Control of Rapid Ventricular Response by Radiofrequency Catheter Modification of the Atrioventricular Node in Patients With Medically Refractory Atrial Fibrillation

Gregory K. Feld, MD; R. Peter Fleck, MD; Osamu Fujimura, MD; David L. Prothro, MD; Tristram D. Bahnson, MD; Manuel Ibarra, MD

Background Pharmacological control of rapid ventricular response to atrial fibrillation may be difficult in some patients. Alternative treatments, including curative surgery or atrioventricular (AV) node ablation with pacemaker implantation, have significant potential morbidity. In view of evidence that dual AV nodal physiology may exist in a significant percentage of the population, even in those without AV nodal reentrant tachycardia, we postulated that control of ventricular response might be achieved by radiofrequency (RF) catheter ablation in the region of the AV nodal slow pathway with its short refractory period.

Methods and Results Ten patients underwent attempted AV node modification using a 4-mm-tipped electrode catheter positioned in the middle or posterior septum, between the His bundle and coronary sinus ostium on the tricuspid valve annulus. RF energy was applied at 16 to 30 W for up to 60 seconds, until average ventricular response fell below 100 beats per minute. Reduction of maximal ventricular response below 120 beats per minute was confirmed with atropine 1 mg IV. If required, additional ablations were performed progressively more posteriorly up to the coronary sinus ostium. Patients with successful AV node modification were discharged off AV node-blocking drugs and followed in the clinic at regular intervals. Twenty-four-hour ambulatory ECG recordings and/or treadmill stress tests were obtained before and after ablation for statistical comparison of maximum ventricular rate. Resting average ventricular rate was determined during electrophysiology study before and after ablation. In 7 of 10 patients (70%), maximum ventricular rate was reduced from a mean of 164±12 to 123±16 beats per minute (P<.01) and average ventricular rate from a mean of 128±11 to 83±10 beats per minute after ablation. Mean minimum ventricular rate was 54±11 beats per minute after ablation. These 7 patients have remained symptom free from rapid ventricular response for a mean of 14±8 months (range, 1 to 22). Three remain off all AV node-blocking drugs, 3 remain on digoxin alone, which was previously ineffective, and 1 remains on a β-blocker for angina. In the 3 patients who did not respond to AV node modification, complete AV node ablation and permanent pacemaker implantation was performed in 2 and DC cardioversion after amiodarone loading was performed in 1.

Conclusions RF catheter modification of AV node conduction is effective in controlling rapid ventricular response to atrial fibrillation in a significant percentage of medically refractory patients. A possible mechanism of RF modification of AV node conduction is AV nodal slow pathway ablation. Large-scale clinical trials will be needed to determine the overall efficacy and safety of this technique. (Circulation. 1994;90:2299-2307.)

Key Words • fibrillation • radiofrequency • ablation • atrioventricular node • pacemakers

Controlling rapid ventricular response to atrial fibrillation in patients with normal atrioventricular (AV) node function is often difficult, requiring large doses of AV node-blocking drugs, which may also produce side effects. Alternative treatments include surgical procedures to prevent recurrence of atrial fibrillation or AV node ablation followed by permanent pacemaker implantation to relieve symptoms associated with rapid ventricular response.1-6 Both of these nonpharmacological approaches have drawbacks, however, including the risk and morbidity associated with open heart surgery and the development of pacemaker dependency associated with AV node ablation.1-6

We proposed that radiofrequency catheter modification of AV nodal conduction for the control of rapid ventricular response to atrial fibrillation might be a preferable alternative if it were found to be safe and effective.7-8 Although requiring an invasive procedure, it would obviate the need for either curative open heart surgery or palliative AV node ablation with permanent pacemaker implantation.7-8 We postulated that ablation should be attempted in the low midseptal or low posteroseptal right atrium, based on several well-known observations.9-22 First, a significant percentage of patients undergoing electrophysiological study for arrhythmias other than AV nodal reentry have been noted to have dual AV nodal physiology, suggesting that they too have more than one atrial input into the AV node.9-21 In addition, experimental studies have shown the presence of dual atrial inputs into the normal canine AV node, consisting of a superior AV bundle and a proximal AV bundle.22 Clinical studies have shown that these separate AV nodal inputs have different electrophysiological characteristics and location, consisting of a fast pathway located anterior to the compact AV node with a long refractory period and a slow pathway located posterior to the AV node with a shorter refractory period.9-20 It is

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this spatial separation of pathways that has resulted in the high degree of efficacy and safety of slow pathway ablation in patients with AV nodal reentrant supraventricular tachycardia. Studies have shown that histograms of the ventricular response to atrial fibrillation may manifest as a bimodal distribution of RR intervals in some patients, which might suggest the underlying presence of dual AV nodal physiology. In patients with rapid ventricular response to atrial fibrillation, we attempted radiofrequency catheter ablation in the low midseptal or posteroseptal right atrium, where the AV nodal slow pathway would be expected. Theoretically, by ablating the AV nodal slow pathway with its short refractory period and leaving intact the AV nodal fast pathway with its long refractory period, we would limit the frequency of impulses traversing the AV node and possibly regularize the response as well.

**Methods**

Ten patients with a history of medically refractory atrial fibrillation and symptomatic rapid ventricular response were studied (Table 1). In each patient, symptomatic periods of rapid ventricular response to atrial fibrillation were documented by 24-hour ambulatory ECG and/or treadmill stress test.

All patients were admitted to the hospital, where they underwent routine preoperative evaluation including history and physical examination, chemistry, hematology, and coagulation panel, urinalysis, and 12-lead ECG. If tolerated, all AV node–blocking drugs were withheld for 24 hours. Each patient gave written informed consent for both AV node modification and AV node ablation with permanent pacemaker implantation. The use of radiofrequency catheter ablation for the treatment of cardia arrhythmias was approved by the Human Subjects Committee at UCSD School of Medicine.

Under local anesthesia and light sedation, using the Seldinger technique, 6F quadrupolar electrode catheters (Bard Electrophysiology, Inc) were placed in the coronary sinus through the right internal jugular vein and the right ventricular apex and His bundle region through the left femoral vein. A 6F steerable quadrupolar electrode catheter (Mansfield, Inc) with a 4-mm distal electrode for ablation was positioned at the low midseptal or posteroseptal right atrium through the right femoral vein. Surface ECG leads I, aVF, V₁, and right atrial, coronary sinus (proximal, middle, and distal bipoles), His bundle, and right ventricular endocardial electrograms were recorded on an EVR-16 physiological recorder (PPG Industries, Inc). Blood pressure was monitored by automated cuff. Radiofrequency catheter ablation was performed using a custom-built generator providing 550 Hz of unmodulated current (American Cardiac Ablation Co, Medical Scientific, Inc) through the 4-mm distal electrode on the steerable mapping catheter, grounded to the patient’s back with a standard electrosurgical grounding pad. During ablation, applied voltage and measured current were recorded continuously, and impedance was monitored. Radiofrequency energy was delivered at 16 to 30 W for up to 60 seconds, during which time the filtered surface ECG was monitored for change in ventricular response.

Initially, the 4-mm distal ablation electrode was positioned midway between the His bundle and coronary sinus ostium on the tricuspid valve anulus (Fig 1) so that approximately a 1:4 to 1:2 atrial:ventricular electrogram ratio was recorded, with no His bundle potential (Fig 2). Radiofrequency energy was then applied until a decrease in ventricular response below 100 beats per minute or an impedance rise was observed. Additional ablations were performed progressively more posteriorly, every few millimeters up to the coronary sinus ostium if ablation at the initial site proved to be unsuccessful in reducing ventricular response.

If attempts to modify AV node conduction initially appeared successful, patients were given 1 mg atropine IV to ensure that their maximal ventricular response did not exceed 120 beats per minute, in which case additional radiofrequency energy was applied at additional sites until the maximal ventricular response was below 120 beats per minute.

If no decrease in ventricular response was observed after multiple ablation attempts in the low middle to posterior right atrium, the 4-mm distal electrode was then positioned near the His bundle catheter, where the largest His potential was recorded, withdrawn just proximal to this area, and the AV node ablated. After complete AV block was confirmed, all catheters except the right ventricular were removed, and a permanent rate-responsive ventricular pacemaker was implanted.

All patients were observed on telemetry for 24 to 48 hours after their procedure and then discharged to follow-up in the arrhythmia clinic at 1 week, 1, 3, and 6 months, and every 6 months thereafter. At 1 week, a history of arrhythmia symptoms, physical examination, and 12-lead ECG were performed. Just before the 1-month visit, a 24-hour ambulatory ECG and treadmill stress test were performed. At all subsequent visits, only history, physical examination, and a 12-lead

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**Table 1. Clinical Characteristics of Patients**

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<th>Patient</th>
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<th>Duration AFIB, mo</th>
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Mean±1 SD: 62±10 58±45

AFIB indicates atrial fibrillation; CAD/CABG, coronary artery disease/coronary artery bypass grafting; DCM, dilated cardiomyopathy; LVEF, left ventricular ejection fraction; MS, mitral stenosis; MVR, mitral valve replacement; RHD, rheumatic heart disease; and NL, normal.
ECG were performed unless a change in patient symptoms was reported. Routine pacemaker checks were performed on those patients receiving permanent pacemakers after complete AV node ablation.

**Statistical Analysis**

Mean ventricular rate was determined for each patient during a 1-minute ECG recording obtained immediately before radiofrequency ablation and again at the completion of the procedure. The maximum ventricular rate, defined as the shortest RR interval observed for each patient, was determined from the 24-hour ambulatory ECG, treadmill stress test, or the electrophysiology study before and after radiofrequency ablation. The mean and standard deviation for each parameter for the group was determined, and the statistical significance of the difference before and after radiofrequency ablation was calculated using the Student's t test for paired data. A value of $P<.05$ was considered statistically significant.

**Results**

**Clinical Characteristics of Patients**

There were 7 male and 3 female patients, ranging in age from 38 to 75 (62±10 years), all except 2 of whom had chronic atrial fibrillation (58±45 months) and reported symptoms of severe dizziness, shortness of breath, chest pain, fatigue, or syncope during periods of rapid ventricular response (see Table 1). Two patients (patients 5 and 10) had paroxysmal atrial fibrillation and a rapid ventricular response causing dyspnea, dizziness, and/or syncope. All patients had failed attempts to control rapid ventricular response to atrial fibrillation with two or more AV node–blocking drugs. Six patients (patients 1, 4, 7, 8, 9, and 10) had documented underlying heart disease, including rheumatic mitral stenosis with previous mitral commissurotomy (patient 1), idiopathic dilated cardiomyopathy (patients 7, 8, and 9), and coronary artery disease with or without coronary artery bypass surgery (patients 4 and 10). Two patients (patients 2 and 10) had a permanent pacemaker previously implanted for sick sinus syndrome, which was programmed to VVI at a low rate and was rarely observed to pace on 24-hour ECG. The remaining three patients (patients 3, 5, and 6) had no evidence of clinically significant heart disease other than atrial fibrillation.

**Short-term Effects of Radiofrequency Catheter Modification of AV Node Conduction**

In 7 of 10 patients (patients 1, 2, 3, 6, 7, 9, and 10), an abrupt decrease in ventricular response to atrial fibrillation was observed during one or more radiofrequency energy applications (17±12; range, 2 to 34 for 5 to 60 seconds) to the low midseptal or posteroseptal right atrial septum. In these 7 patients, the mean maximum ventricular rate before ablation was 164±12 beats per minute, which decreased to 123±16 beats per minute after ablation ($P<.01$). The mean average ventricular rate for the group was 128±11 beats per minute before ablation and decreased to 83±10 beats per minute after ablation. The mean minimum ventricular rate observed during 24-hour ECG recordings after ablation in these 7 patients was 54±11 beats per minute, including the 2 patients (patients 2 and 10) with previously implanted pacemakers that were rarely noted to pace.

An example of a successful radiofrequency modification of AV nodal conduction is seen in Fig 3 from
FIG 3. A, Rhythm strip recorded immediately before electrophysiology study on patient 1 shows resting ventricular rate of 190 beats per minute. B, Surface ECG leads I, aVF, and V1, right ventricular endocardial, current and voltage recordings from patient 1. Note abrupt decrease in ventricular response to atrial fibrillation immediately after onset of radiofrequency energy application. C, 12-Lead ECG recorded immediately after completion of atrioventricular node modification on patient 1 shows controlled ventricular rate of 70 beats per minute.
patient 1. Just before entering the electrophysiology laboratory, within 24 hours of withholding AV node-blocking drugs, a rhythm strip shows an average resting ventricular rate of 190 beats per minute (Fig 3A). Immediately after the onset of radiofrequency energy application to the low midseptal right atrium, a persistent decrease in average ventricular rate is noted to 75 beats per minute, along with some degree of regularization of ventricular rhythm (Fig 3B). The day after ablation, a resting 12-lead ECG shows a controlled average ventricular rate of 70 beats per minute (Fig 3C). In follow-up 1 month after ablation, a treadmill stress
test produced a maximum ventricular rate of 120 beats per minute off medication compared with a preablation maximum ventricular rate of 190 beats per minute on medication.

In another patient (patient 3), a resting average ventricular rate of 150 beats per minute is noted before ablation while still on AV node–blocking drugs (Fig 4A). Shortly after the onset of radiofrequency energy application in the low posteroceptal right atrium, an abrupt decrease in ventricular rate to an average of 60 beats per minute is noted (Fig 4B). A 12-lead ECG obtained in follow-up clinic 1 month after ablation shows a controlled average ventricular rate of 70 beats per minute off all AV node–blocking drugs (Fig 4C). In this same patient (patient 3), a treadmill stress test performed after ablation demonstrates a progressive yet controlled increase in ventricular rate from 75 beats per minute at rest to 120 beats per minute after 6 minutes of exercise (Bruce protocol, stage 2, 6 METS), followed by a gradual decrease in ventricular rate to baseline values within 5 minutes into recovery (Fig 5, A and B). An hourly plot of maximum ventricular rate recorded on 24-hour ambulatory ECG from patient 3 before and after AV node modification demonstrates a reduction in

![Graph](http://circ.ahajournals.org/)

**Fig 4.** C, 12-Lead ECG from patient 3 1 month after atroventricular node modification shows resting ventricular rate of 70 off all atroventricular node–blocking drugs.

<table>
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| Mean±1 SD | 164±12 | 123±16* | 128±11 | 83±10* | 54±11 |

**Table 2.** Results of RF Catheter Modification of the AV Node for Control of Rapid Ventricular Response to Atrial Fibrillation

HR indicates heart rate; RF, radiofrequency; AV, atrioventricular; A, patient treated with amiodarone and then cardioverted; and P, AV node modification unsuccessful, complete AV block induced by anterior approach and pacemaker implanted.

*P<.01 comparing maximum and mean heart rates before and after AV node modification (statistical analysis does not include patients 4, 5, 8, who did not respond).
maximum ventricular rate from baseline at nearly all hours of the day and night (Fig 6).

In 3 patients (patients 4, 5, and 8), no decrease in ventricular response was observed during attempted AV nodal modification. Therefore, in 2 patients (patients 4 and 5), complete AV node ablation was accomplished using the anterior approach, and permanent rate-responsive pacemakers were implanted. The third patient (patient 8) chose to undergo DC cardioversion of atrial fibrillation after 1 month of amiodarone loading.
and has been maintained in normal sinus rhythm on chronic amiodarone therapy. In patient 5, with paroxysmal atrial fibrillation, programmed stimulation during sinus rhythm demonstrated no evidence of dual AV nodal physiology before induction of atrial fibrillation and AV node ablation.

**Long-term Outcome and Symptom Control After Radiofrequency AV Node Modification**

The 7 patients (patients 1, 2, 3, 6, 7, 9, and 10) with initially successful AV nodal modification have been followed up for a mean of 14±8 months (range, 1 to 22) and have remained symptom free from rapid ventricular response to atrial fibrillation. One patient (patient 6) had a slight, gradual increase in his maximum ventricular rate up to 150 beats per minute on 24-hour ECG within several months after ablation, but this has been controlled on oral digoxin 0.25 mg daily, which was previously ineffective. Two patients (patients 7 and 9) remained on digoxin after ablation for a history of congestive heart failure and 1 (patient 10) on a β-blocker for a history of angina. Thus, a chronic decrease in ventricular rate was achieved in 7 of 10 patients, or 70%. (See Table 2).

In the 3 patients (patients 4, 5, and 8) who did not respond to AV node modification, in whom complete AV node ablation with permanent ventricular rate-responsive pacemaker implantation (patients 4 and 5) and DC cardioversion after oral loading with amiodarone was performed (patient 8), symptoms related to rapid ventricular response have also been eliminated.

Late AV block or symptomatic bradycardia has not been observed in any of the patients undergoing a successful AV node modification (patients 1, 2, 3, 6, 7, 9, and 10) or in the patient in whom the procedure was unsuccessful who underwent subsequent cardioversion on amiodarone (patient 8).

**Discussion**

This study has demonstrated the feasibility of chronically controlling rapid ventricular response to atrial fibrillation by radiofrequency catheter modification of AV node conduction. Although we cannot predict the percentage of patients in the general population with atrial fibrillation who will ultimately respond to this approach based on this small study, it is nevertheless a promising alternative to chronic pharmacological rate control or complete AV node ablation and permanent pacemaker implantation.

**Possible Mechanisms for Slowing of Ventricular Response by Radiofrequency Catheter Modification of AV Node Conduction**

The abrupt and persistent slowing of ventricular response during application of radiofrequency energy to the low midseptal or posteroseptal right atrium, without progression to even transient complete AV block, is consistent with ablation of a slow pathway with short refractory period similar to that responsible for dual AV nodal physiology in patients with AV nodal reentrant tachycardia. Unfortunately, all except one of our patients was in sustained atrial fibrillation at the time of electrophysiological study, so that the presence or absence of dual AV nodal physiology could not be confirmed before ablation. However, one patient with paroxysmal atrial fibrillation who did not respond to AV node modification (patient 5) did not have dual AV nodal physiology.

A recent study showing a decrease in ventricular response to pacing-induced atrial fibrillation after ablation of the AV nodal slow pathway in patients with AV nodal reentrant supraventricular tachycardia lends further support to our hypothesis.28 There is also evidence from analysis of RR interval histogram patterns during atrial fibrillation that supports our hypothesis that some patients have underlying dual AV nodal physiology.23-27 For example, the most commonly observed RR interval histogram patterns, including a bimodal distribution or unimodal distribution with a shoulder to the right, have been postulated to be due electrophysiological phenomena that could result from underlying dual AV nodal physiology.23-27

Alternatively, it is possible that modification of AV nodal conduction is the result of partial ablation or nonspecific damage to the compact AV node. This cannot be ruled out from our present study. However, we feel that this is unlikely because the location of energy applications in our successful cases was anatomically well posterior to the compact AV node and His bundle.14-20 Furthermore, previous attempts to modify AV node conduction using an anatomically anterior or fast pathway approach, which would be much more likely to result in partial ablation or nonspecific damage to the compact AV node, have proven unsuccessful, often resulting in high-grade or complete AV block or recurrence of rapid ventricular response within a short time after ablation.29,30

Conversely, failure of this procedure to control rapid ventricular response in some patients may be due to the absence of underlying dual AV nodal physiology or the inability to eliminate slow pathway conduction, as is sometimes observed in patients with AV nodal reentrant supraventricular tachycardia.17-20

**Conclusions and Limitations**

Radiofrequency catheter modification of AV node conduction for the control of rapid ventricular response to atrial fibrillation appears to be an effective alternative to chronic pharmacological rate control or AV node ablation with permanent pacemaker implantation, in some patients. A possible mechanism of slowing of ventricular response to atrial fibrillation is ablation of the AV nodal slow pathway with its short refractory period. Proof of this hypothesis will require confirmation of the presence of dual AV nodal physiology during sinus
rhythm and that slow pathway ablation is associated with ventricular rate control during atrial fibrillation.

Thus, a potential limitation of this procedure is its overall applicability, since its success may depend on the percentage of patients with atrial fibrillation in whom dual AV nodal physiology exists and can be ablated. Furthermore, although controlling rapid ventricular response largely eliminated associated symptoms in our patients, this procedure may not alleviate symptoms related to heart rate irregularity or reduced exercise tolerance due to loss of atrial contribution to cardiac output. An additional concern is the possibility of development of AV block late after this procedure is performed. Inadvertent development of AV block was not observed in our patients, which may be due in part to limiting ablation to the low midseptal or posteroseptal right atrium.

All of these concerns will need to be evaluated in a large-scale trial to confirm the overall utility, efficacy, and safety of AV node modification for control of rapid ventricular response in atrial fibrillation.

References

Control of rapid ventricular response by radiofrequency catheter modification of the atrioventricular node in patients with medically refractory atrial fibrillation.  
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