Atrioventricular Nodal Reentrant Tachycardia in Patients Referred for Atrial Fibrillation Ablation: Response to Ablation That Incorporates Slow-Pathway Modification

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Background—Although the most common sites of atrial ectopy that trigger atrial fibrillation (AF) are in or around the pulmonary veins (PVs), atrioventricular nodal reentrant tachycardia (AVNRT) can also cause or coexist with AF. We sought to characterize patients with AF and AVNRT and assess clinical outcomes after ablation.

Methods and Results—To determine the prevalence of concomitant AVNRT and AF, 629 consecutive patients referred for catheter ablation between November 1998 and March 2005 were studied. Electrophysiological studies with programmed stimulation during isoproterenol infusion identified atrial ectopy that initiated AF and the presence of inducible AVNRT. AF ablation consisted of proximal isolation of PVs and elimination of any non-PV trigger of AF, including AVNRT. There were 27 patients (4.3%) who had inducible AVNRT at the time of AF ablation. Of these, 13 underwent AVNRT ablation without PV isolation. Compared with the rest of the cohort, patients with AVNRT and AF were younger at the time of symptom onset (age 36.8±13.8 years versus 48.2±11.7 years; P<0.01). Freedom from AF with or without previously ineffective antiarrhythmic medication was similar in both groups (96.3% versus 90.7%; mean follow-up 21.4±9.4 months); however, patients with AVNRT targeted for ablation were more likely to be AF free while not taking any antiarrhythmic medication after a single procedure during the follow-up period (87.5% versus 54.7%; P<0.01) and had fewer complications (0% versus 2.5%; P=0.30). Twelve of the 13 patients who underwent slow-pathway ablation without left atrial ablation remained AF free without the need for antiarrhythmic medication after a single procedure.

Conclusions—AVNRT is an uncommon AF trigger seen more frequently in younger patients. Ablation of AVNRT in patients with AF was associated with improved outcomes compared with those with other triggers of AF. (Circulation. 2006;114:191-195.)

Key Words: atrioventricular node reentry fibrillation catheter ablation

Effect of atrial ectopy that triggers atrial fibrillation (AF) can cure patients with AF.1–4 Although the most common sites of atrial ectopy that trigger AF are in or around the pulmonary veins (PVs), rapid atrial activity from other sites can also initiate AF, as can atrioventricular nodal reentrant tachycardia (AVNRT).5–7 In addition, AF often coexists with AVNRT, and AVNRT may trigger remote atrial ectopy that, in turn, initiates AF.7–9

If AVNRT plays a role in AF initiation in a particular patient, elimination of this tachycardia is likely required for the success of catheter ablation of AF. Identification of patients with AVNRT-induced AF could affect ablation strategy and improve the efficacy of the procedure. The incidence of AVNRT as a trigger of AF or a contributor to the maintenance of AF has not been described previously. We therefore sought to investigate the incidence of AVNRT as a trigger of AF and assess the efficacy of slow-pathway modification as part of the ablation strategy for treatment of AF in these patients.

Methods

Study Population and Data Collection

The cohort of patients referred to the University of Pennsylvania Health System for AF ablation from November 1998 through March 2005 was analyzed to assess any differences in clinical characteristics and outcomes in patients with AVNRT induced at the time of the ablation procedure. Prospective data collection at the time of the procedure included clinical characteristics of patients, observations noted during ablation, and electrophysiological data. Clinical and demographic data, including past medical history and prior medication use, were obtained from patient interview and review of the medical record. Before the
procedure, a review of available cardiac monitor, ECG, echocardiographic, and angiographic data was recorded for later analysis. Before ablation, documentation of AF with transtelephonic ECG monitoring, 12-lead ECGs, or 24-hour Holter recordings was required in all patients.

Identification and Ablation of AF Triggers

Details about the AF trigger-guided ablation procedure have been described elsewhere. Briefly, antiarrhythmic drug therapy was stopped at least 5 half-lives, or 2 weeks for those taking amiodarone, before the patient was brought to the electrophysiology laboratory. Programmed stimulation in the atrium and ventricle was used to exclude the presence of AVNRT or an accessory AV pathway. If dual-pathway physiology with at least a single echo beat was observed, programmed stimulation on low-dose isoproterenol was performed to determine the inducibility of sustained AVNRT. Confirmation of AVNRT as the arrhythmia mechanism included the observation of dual AV nodal pathways, a critical prolongation of the atrial–His bundle (AH) interval before supraventricular tachycardia (SVT) initiation, a short ventricular–atrial (VA) time of <70 ms during tachycardia, entrainment maneuvers to differentiate AVNRT from atrial tachycardia or AVRT, and/or the HA time during tachycardia being less than the VA time (~40 ms) with pacing at the tachycardia cycle length. AVNRT was considered a non-PV trigger if this rhythm degenerated into AF at any time after its induction or if this was the only rhythm initiated and no PV or other non-PV triggers for AF could be identified. After AVNRT was excluded as an AF trigger, a transseptal puncture was performed, and catheters were placed into the superior PVs for identification of spontaneous PV ectopy. Patients underwent a protocol to provoke AF triggers that included a graded isoproterenol infusion (3, 6, 12, and 20 μg/min) followed by internal cardioversion of spontaneous or burst pacing–induced AF on low-dose (2 μg/min) isoproterenol, if necessary. Although this protocol was advised for all operators, there was some variation in the sequence of trigger identification depending on whether a patient was in AF on arrival to the electrophysiology laboratory, which would prevent programmed stimulation. In addition, there were rare cases when isoproterenol or programmed stimulation did not occur at the discretion of the operator. In the event that AVNRT was discovered as an AF trigger, slow-pathway modification was performed by a standard anatomic approach. On confirmation of successful slow-pathway modification and lack of inducible AVNRT, isoproterenol infusion, as described above, would be initiated to identify potential left atrial AF triggers or PV ectopy, with analysis of surface P-wave morphology and intracardiac electrogams. If there was no evidence of PV ectopy or left atrial AF triggers, a transseptal puncture was not performed.

Electrical isolation of arrhythmogenic PVs (PVs that triggered atrial premature contractions or AF) was guided by a circular mapping catheter and intracardiac echocardiography. Non-PV atrial ectopic foci were targeted only if they triggered AF. Entry and exit block were confirmed for each isolated PV. Isolation of all PVs was performed if no triggers were identified. In addition, the isolation of both ipsilateral PVs was performed if a PV trigger was identified and a single ipsilateral vein source could not be reliably demonstrated or if the ipsilateral PVs were isolated as a single vein source because of the presence of a common ostium. After PV isolation, the isoproterenol infusion protocol was repeated, and any new or recurrent triggers of AF were targeted for ablation. In patients who returned for a repeat ablation procedure, all PVs were isolated or reisolated and any non-PV triggers of AF were again identified with infusion of isoproterenol.

Clinical Follow-Up

Patients were prescribed antiarrhythmic medications, typically class IC if the patient was without structural heart disease or sotalol if they had heart disease, for 6 weeks (paroxysmal AF) to 6 months (persistent and permanent AF). Follow-up included transtelephonic monitoring for 3 weeks before and after ablation, with recordings taken twice daily and with the appearance of any symptoms. Transtelephonic monitoring was also used subsequently if patients experienced recurrent symptoms, as well as for routine monitoring after discontinuation of antiarrhythmic drugs, routinely at 6 to 8 weeks and repeated at 6 months, and before a decision to discontinue warfarin. Outpatient follow-up routinely occurred at 6 to 8 weeks, 6 months, and every 6 months thereafter for at least 1 year. Patients were also instructed to call with any symptoms or documented AF recurrences. A telephone survey of all patients who underwent PV isolation was performed every 6 to 12 months to update the long-term outcome database and track patients lost to follow-up in our healthcare system. The first 6 weeks after ablation were censored from follow-up. Procedural success was defined as those patients with no AF occurrences while not taking any antiarrhythmic medication or while taking previously ineffective antiarrhythmic medication. Any recurrent AF while taking antiarrhythmic therapy was considered a failure of ablative therapy for the purposes of the present study.

Statistical Analysis

Results are expressed as mean±SD. The Fisher exact test was used to compare clinical characteristics between AF patients with and without AVNRT. The Wilcoxon rank-sum test was used for continuous covariates. Multivariable logistic regression was used to measure the association between AVNRT ablation and clinical outcomes after adjustment for potentially confounding variables. Statistical analyses were performed with the SPSS (version 12.0) software program (SPSS, Inc, Chicago, Ill), and statistical significance was defined as a 2-sided probability value <0.05.

The authors had full access to the data and take full responsibility for its integrity. All authors have read and agree to the manuscript as written.

### Results

#### Clinical Characteristics of the Cohort

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<th>AF Without AVNRT (n=602)</th>
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<td>LVEF, %</td>
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<td>LA size, cm</td>
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<td>Paroxysmal AF, n (%)</td>
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LVEF indicates left ventricular ejection fraction; LA, left atrium; CAD, coronary artery disease; HTN, hypertension; CVA, cerebrovascular accident; TIA, transient ischemic attack; MR, mitral regurgitation; SSS, sick sinus syndrome; DM, diabetes mellitus; and CHF, congestive heart failure.

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Catheter ablation of AF was performed in 629 patients from November 1998 through March 2005. Of these, 27 (4.3%) had AVNRT induced during electrophysiological study (EPS) that was targeted for ablation. Of note, none of the 27 patients had preprocedural documentation of regular narrow complex tachycardia or history of palpitations described as regular. Table 1 displays the clinical characteristics of the study cohort. In the unadjusted analy-
this group, 5 patients underwent isolation of all 4 veins, 5 patients had 3 PVs isolated, 3 patients had 2 PVs isolated, and 1 patient had 1 PV isolated. There were 3 patients who had ectopy from the non-PV atrial sites triggering AF that were ablated, in addition to arrhythmogenic PV isolation. Overall, patients with AVNRT had fewer additional triggers of AF than those without AVNRT and therefore had fewer PVs isolated.

There were 3 patients who did not have AVNRT identified at the time of the first AF ablation procedure who subsequently underwent slow-pathway modification. In 1 patient, maintenance of sinus rhythm was not possible until all PVs were isolated and after administration of a procainamide infusion. Programmed stimulation was not performed at this initial study. AVNRT was subsequently discovered as an AF trigger, in addition to reconnected PVs, at the time of the second ablation procedure. In another patient, programmed stimulation was performed while the patient was not taking isoproterenol after PV isolation (which was required to maintain sinus rhythm after cardioversion) without evidence of dual AV nodal physiology or echo beats. After AF recurrence, programmed stimulation during isoproterenol infusion initiated AVNRT. In the third patient, empiric isolation was performed without administration of isoproterenol or programmed stimulation on the first procedure. After AF recurrence, the second procedure identified reconnected PVs and inducible AVNRT.

Outcomes After Slow-Pathway Modification for Treatment of AF and AVNRT

The mean follow-up duration for the entire cohort was 21.4±9.4 months. For the group of patients who underwent slow-pathway modification as part of their ablation procedure, freedom from AF while not taking any antiarrhythmic medication was achieved in 21 (87.5%) of the 24 patients who had AVNRT identified at the time of their initial procedure. This group of 24 patients comprised 9 (81.8%) of 11 patients with and 12 (92.3%) of 13 patients without additional PV isolation as part of their first procedure. This compares with 329 (54.7%) of 602 patients (P<0.01) cured of AF after a single procedure. It is important to note that this latter group represented a diverse group of patients with paroxysmal and persistent AF over a 6-year period who underwent only focal ablation before November 2000 and Lasso-guided PV isolation and focal ablation of non-PV triggers after that date. After adjustment for age, gender, duration of AF, and paroxysmal type of AF, identification and ablation of AVNRT at the time of the initial procedure remained significantly associated with a higher rate of freedom from AF while not taking antiarrhythmic medication (odds ratio [OR] 3.58, 95% confidence interval [CI] 1.31 to 6.18, P=0.03). When we considered those patients who continued to take previously ineffective antiarrhythmic medication, there was no significant difference in rates of sinus rhythm maintenance (87.5% versus 70.9%; P=0.12).

After an additional procedure, 26 (96.3%) of 27 patients with AVNRT (including the 3 patients with AVNRT identified only at the time of the second procedure) achieved sinus rhythm maintenance regardless of whether they continued to take previously ineffective antiarrhythmic medication, compared with 547 (90.9%) of 602 (P=0.31) in the remainder of the cohort. All 3 patients who were found to have AVNRT at the
time of repeat ablation but not at the time of the initial EPS were
cured of AF after slow-pathway modification was performed as
part of this additional procedure, in addition to isolation of all
PVs. In 1 of the 3 patients, ectopy from a reconnected PV
triggered AF, in addition to AVNRT. In the other 2 patients,
ectopy from reconnected PVs, without AF initiation, was
observed before reisolation.

AF cure while not undergoing antiarrhythmic therapy was
achieved in 12 of the 13 patients treated with slow-pathway
modification alone without left atrial ablation. During repeat
EPS, the 1 person with recurrent AF, which occurred during the
first week after ablation, was found to have inducible AVNRT
that required a repeat slow-pathway modification with subse-
quently complete resolution of symptoms.

Complications
There were no incidences of heart block, tamponade, PV
stenosis, or stroke in any of the procedures for the 27 patients
in the group with AVNRT. This freedom from major com-
lications compares favorably to the 2.5% major complica-
tion rate observed in the rest of the cohort.

Discussion

Study Results
In this study, we report a 4.3% incidence of inducible
AVNRT in patients who presented exclusively with drug-
refractory AF at the time of catheter ablation. Although this
represents a small proportion of the total population of
patients who present with AF, it is not insignificant. Furthe-
more, we have demonstrated a high success rate and low
complication rate for long-term cure of AF in these selected
patients treated with slow-pathway modification.

Younger patients with paroxysmal AF were more likely than
others to have inducible AVNRT at the time of ablation.
However, younger age at the time of ablation was not ex-
clusively related to the presence of AVNRT. In fact, 1 patient
with AVNRT as an AF trigger was 75 years old at the time of
ablation. In addition, although the majority of patients with
AVNRT in the present cohort had paroxysmal AF, AVNRT was
also identified in 6 patients with persistent AF and 1 patient with
permanent AF. It remains possible that AF provoked by the
AVNRT in such patients could progress to persistent and
permanent subtypes, without the initial AF trigger recognized.
On the basis of the results of the present study, we suggest a
strategy that includes programmed stimulation in all patients
undergoing AF ablation to identify AVNRT. In young patients
with paroxysmal AF at the time of ablation, it is suggested that
the stimulation be performed before transseptal puncture and left
atrial catheterization and ablation.

The fact that 3 patients who returned for repeat AF ablation
were found to have AVNRT reemphasizes the need to make
programmed stimulation that specifically attempts to identify the
presence of dual AV nodal physiology and retrograde fast-
pathway conduction a standard part of the evaluation at the time
of AF ablation. In each of these 3 patients, either programmed
stimulation was not performed or the atrial refractory period was
reached before AV nodal refractoriness was demonstrated, and
the presence of the substrate for AV nodal reentry was missed.
The presence of complete AF control after the repeat procedure
suggested that this arrhythmia had been playing an important
role as an AF trigger.

A Tale of 2 Tachycardias: Coexistence of AVNRT
and AF
AVNRT as an isolated trigger of AF was first described by Brugada
et al. In that case series, 4 patients who presented exclusively with
AF were subsequently successfully treated with slow-pathway
modification after AVNRT was discovered to be the AF trigger. In
a separate study, Delise et al. reported an AF cure rate of 70% of
patients presenting with both AVNRT and AF with slow-pathway
modification alone. In addition, AF triggered by PV ectopy during
AVNRT has also been described and likely explains why some
patients with AVNRT present with recurrent AF after successful
slow-pathway modification. In the present study, we report
AVNRT as an isolated trigger of AF in addition to its coexistence
with PV and non-PV triggers of AF. In fact, AF triggers were seen
in just over half of the patients with AF and AVNRT in the present study. Notably, only 9 of the 14 patients with slow-pathway modification and PV isolation had sustained AF due to PV ectopy with provocative maneuvers. In the other 5 patients, AVNRT may have been the primary trigger for the arrhythmia. Slow-pathway modification alone may have made a clinical impact in some of these patients if AVNRT were critical to the maintenance of AF, as suggested by other investigators. More investigation is required to elucidate the exact relationship between AVNRT and the triggering mechanism for AF.

Outcomes Related to Ablation of AVNRT and AF

The success rate for cure of AF with slow-pathway modification and targeted ablation of AF triggers was superior to targeted ablation in the rest of the population without inducible AVNRT. There are 2 possible explanations for this outcome. First, the patients with concomitant AVNRT were younger and had less structural heart disease than patients without AVNRT. This likely selects a cohort with a good outcome after ablative therapy for AF. However, after multivariable adjustment that included covariates for age and duration of AF symptoms, identification of AVNRT remained associated with improved outcomes. Second, this may reflect the high success rate of slow-pathway modification for cure of AVNRT compared with the high incidence of recurrent PV conduction after PV isolation.14

In the small subgroup of patients with AVNRT identified as the only trigger for AF, a high rate of freedom from AF was observed with slow-pathway modification alone. For these individuals, the risks associated with transseptal punctures, left atrial ablation, and anticoagulation can be avoided.

Conclusions

AVNRT is an important finding that is identified in a small subgroup of patients undergoing AF ablation. An initial EPS before AF ablation may reveal unsuspected AVNRT that triggers AF, especially in younger patients. A higher rate of success and a lower complication rate are observed when AVNRT is ablated after it has been identified, compared with a strategy that includes targeting the PVs. In addition, AVNRT ablation may be the only procedure that is necessary for cure of AF in select patients.

Disclosures

None.

References


Clinical Perspective

Catheter ablation strategies for treatment of atrial fibrillation (AF) have focused on the role of the left atrium and pulmonary veins (PVs) in initiating and maintaining the arrhythmia. Although the most common sites of atrial ectopy that trigger atrial fibrillation (AF) are in or around the PVs, other supraventricular tachycardias can also coexist with AF. Atrioventricular nodal reentrant tachycardia (AVNRT) is a common supraventricular tachycardia in adults, but the importance of this arrhythmia as a potential cause of AF has not been well studied. In the present study, we identified 27 patients (4.3%) with AVNRT among a cohort of 629 consecutive patients referred for ablation of AF. Of these patients, 13 underwent AVNRT ablation (slow-pathway modification) without PV isolation. Compared with the rest of the cohort, patients with AVNRT and AF were younger at the time of symptom onset. AVNRT was also associated with a better outcome, fewer procedural complications, and a greater proportion of patients free from AF without antiarrhythmic medication after a single procedure. These findings show that AVNRT is an uncommon AF trigger that is seen more frequently in relatively younger patients. It is an important consideration because ablation of AVNRT prevents recurrent AF in some patients. Screening for AVNRT is an important consideration, particularly in young patients without other risk factors for AF, before the patient is subjected to the risks associated with extensive left atrial ablation.

Disclosures

None.
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