Modification of Human Atrioventricular Nodal Function by Selective Atrioventricular Nodal Artery Catheterization

Paul J. Wang, MD, Guillermo Sosa-Suarez, MD, and Peter L. Friedman, MD, PhD

The hypothesis that human atrioventricular (AV) nodal function can be modulated selectively with a new technique of AV nodal artery catheterization was tested in eight subjects referred for diagnostic cardiac catheterization or electrophysiological studies. Three patients had no history of arrhythmias. Three patients had supraventricular tachycardia (SVT) due to reentry confined to the AV node (AVNRT). One patient had SVT due to reentry over a concealed AV bypass tract (AVRT-CBT), and one patient had nonsustained ventricular tachycardia. In each subject, sinus cycle length, AH interval, HV interval, AV nodal effective refractory period (AVN-ERP), and Wenckebach paced cycle length were measured in a control state. A flexible infusion catheter was then positioned selectively in the AV nodal artery of each subject. Through this catheter, a constant infusion of 0.1 mg/min procainamide at a flow rate of 0.125 ml/min (n=1) or 50 μg/min acetylcholine at a flow rate of 0.25 ml/min (n=4) was administered. Electrophysiological parameters were determined again during selective AV nodal artery drug infusion and during infusion of saline at identical rates. Two subjects developed transient AV nodal block during selective AV nodal catheterization alone and did not receive an infusion of drug or saline. A stable position of the AV nodal artery catheter could not be achieved in one other subject, who also received no drug or saline. In the other five subjects, drug infusion caused an increase in AVN-ERP from a control value of 312±52 msec to a value of 543±228 msec (p<0.05) and an increase in Wenckebach paced cycle length from a control value of 360±47 msec to a value of 572±217 msec (p<0.05). These parameters were unchanged from control during selective saline infusion. In two patients with AVNRT, drug infusion abolished SVT by causing complete blockade of ventriculoatrial conduction as well as lengthening of anterograde AVN-ERP. In the patient with AVRT-CBT, drug infusion abolished SVT by preventing repetitive anterograde AV conduction. Saline had no effect on SVT inducibility. Selective AV nodal artery catheterization enables AV nodal function to be modulated exclusively. Delivery of ablative agents to the AV node by this technique may be useful in patients with refractory SVT. (Circulation 1990;82:817–829)

Catheter ablation of atrioventricular (AV) conduction has emerged as a useful therapeutic alternative to surgery in the management of patients with a variety of drug-resistant supraventricular tachycardias. Delivery of high-energy DC shocks through an electrode catheter positioned against the AV junction, the technique usually used to ablate AV conduction, has several limitations. In particular, local complications result from barotrauma to the heart, and production of complete AV block occurs in a high but unpredictable proportion of patients undergoing the procedure, necessitating prophylactic insertion of a permanent pacemaker. Inoue et al have advanced the hypothesis that the coronary arterial circulation might be used to deliver ablative agents to sites of origin of cardiac arrhythmias regardless of their location in the heart. The validity of this approach in selected patients with ventricular tachycardia has been established by the demonstration that the arrhythmia can be temporarily abolished by infusion of iced saline or antiarrhythmic drugs and permanently ablated by infusion of ethanol selectively into the coronary arterial branch supplying the site of origin of the tachycardia. However, the feasibility of this approach for permanently altering AV nodal function in patients with refractory supraventricular
tachycardia has not yet been evaluated. As a first step in this evaluation, the present study was designed to test the hypothesis that AV nodal function can be modified exclusively by local infusion of antiarrhythmic drugs with a new technique of selective AV nodal artery catheterization.

Methods

Between June 1988 and October 1989, 34 patients referred to the Brigham and Women’s Hospital for diagnostic cardiac catheterization or electrophysiological studies were asked to participate in a study of the feasibility of selective catheterization of the AV nodal artery according to a protocol approved by the hospital’s Committee for the Protection of Human Subjects From Research Risks. These 34 individuals were approached because they had normal left ventricular systolic function and were considered likely to have normal coronary arteries; both criteria were required for entry into the study. After reading the consent form approved by the hospital’s human subjects research committee that stated participation in the study could add an additional 90 minutes to the duration of the catheterization, 18 of the 34 patients declined to participate in the study. Of the remaining 16 patients who agreed to participate, four were found to have significant coronary artery disease at the time of cardiac catheterization, thus excluding them from the study. One patient who initially agreed to participate changed his mind at the conclusion of the diagnostic portion of his cardiac catheterization and decided not to participate in the research portion. In one patient, stable positioning of an angioplasty guiding catheter in the right coronary artery could not be achieved, precluding safe catheterization of the AV nodal artery. One patient developed a vagal reaction during the final portions of her diagnostic catheterization and required treatment with intravenous atropine, thereby excluding her from participation. In another patient, spasm of the ostium of the right coronary artery occurred soon after an attempt at selective AV nodal artery catheterization had begun (see below). Intracoronary nitroglycerin was administered, and after removal of the angioplasty guiding catheter, the spasm resolved. However, recatheterization of the right coronary artery was deemed too hazardous to allow completion of the research portion of the study. Finally, in one patient with severe aortic regurgitation, the force of ventricular systole was so great that it rendered precise control of movement of the guide wire in the distal right coronary artery impossible (see below). Because of concern that excessive guide wire motion might cause dissection of the artery, attempts at selective catheterization of the AV nodal artery in this patient were abandoned.

Selective catheterization of the AV nodal artery was accomplished successfully in eight patients and was always accomplished within the 90 minutes allotted by the human subjects committee (minimum study time, 45 minutes; maximum study time, 80 minutes). These eight individuals constitute the study population of the present report. Three of the eight patients (patients 1, 2, and 6; Table 1) had been referred for diagnostic cardiac catheterization, whereas five individuals (patients 3–5, 7, and 8; Table 1) had been referred for electrophysiological studies because of recurrent supraventricular or ventricular arrhythmias. Patients were studied in a fasted state while lightly sedated with diazepam as needed to allay anxiety. All antiarrhythmic drugs were withheld for at least five half-lives of elimination before study in each patient, with the exceptions of patient 1, who had taken diltiazem and propranolol; patient 2, who had taken digoxin and propranolol; and patient 6, who had also taken diltiazem the day before the study.

Electrophysiological Studies

After local anesthesia with 2% lidocaine, venous sheaths were placed into both femoral veins, and an 8F sheath with side-arm was introduced into the right femoral artery. Electrode catheters were then advanced under fluoroscopic guidance and positioned in the high right atrium, coronary sinus (patients 3–5 and 7), and right ventricular apex and across the tricuspid anulus, the latter position being used to record a His bundle electrogram. Once the sheaths and catheters were in place, 5,000 units i.v. heparin was administered. Intracardiac bipolar electrograms, filtered between 30 and 500 Hz, were displayed with three surface electrocardiographic
leads on an Electronics for Medicine VR16 oscillographic recorder (Honeywell Inc.) and recorded continuously on a direct-writing ink-jet recorder (Siemens-Elema Mingograf) at paper speeds of 100 mm/sec.

Programmed electrical stimulation of the atria and ventricles was performed with previously described methods.\(^5\) Control values for atrial, AV nodal, and ventricular effective refractory periods as well as measurements of the AV nodal Wenckebach paced cycle lengths and basic conduction intervals during sinus rhythm were determined in each patient. In patients 3–5 and 7 (who had been referred because of recurrent supraventricular tachycardia), the patients' clinical arrhythmias were induced, allowing delineation of the mechanism of tachycardia. Once sustained supraventricular tachycardia had been induced, it was terminated by programmed stimulation. In each of these four patients, the stimulation sequence that had initiated arrhythmia was repeated at least twice to confirm that induction of the tachycardia was reproducible.

**AV Nodal Artery Catheterization**

After determination of electrophysiological parameters and delineation of the mechanism of induced supraventricular tachycardia in the control state, the dominance of the coronary arterial circulation was established in each patient by inspection of diagnostic angiograms of the right and left coronary arteries. Six individuals (patients 1–4, 6, and 8) had a right-dominant circulation in which the AV nodal artery could be identified as a superiorly directed branch arising from the posterior descending artery at its point of origin from the right coronary artery (Figure 1, top panel). In patients 5 and 7, the coronary circulation was left dominant—the AV nodal artery arising from the posterior descending branch at its point of origin from the left circumflex coronary artery (Figure 2, top panel). An 8F left or right Judkins coronary angioplasty guiding catheter was introduced through the femoral arterial sheath and positioned in the ostium of the appropriate coronary artery. Then, an additional 5,000 units i.v. heparin was administered. In each patient, the AV nodal artery was selectively catheterized. A 0.014-in.-diameter guide wire (Hi-Torque Floppy, Advanced Cardiovascular Systems, Inc., or Seeker, Target Therapeutics, Inc.) was first advanced through the coronary guiding catheter and positioned in the AV nodal artery. After the guide wire was positioned, an infusion catheter with a 3F proximal shaft diameter, a 2.2F distal shaft diameter, and a radiopaque tip marker (Tracker, Target Therapeutics) was then advanced over the guide wire and positioned in the AV nodal artery. Proper positioning of the infusion catheter was confirmed by injection of radiographic contrast material through the guiding catheter (Figures 1 and 2, bottom panels). Once the infusion catheter was properly positioned, the guide wire was removed, enabling the proximal end of the infusion catheter to be connected to an infusion pump (Harvard Apparatus, South Natick, Mass.).

In patients 2, 4, 5, and 8, acetylcholine dissolved in normal saline was infused into the AV nodal artery at a dose of 50 \(\mu\)g/min with a constant flow rate of 0.25 ml/min. Patient 3 received a selective AV nodal artery infusion of 0.1 mg/min procainamide at a flow rate of 0.125 ml/min. In each of these five patients, selective AV nodal artery drug infusion was administered for 10 minutes, after which a complete sequence of programmed stimulation was performed several times according to the same protocol used in the control state. On completion of the stimulation protocol, the intracoronary drug infusion was discontinued, and 10 minutes were allowed for the effects of the intracoronary drug infusion to dissipate. After this period of drug washout, selective AV nodal artery infusion of normal saline was begun, again with a flow rate of 0.25 ml/min (patients 2, 4, 5, and 8) or 0.125 ml/min (patient 3). Ten minutes after beginning the selective intracoronary saline infusion, a complete sequence of programmed stimulation was again repeated several times with the same stimulation protocol as used in the control state. In patient 8, after completion of selective AV nodal artery infusion of acetylcholine and saline, the infusion catheter was withdrawn several centimeters and positioned in the midportion of the right coronary artery, proximal to the origin of the AV nodal artery. With the catheter in this position, acetylcholine was again infused at a dose of 50 \(\mu\)g/min and an infusion rate of 0.25 ml/min, after which a complete sequence of programmed stimulation was repeated. Patients 1 and 6 developed varying degrees of AV block soon after the infusion catheter had been positioned in the AV nodal artery (see below). Therefore, these patients did not receive a selective intracoronary infusion of drug or saline. Patient 7, in whom it was not possible to achieve a stable position of the infusion catheter in the AV nodal artery (see below), also did not receive a selective intracoronary infusion of drug or saline. In each patient at the conclusion of the study, the AV nodal artery catheter was removed, and a final coronary angiogram was performed to determine whether any acute arterial damage had occurred during the procedure.

**Statistics**

Electrophysiological parameters, including sinus cycle length, AH interval during sinus rhythm, HV interval during sinus rhythm, Wenckebach paced cycle length, and AV nodal effective refractory period, were examined in the control state and compared with values obtained during selective AV nodal artery drug and saline infusion with a repeated-measures analysis of variance (Statview 512+, Macintosh computer). A \(p\) value of less than 0.05 was considered statistically significant for all analyses.
Results

Pertinent clinical features of the eight patients and the mechanisms of arrhythmia in patients 3–5, 7, and 8 are summarized in Table 1. Patient 1, a 61-year-old woman with a chest pain syndrome and normal coronary arteries, patient 2, a 49-year-old woman with moderate mitral regurgitation and normal coronary arteries, and patient 6, a 47-year-old woman with chest pain and normal coronary arteries, had no clinical history of supraventricular or ventricular tachycardia, nor could any arrhythmia be provoked by programmed electrical stimulation. In patients 3 and 7, recurrent supraventricular tachycardia was due to AV nodal reentry of the usual variety in which anterograde conduction during tachycardia occurred over a slow AV nodal pathway and retrograde conduction occurred over a fast AV nodal pathway. Patient 4 had repeated paroxysms of “unusual” AV nodal reentrant supraventricular tachycardia characterized by anterograde conduction over a fast AV nodal pathway and retrograde conduction over a slow AV nodal pathway. Patient 5 had AV reentrant supraventricular tachycardia using a left-sided concealed accessory pathway as the retrograde limb of the reentrant circuit. Patient 8, a 65-year-old man with chronic pulmonary disease, normal coronary arteries, and normal left ventricular systolic function, had been referred because of frequent episodes of asymptomatic nonsustained ventricular tachycardia.

The results of selective AV nodal artery catheterization in patient 1 are given in Figure 3. Before placement of the infusion catheter in the AV nodal
artery, there was 1:1 AV conduction during sinus rhythm with an AH interval of 140 msec and an HV interval of 55 msec (Figure 3, top panel). The AV nodal Wenckebach paced cycle length was 500 msec, and the AV nodal effective refractory period, measured at a cycle length of 600 msec, was 430 msec. Soon after positioning the infusion catheter in the AV nodal artery, spontaneous AV nodal block of varying degrees appeared, including 4:3 type I second-degree AV nodal block (Figure 3, middle panel) and 2:1 AV nodal block (Figure 3, bottom panel). Consequently, the effects of selective drug infusion into the AV nodal artery could not be assessed in this patient. Within 30 seconds after withdrawing the infusion catheter from the AV nodal artery, 1:1 AV conduction resumed. Provocation of varying degrees of AV nodal block by positioning the infusion catheter in the AV nodal artery was also encountered in patient 6, also precluding assessment of the effects of selective drug and saline infusions in this individual. In patient 7, an individual with a left-dominant coronary circulation, the angle of origin of the AV nodal artery was extremely acute. Although a guide wire could be successfully positioned in the AV nodal branch and the infusion catheter could be advanced into proper position over the guide wire, as soon as the guide wire was withdrawn and the catheter was gently flushed, it prolapsed out of the AV nodal artery back into the distal circumflex artery or posterior descending branch. Repeated attempts failed to achieve a stable position of the catheter in the AV nodal artery. Thus, this was
a third individual in whom the effects of selective drug or saline infusion could not be tested.

**Effects of Selective Drug Infusion on AV Nodal Function**

Patients 1 and 6 were the only individuals in whom impaired AV nodal conduction during sinus rhythm appeared as a consequence of selective AV nodal artery catheterization. Table 2 lists individual and mean values for patients 2–5 and 8 of sinus cycle length, and AH and HV intervals during sinus rhythm in the control state and compares them with values determined after selective AV nodal artery catheterization during selective infusion of drug and normal saline. By repeated-measures analysis of variance, sinus cycle length and HV intervals did not change significantly during AV nodal artery infusion of drug or saline compared with control values (Table...
2). Although AH intervals during selective drug infusion showed a trend toward being longer than those in the control state or during saline infusion, this difference did not achieve statistical significance (Table 2).

In contrast to the minimal effect observed on AH intervals during sinus rhythm, selective AV nodal artery drug infusion caused marked prolongation of AV nodal effective refractory period and Wenckebach paced cycle length in patients 2–5 and 8, an effect that was not seen during selective infusion of saline (p<0.05, Figure 4). For the group, the mean AV nodal effective refractory period increased to 543±228 msec during drug infusion compared with a control value of 312±52 msec, whereas AV nodal effective refractory period was essentially unchanged during infusion of saline (326±41 msec, Figure 4). Similarly, AV nodal Wenckebach paced length was longer during drug infusion (572±217 msec) than in the control state (360±47 msec) or during infusion of saline (374±73 msec, p<0.05, Figure 4). These effects of selective AV nodal artery drug infusion were not due to accidental reflux of drug out of the AV nodal artery with delivery of drug to other sites, as evidenced by results in patient 8. In this individual, administration of acetylcholine into the midportion of the right coronary artery proximal to the origin of the AV nodal branch did not prolong AV nodal effective refractory period (340 msec) or Wenckebach cycle length (400 msec) compared with control values (310 and 400 msec, respectively). However, the same dose of acetylcholine infused into the AV nodal artery caused marked prolongation of AV nodal effective refractory period (880 msec) and Wenckebach cycle length (880 msec).

### Effects of Selective Drug Infusion on Arrhythmia Induction

Selective AV nodal artery infusion of procainamide or acetylcholine had a striking effect on inducibility of arrhythmias in patients 3–5, three individuals who had been referred for study because of recurrent supraventricular tachycardia. However, saline infusion had no effect on inducibility of arrhythmia. Results in patient 3 were illustrative of the effect of drug infusion. In the control state, sustained AV nodal reentrant supraventricular tachycardia could be easily and reproducibly induced but only by two closely coupled atrial premature stimuli delivered during atrial pacing (Figure 5, top panel). With this mode of stimulation, the second atrial premature depolarization blocked antegrade in the fast AV nodal pathway, conducted antegrade with delay over the slow AV nodal pathway (note prolonged A3H3 interval, Figure 5, top panel), and then conducted retrogradely over the slow AV nodal pathway, initiating sustained AV nodal reentry. During infusion of procainamide into the AV nodal artery, tachycardia could no longer be induced with this or any mode of programmed stimulation, even though the entire range of premature beat intervals that had induced tachycardia in the control state was carefully scanned. Although the second of two atrial premature depolarizations provoked during atrial pacing was still able to conduct antegrade over the slow AV nodal pathway with considerable delay (note markedly prolonged A3H3 interval, Figure 5, bottom panel), retrograde conduction over the fast AV nodal pathway was abolished (note absence of atrial echo after A3, Figure 5).

### Table 2. Conduction Intervals During Sinus Rhythm

<table>
<thead>
<tr>
<th>Patient</th>
<th>SCL (msec)</th>
<th>AH (msec)</th>
<th>HV (msec)</th>
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<tbody>
<tr>
<td></td>
<td>C</td>
<td>D</td>
<td>S</td>
</tr>
<tr>
<td>2</td>
<td>1,100</td>
<td>1,200</td>
<td>1,050</td>
</tr>
<tr>
<td>3</td>
<td>680</td>
<td>715</td>
<td>810</td>
</tr>
<tr>
<td>4</td>
<td>1,300</td>
<td>1,150</td>
<td>1,000</td>
</tr>
<tr>
<td>5</td>
<td>580</td>
<td>590</td>
<td>620</td>
</tr>
<tr>
<td>8</td>
<td>1,050</td>
<td>880</td>
<td>800</td>
</tr>
<tr>
<td>Mean±SD</td>
<td>942±302</td>
<td>907±266</td>
<td>856±173</td>
</tr>
<tr>
<td>p value*</td>
<td>0.43</td>
<td>0.09</td>
<td>0.08</td>
</tr>
</tbody>
</table>

C, control; D, selective AV nodal artery drug infusion; S, selective AV nodal artery saline infusion; SCL, sinus cycle length.

*By repeated-measures analysis of variance.

![Figure 4. Plots of AV nodal effective refractory periods (AVN-ERP, left panel) and AV nodal Wenckebach paced cycle length (WCL, right panel) in patients 2–5 and 8 measured in control state, during AV nodal artery drug infusion, and during AV nodal artery saline infusion. ACH, acetylcholine; PCA, procainamide.](http://circ.ahajournals.org/doi/abs/10.1161/01.CIR.89.4.823?journalCode=circ)
Patient 4 was another individual in whom selective AV nodal artery drug infusion prevented induction of supraventricular tachycardia by blockade of ventriculoatrial conduction. This patient's repetitive brief paroxysms of supraventricular tachycardia were due to "unusual" AV nodal reentry; the tachycardia in the control state could be reproducibly provoked by ventricular premature stimuli delivered over a wide range of coupling intervals during sinus rhythm. Tachycardia induction was always due to retrograde conduction of the ventricular premature depolarization over a slow AV nodal pathway (note prolonged S1A1 interval and normal retrograde atrial activation sequence of A1, Figure 6, top panel), followed by anterograde conduction over a fast AV nodal pathway (Figure 6, top panel). During selective AV nodal artery infusion of acetylcholine, tachycardia could no
Figure 6. Tracings of effect of AV nodal artery infusion of acetylcholine (ACH) on inducibility of "unusual" AV nodal reentrant supraventricular tachycardia in patient 4. Note absence of ventriculoatrial conduction of ventricular paced beats during ACH infusion with presence of sinus capture beats (last three QRS complexes, bottom panel). One-second time lines are at top of upper panel. (See text for discussion.) HRA, CSp, CSd, HIS, RV, bipolar electrograms recorded from high right atrium, proximal coronary sinus, distal coronary sinus, His bundle, and right ventricular apex, respectively; Ae, atrial echoes; P, sinus P waves.
longer be induced with this or any other mode of programmed stimulation due to complete blockage of ventriculoatrial conduction by the drug (note presence of sinus P waves and capture beats during ventricular pacing, Figure 6, bottom panel).

In patient 5, an individual with AV reentrant supraventricular tachycardia using a concealed left-sided accessory pathway as the retrograde limb of the circuit, selective AV nodal artery infusion of acetylcholine also abolished tachycardia but by a different mechanism than that seen in patients 3 and 4. In the control state, sustained tachycardia was reproducibly provoked by single atrial premature stimuli delivered over a range of coupling intervals during atrial pacing (Figure 7, top panel). These atrial premature depolarizations conducted anterogradely over the AV node and then retrogradely over the accessory pathway (note eccentric atrial activation sequence of atrial echoes after A2, Figure 7, top panel), thereby initiating sustained reentry. Tachycardia could also be provoked by ventricular premature stimuli delivered during sinus rhythm or ventricular pacing. During selective AV nodal artery infusion of acetylcholine, AV reentry could no longer be provoked by atrial premature stimuli. Atrial impulses that previously provoked tachycardia now blocked anterogradely in the AV node (note absence of H2 after A2, Figure 7, middle panel). Of interest, one or two AV reentrant beats could still be induced by programmed ventricular stimulation (note ventricular echo after retrogradely conducted A2, Figure 7, bottom panel). During selective AV nodal artery infusion of saline in patient 5, sustained tachycardia was again easily induced (Figure 8). Hand-injection of 2.0 μg acetylcholine into the AV nodal artery during this tachycardia caused prompt termination of the arrhythmia due to anterograde block in the AV node (note tachycardia termination after a retrograde P wave, Figure 8).

Discussion

Sustained ventricular tachycardia can be interrupted by selective injection of iced saline into a branch of the coronary arterial circulation considered to supply a part of the reentry circuit responsible for the tachycardia.4 Similarly, induction of sustained ventricular tachycardia by programmed stimulation can be prevented by selective infusion of an antiarrhythmic drug into the appropriate coronary artery.5 More recently, Brugada et al6 demonstrated that intracoronary ethanol injection can result in permanent ablation of drug-resistant incessant ventricular tachycardia. These data support the hypothesis first advanced by Inoue et al that the coronary arterial circulation can be used for delivery of ablative agents to sites of origin of arrhythmias regardless of the location in the heart.

The feasibility of intracoronary chemical ablation to manage drug-resistant supraventricular arrhythmias is just beginning to be explored. Because the AV node receives its blood supply from a discrete source, the AV nodal artery, it should be possible to cause permanent impairment of AV nodal function exclusively by delivery of an ablative substance through this vessel. Such an effect might in turn aid in the management of drug-resistant paroxysmal supraventricular tachycardias that use the AV node as part of the reentry circuit as well as atrial flutter or fibrillation accompanied by an unacceptably rapid ventricular rate. Previous studies in dogs, a species in which the coronary arterial circulation is invariably left dominant, have shown that the AV nodal artery can be selectively catheterized with steerable guide wires and flexible infusion catheters, which are routinely used during diagnostic cardiac catheterization.7-9 Such studies have also demonstrated that selective catheterization of the AV nodal artery in dogs can be used to manipulate AV nodal function.7-9 More recently, similar techniques have been applied in human subjects.10,11

Limitations of AV Nodal Artery Catheterization

The present study, which was carried out in a small, highly selected group of individuals, clearly demonstrates that selective AV nodal artery catheterization can be performed successfully in human subjects with either right- or left-dominant coronary arterial circulations. However, our results also demonstrate that this method of manipulating AV nodal function is technically demanding and may not be feasible in all patients. In the present study, AV nodal artery catheterization was not attempted in three subjects who were otherwise appropriate candidates for study because of the inability to achieve a stable position of the angioplasty guiding catheter in the right coronary artery (two subjects) or provocation of ostial spasm by the guiding catheter (one subject). Among the eight individuals in whom AV nodal artery catheterization was actually attempted, the artery could not be selectively catheterized in one subject; a complete study of the effects of selective drug and saline infusion on AV nodal function was possible in only five of the eight subjects (62%) in whom it was attempted. Of note, only individuals with angiographically normal coronary arteries were recruited for this study. If the study population had included individuals with coronary artery stenoses, the success rate might have been even lower.

Although none of the patients in the present study experienced any complications resulting from selective AV nodal artery catheterization, one cannot ignore the potential risks associated with this technique, including the possibility of causing severe acute coronary arterial damage with resulting myocardial infarction.11 Coronary angiography performed in each of our subjects at the conclusion of their study revealed no angiographic evidence of acute damage to the AV nodal artery or other coronary arterial branches. However, it is conceivable that manipulation of intracoronary guide wires and catheters might cause subtle injury to the endothelium that could lead to intimal hyperplasia and
Figure 7. Tracings of effect of AV nodal artery infusion of acetylcholine (ACH) on inducibility of AV reentrant supraventricular tachycardia in patient 5. Time lines are at top of upper panel. (See text for discussion.) HRA, CSp, CSd, HIS, bipolar electrograms recorded from the high right atrium, proximal coronary sinus, distal coronary sinus, and His bundle, respectively; H (bottom panel), His potential resulting from anterograde AV nodal conduction of $A_2$; Ae, atrial echo due to retrograde conduction over a concealed left-sided accessory pathway.
eventual stenosis at the site of injury, particularly in individuals prone to atherosclerotic coronary artery disease. To minimize these potential acute and chronic complications, selective AV nodal artery catheterization should be performed only by individuals with considerable experience in the techniques of selective intracoronary catheterization.

**Clinical Implications**

After selective catheterization of the AV nodal artery, patients 1 and 6 developed varying degrees of AV block that could be localized to the AV node. AV conduction returned quickly to normal after withdrawal of the AV nodal artery catheter, suggesting that the catheter itself had been at least partially occluding the vessel and that the transient AV nodal block had been due to ischemia. It is noteworthy that both of these patients were women of small stature in whom the AV nodal artery was relatively small. It is also of interest that both of these patients were being treated with a calcium channel–blocking agent at the time of study, which may have rendered their AV nodes more susceptible to ischemia. In contrast, there was no evidence for catheter-induced ischemia of the AV node in patients 2–5 or 8. In these individuals, objective measures of AV nodal function during selective AV nodal artery infusion of saline were unchanged compared with the control state.

Because AV nodal function and inducibility of arrhythmia were the same during AV nodal artery saline infusion as in the control state in patients 2–5 and 8, the effects observed during AV nodal artery infusion of procainamide and acetylcholine must have represented true drug effects. All five of these individuals had significant increases in AV nodal effective refractory period and Wenckebach paced cycle length during drug infusion. Furthermore, in three patients with a history of paroxysmal supraventricular tachycardia, the effects of selective AV nodal artery drug infusion on AV nodal function led to abolition of the arrhythmia that had prompted their referral. Impairment of anterograde AV nodal conduction was the mechanism by which drug infusion abolished arrhythmia in patient 5. Atrial premature impulses that had initiated AV reentrant supraventricular tachycardia in the control state were no longer able to do so during drug infusion due to anterograde block in the AV node. Furthermore, sustained tachycardia induced by ventricular premature impulses in the control state was no longer possible during drug infusion because eventual anterograde blockade of the atrial echoes occurred in the AV node.

Of particular interest were observations made in patients 3 and 4. These individuals also had impairments of anterograde AV nodal function during selective drug infusion but in addition had even more striking drug effects on retrograde AV nodal conduction. In fact, complete blockade of retrograde AV nodal conduction occurred, an effect that was the principal mechanism by which tachycardia was abol-

**Figure 8.** Tracings of termination of AV reentrant supraventricular tachycardia by bolus injection of acetylcholine (ACH) into the AV nodal artery of patient 5. Note termination of tachycardia due to failure of a retrograde P wave (A) to conduct anterogradely to the ventricles. One-second time lines are at top of figure. (See text for discussion.) HRA, bipolar electrogram recorded from the high right atrium.
ished. During selective drug infusion, atrial premature impulses in patient 3, although still able to conduct anterogradely over the slow AV nodal pathway, were unable to conduct retrogradely over the fast AV nodal pathway and therefore could not initiate tachycardia. Similarly, abolition of ventricular prema-

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