutes of shock); ‡third-degree atrioventricular block; §1 *AVB persisted for 15 minutes.
Table 3. Electrophysiologic Findings

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Early (2–4 days)

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BCL, Blocked cycle length; AVN, atrioventricular node; VA, ventriculo-atrial; AVNR, atrioventricular nodal reentrant; a, atrioventricular nodal reentrant tachycardia induced; b, inducible atrial tachycardia.

*Findings on surface ECG.
†After repeat modification.

in the reentrant circuit. Low right atrial reentry with AV Wenckebach conduction is an alternative but less likely explanation for these findings, because these echoes occurred only after a prolonged AH interval similar to that which produced AVNRT at baseline. In one patient (9), in whom a perinodal (zone 1) shock of 100 J resulted in right bundle branch block and transient prolongation of the HV interval from 40 to 60 msec, AVNRT was still inducible with the identical tachycardia cycle length and His-to-atrial-echo (HAe) interval as compared with the baseline study (Figure 6). In addition, retrograde HA intervals following ventricular extrastimulation prolonged from 30 to 45 msec. Because the perinodal shock probably resulted in damage to the most proximal portion of the His bundle, HAe prolongation would have been anticipated if the His bundle itself was a critical component of the tachycardia circuit. However, this does not exclude possible involvement of an alternative, very proximal, His

Figure 4. Graph depicting the relation between progressively premature atrial extrastimuli (A1-A2) and corresponding atrioventricular nodal response time (A2-H2) before (PRE) and after (POST) modification for patient 6 at a basic drive cycle length of 600 msec. Note the upward and rightward shift denoting prolongation of refractory for both fast and slow pathways.
connection. Prolongation of the HA interval in response to ventricular extrastimuli, compared with a constant short HAe during tachycardia, likewise supports the presence of an intranodal circuit. In one patient, retrograde VA conduction block was present after delivery of perinodal shocks, but after atropine, both VA conduction and isolated echo beats could be induced. These observations also suggest, but do not prove, that the retrograde limb of the tachycardia circuit involves nodal tissue.

Long-term Follow-up

After a mean follow-up interval of 12.3±4.1 months, other atrial arrhythmias have been documented in four patients (Table 4). Three patients (1, 3, and 9) developed transient atrial tachycardia. In one (3), atrial tachycardia was initially controlled with atenolol therapy, but resolved spontaneously within 6 months, and she has been arrhythmia free without antiarrhythmic therapy for 6 months. Another patient (9) was treated with flecainide to control atrial tachycardia, but the arrhythmia resolved within 1 month and the patient has remained arrhythmia free without drugs. One patient (2) had documented episodes of paroxysmal atrial fibrillation with moderate ventricular response and is well controlled on digoxin therapy. She reports only mild palpitations occurring once every 1 to 2 months, whereas one patient (8) with recurrent AVNRT is well controlled with flecainide (no recurrences). The procedure failed in two patients (1 and 4), and both underwent complete AV junction ablation with insertion of a permanent pacemaker. One of these patients (1) showed new atrial arrhythmias. No patient has shown spontaneous progression to higher degrees of AV block during follow-up, and no patient has had syncope or presyncope after the procedure.

The five patients (3, 5–7, and 9) receiving no antiarrhythmic therapy and one patient (8) treated with flecainide have had a marked improvement in symptoms, reporting only a rare skipped beat, but no

FIGURE 5. Premature atrial extrastimulus (S2) induced 240 msec after a basic drive (S1) shows initiation of two atrial echo complexes with low septal right atrial electrogram (HBE) preceding that from the high right atrium (HRA) and atrial deflection simultaneous with and preceding the His-bundle deflection (H). Although the His-bundle deflection of the two echo complexes is somewhat obscured by the atrial electrogram, the same HV relation is preserved (40 msec) as seen in the first paced beat and the normal sinus beat at the end of the recording. Atrial electrogram of the echo beats clearly advances in front of the His and ventricular electrograms. Ladder diagram illustrates the presumed mechanism involving anterograde Wenckebach conduction over the slow pathway with constant retrograde conduction over the fast pathway.
Palpitations. One patient receiving antiarrhythmic therapy (2) reports occasional palpitations (atrial fibrillation) that do not interfere with her daily life. Two patients (1 and 4) experienced recurrent AVNRT and underwent complete AV junctional ablation. One of these patients (1) desired complete AV junctional ablation rather than attempted repeat modification or surgery, whereas complete AV junctional ablation was recommended for the other patient with hypertrophic cardiomyopathy (4) because of severe symptoms (syncope, severe hypotension) associated with her arrhythmia and the increased risk of cardiac surgery. Both patients underwent cardiac pacemaker insertion and are free of symptoms.

**Figure 6.** Panel A (baseline): Shows a sinus complex with an infranodal conduction time (HV) equal to 40 msec. Panel B: Baseline recording during atrioventricular nodal reentrant tachycardia with HAe (His-to-atrial echo) interval of 30 msec. After delivery of a perinodal shock (C), HV prolongs to 60 msec, but during atrioventricular nodal reentrant tachycardia (D), the HAe remains 30 msec. Premature ventricular extrastimuli (S2) (E) shows prolongation of the HA interval from 30 to 45 msec. HBE, His-bundle electrogram; CS, coronary sinus.

**Procedure Variations and Success**

Patients received a mean of 3.4 shocks each, with a cumulative delivered energy of 500±364 J. The zone in each patient in which shocks produced noninducibility of AVNRT were zone 1 (n=2), zone 2 (n=2), and zone 3 (n=5). A comparison of the initial procedure in patients with (n=4) and without (n=5) recurrent or inducible AVNRT shows that the number of shocks (3.3 vs. 3.6), cumulative delivered energy (450±265 vs. 540±456 J), and location of successful shock (zone 1: n=1; 2, n=2; 3, n=2 vs. zone 1: n=1; 2, n=2; 3, n=3) appear similar in both groups. Likewise, the locations of shocks in the four patients developing new atrial

**Table 4. Follow-up**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Symptomatic improvement</th>
<th>Spontaneous arrhythmias</th>
<th>Inducible arrhythmia</th>
<th>Therapy</th>
<th>Follow-up (mo)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>NO</td>
<td>AVNRT</td>
<td>AVNRT</td>
<td>His ablation</td>
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</tr>
<tr>
<td>2</td>
<td>YES</td>
<td>A-Fib</td>
<td>NO</td>
<td>Digoxin</td>
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</tr>
<tr>
<td>3</td>
<td>YES</td>
<td>Atrial tach*</td>
<td>NO</td>
<td>None</td>
<td>14.5</td>
</tr>
<tr>
<td>4</td>
<td>NO</td>
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<td>AVNRT</td>
<td>His ablation</td>
<td>13</td>
</tr>
<tr>
<td>5</td>
<td>YES</td>
<td>NO</td>
<td>NO</td>
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<td>NA</td>
<td>None</td>
<td>14</td>
</tr>
<tr>
<td>8</td>
<td>YES</td>
<td>AVNRT†</td>
<td>NO†</td>
<td>Flecainide</td>
<td>7</td>
</tr>
<tr>
<td>9</td>
<td>YES</td>
<td>Atrial tach*</td>
<td>NA</td>
<td>None</td>
<td>6.5</td>
</tr>
</tbody>
</table>

Atrial tach, Atrial tachycardia; A-fib, atrial fibrillation; NA, not available.

*Transient atrial tachycardia, patients now arrhythmia free.
†No spontaneous or inducible arrhythmia with flecainide.
arrhythmias did not differ significantly from those in the patients who did not develop new arrhythmias (Table 2). The mean number of shocks and cumulative energy were 3.75 and 600±510 J, compared with 3.2 and 420±228 J in the patients with and without atrial arrhythmias. Although there is a trend suggesting that the patients developing atrial arrhythmias received more shocks and higher energy, these differences were not statistically significant. As stated earlier, an attempt was made to use the location of retrograde atrial breakthrough to direct the perinodal shocks. Two patients had success with single shocks (3 and 4). In three patients, shocks were predominantly anterior (6 and 9) or posterior (7), although more than one shock was delivered to more than one zone. In four patients, multiple shocks were delivered to all three zones.

**Discussion**

Eight patients with recurrent symptomatic AVNRT refractory to drug and/or device therapy (two patients) and one patient with sudden cardiac death thought to be precipitated by AVNRT underwent catheter AV nodal modification. The safety and efficacy of percutaneous catheter ablation of the AV node junction and accessory pathways has been reported. The goal of AV nodal modification was to achieve the same degree of tachycardia control without interrupting AV conduction.

**Efficacy**

No spontaneous or inducible AVNRT has occurred in six of the nine patients (67%) during a follow-up of 12.3±4.1 months. These patients all had disabling symptomatic AVNRT and had failed a mean of 6.4±2.5 drugs (range, 3–10 drugs) and/or antiarrhythmic pacing (3 and 7). AVNRT recurred or was induced after the modification procedure in three patients (33%). One of these patients (8) has achieved tachycardia control with flecainide, whereas ablation of the AV junction with pacemaker placement was required in two patients (1 and 4). Therefore, AV nodal modification obviated the need for AV junctional ablation with permanent pacemaker implantation in seven of nine patients (78%).

**Predictors of Outcome**

Due to the small number of patients, no finding clearly predicted procedure success. The number of shocks, cumulative delivered energy, and zone of shock were similar in both groups (patients with and without recurrent or inducible AVNRT). Attempting to direct initial shock location by mapping did not appear to improve success, although five patients required predominantly anterior (n=4) or posterior (n=1) shocks to achieve procedure end points. The data suggest that, in some patients, success may be due to nonspecific cumulative damage to the AV node rather than site specificity. The presence or absence of anterograde conduction modification or retrograde VA block immediately after the procedure was not always predictive of success. It is noteworthy that there were no recurrences in the two patients with persistent retrograde VA block. The presence or absence of AV nodal reentrant echoes at early restudy did not predict outcome.

**Mechanisms of Tachycardia Control and Atrioventricular Nodal Reentrant Tachycardia**

The mechanism of tachycardia control varied. Three patients had retrograde block in the fast pathway, and three patients had attenuation of anterograde conduction over the slow pathway. The precise anatomic location of these pathways is not clear. Functional, as well as anatomic data have been interpreted as being consistent with the presence of specialized His-atrial fibers, which serve as the retrograde pathway. This hypothesis serves as one rationale for recently introduced surgical procedures purportedly designed to destroy the atrial link in the tachycardia circuit. Repeated attempts to induce tachycardia after the delivery of perinodal shocks allowed for observations suggesting the His-bundle may not be a critical part of the tachycardia circuit. In one patient, for example, marked prolongation of the HV interval after DC shock was not associated with prolongation of the HAt during tachycardia. In another, inscription of otherwise classical AV nodal reentrant echo beats preceded the His-bundle deflection. An alternative explanation is that the tachycardia circuit is entirely intranodal and that various surgical or catheter ablative techniques disrupt or modify intranodal conduction. Our results are compatible with damage to both anterograde and retrograde conduction.

**Adverse Effects**

In four patients, supraventricular arrhythmias other than AVNRT were documented for the first time after ablation. In patient 2, paroxysms of atrial fibrillation were associated with only moderate ventricular rates and were readily controlled with digoxin therapy. Two patients developed transient atrial tachycardia that required a brief course of antiarrhythmic therapy. One patient developed complete AV block, which gradually resolved over a 4-month follow-up interval. Permanent pacing was not required for any patient. Although our results are encouraging, they must be considered as only preliminary, because the long-term effects of this procedure on AV conduction and late atrial arrhythmias are not known.

**Other Reports of Atrioventricular Nodal Modification**

In contrast to our technique of delivering multiple perinodal shocks, Haissaguerre et al delivered DC shocks 5–10 mm from the site of maximal His-bundle recording. Shocks were delivered if the His-bundle deflection was ≤0.1 mV and differed from our technique, which avoided delivery of
shocks to areas from which any His-bundle recording was evident. In addition, they required attainment of complete retrograde VA block as the procedure end point. The efficacy and side effects reported were similar to ours except that two of their patients developed complete AV block (10%), whereas none in our report had permanent complete block. This difference may be due to either their delivery of shocks to areas recording small His-bundle deflections or the greater amounts of individual or cumulative energy delivered.

**Summary**

We have described a catheter technique for modification of AV nodal function by administering DC perinodal shocks in patients with disabling or life-threatening AVNRT. We found that, in the majority of such patients, arrhythmia control was possible without resorting to total ablation of the conduction system. The only adverse effects were initiation of new supraventricular arrhythmias (usually short lived and responsive to standard antiarrhythmic drug therapy) and the development of transient complete AV block in one patient. This procedure should be reserved for patients with AVNRT who are resistant to conventional therapy and considered suitable candidates for total AV junctional ablation.

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