Clinical Investigation

Role of Isoproterenol in Predicting the Success of Catheter Ablation

in Patients with Reproducibly Inducible Atrioventricular Nodal Reentrant Tachycardia

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Noninducibility of the arrhythmia is the widely accepted endpoint of successful ablation of atrioventricular nodal reentrant tachycardia (AVNRT). However, to rely upon that as the only endpoint, the arrhythmia must also be inducible before ablation. Despite the fact that AVNRT is not reproducibly inducible in a significant number of cases, the role of reproducible arrhythmia induction and its relationship with the infusion of isoproterenol after successful ablation of AVNRT has not been well defined.

We studied 175 consecutive patients who all underwent successful radiofrequency ablation after showing that they had reproducibly inducible AVNRT without use of isoproterenol. In Group 1 (n=90), isoproterenol was used for arrhythmia reinduction after ablation, whereas in Group 2 (n=85) it was not. The procedural and follow-up data of both groups were recorded, and the results of appropriate statistical tests were analyzed.

During a mean follow-up time of 18.7 ± 4.5 months, 4 patients in Group 1 and 3 patients in Group 2 experienced recurrences. Regardless of elimination or modification of slow-pathway conduction, no significant difference was seen in the recurrence rates of AVNRT between the 2 groups (P=0.72).

We conclude that, when the original arrhythmia in patients with AVNRT is reproducibly inducible in the basal state, the use of isoproterenol after ablation in order to confirm the noninducibility of AVNRT does not appear to alter the recurrence rates and can be omitted.
(Tex Heart Inst J 2014;41(3):280-5)

Key words: *Arrhythmia induction; arrhythmias, cardiac/prevention & control; catheter ablation; isoproterenol; recurrence; retrospective studies; tachycardia, atrioventricular nodal reentry; treatment outcome*

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Catheter ablation of the slow pathway has been accepted as a highly effective treatment, with low recurrence rates, for patients with atrioventricular nodal reentrant tachycardia (AVNRT).[1] Complete elimination of the slow pathway is not necessary for long-term symptomatic relief of the arrhythmia; the most widely accepted endpoint for acute success of the procedure is noninducibility of the arrhythmia.[1,2]

Routinely after slow-pathway ablation, attempts at reinduction of arrhythmia are performed, with or without isoproterenol or other provocative medications. Some physicians use isoproterenol after ablation regardless of its use before ablation (strategy 1),[3-7] whereas others use isoproterenol after ablation of AVNRT only when it had been necessary for arrhythmia induction before ablation (strategy 2).[8-12] However, the published data available for the comparison of these 2 strategies are inadequate.[13,14] In any event, the effectiveness of arrhythmia reproducibility in evaluating the success of AVNRT ablation has not been well defined: AVNRT inducibility is not reproducible in more than one third of cases,[15] and that failure of inducibility after ablation might sometimes indicate the absence of reproducibility, rather than the success of ablation.

To avoid the confounding effect of nonreproducibility, we designed this study to investigate whether the post-ablation administration of isoproterenol for reinduction of AVNRT (in patients in whom the original arrhythmia had been reproducibly inducible without the use of any provocative agent) has any effect on the long-term recurrence rate of the arrhythmia. In both strategies stated above, isoproterenol—when necessary for arrhythmia induction before ablation—is also used to evaluate the inducibility of AVNRT after ablation. Therefore, we did not include in our study any patients who needed isoproterenol for initial induction of AVNRT.

Patients and Methods

In this retrospective study, we enrolled 175 consecutive patients whose AVNRT had been diagnosed during electrophysiologic (EP) study, who had reproducibly inducible arrhythmia without the use of provocative agents, and who had undergone successful radiofrequency (RF) catheter ablation of the slow pathway from October 2007 through October 2009. Before October 2008, we had routinely used isoproterenol after the ablation of AVNRT, regardless of its use before ablation (strategy 1). To the best of our knowledge, there was no strong evidence to advocate this strategy, and the study by Weismuller and colleagues[13] was in favor of the alternative strategy. Subsequently, we decided, as a protocol, to abandon the use of isoproterenol after ablation when the original arrhythmia was reproducibly inducible without the use of isoproterenol (strategy 2).

Consequently, we placed all 175 patients (mean age, 50.6 ± 14.7 yr; 127 women [72.6%]) into 2 groups, with no crossover cases. Patients of Group 1 (n=90) had, before ablation, reproducibly inducible AVNRT without the application of any provocative agent. The inducibility of the arrhythmia after successful ablation was checked both in the basal state and during isoproterenol infusion. Isoproterenol was administered at a dose of 0.5 to 4 μg/min to increase the rate of sinus rhythm 25% over the rate before administration.[16] Patients in Group 2 (n=85), also had, before ablation, reproducibly inducible AVNRT without the use of any provocative agent; however, after successful ablation, their arrhythmia inducibility was checked only in the basal state. (In the current study, the "basal state" is the state without the application of any provocative agent.) Reproducibly inducible arrhythmia is defined as 3 or more episodes of electrically induced, sustained AVNRT with the same stimulation protocol.[15]

Electrophysiologic Study

Each patient gave written informed consent before the procedure. All antiarrhythmic drugs were withdrawn at least 5 half-lives before the procedure. While the patients were under local anesthesia, in the fasting nonsedated state, 3 quadripolar electrode catheters were inserted through the femoral vein and placed in 1) the high right atrium, 2) the right ventricular apex, and 3) the bundle of His. We performed a basic EP study, which included atrial and ventricular incremental pacing until the loss of one-to-one conduction through the AV node (Wenckebach point), together with programmed stimulation with a single extrastimulus. We measured and recorded the EP values. If the arrhythmia could not be induced during the basic study, arrhythmia induction was achieved by using atrial programmed stimulation with 2 or more drive trains (cycle lengths of 600 or 500 ms, and 400 ms) with up to 3 extrastimuli—or atrial burst pacing with cycle lengths decreasing until AV nodal refractoriness was achieved.

Radiofrequency Ablation

After confirming the diagnosis of AVNRT with use of the established criteria,[17] we advanced a 4-mm, 7F, solid-tip, Stinger™ Ablation Catheter (C.R. Bard, Inc., Electrophysiology Division; Lowell, Mass) into the right atrium through the femoral vein to localize the slow-pathway potential in the septal side of the tricuspid annulus, anterior to the coronary sinus ostium. There we found low-amplitude, fractionated, slow-pathway potentials that had an AV electrogram ratio of 0.1 to 0.5.[18] Radiofrequency energy was delivered during sinus rhythm by means of an IBI-1500T11 Cardiac Ablation Generator (St. Jude Medical, Inc.; St. Paul, Minn) in a temperature-controlled mode limited to 60 °C and with power titrated from 30 to 50 W. In every RF application, if no junctional rhythm appeared for 20 s, we moved the catheter to a new spot and repeated ablation. If junctional rhythm was observed, we continued RF application for 60 s unless the catheter was displaced or any AV or ventriculoatrial (VA) block occurred.

In the event of repetitive junctional beats or AV/VA block during RF application, inducibility of the arrhythmia was evaluated. The reinduction protocol consisted of programmed stimulation with 2 or more drive trains and 1 to 3 extrastimuli, atrial and ventricular incremental pacing up to the Wenckebach cycle length, a single ventricular extrastimulus, and atrial burst pacing. If electrical stimulation failed to reinduce the arrhythmia, we concluded that the AVNRT was no longer inducible. It should be noted that we did not wait longer than was necessary to perform the reinduction protocol. As mentioned earlier, in Group 1 patients, the endpoint of the procedure was noninducibility of AVNRT, both in the basal state and after pharmacologic provocation with isoproterenol. In Group 2 patients, however, the endpoint was defined as noninducibility of AVNRT only in the basal state. In both groups, we considered an acceptable endpoint to be the persistence after ablation of either single atrial echo beats or a 50-ms or more increment of the atrium-to-His (A–H) interval in response to a 10-ms decrement of the coupling interval of an atrial extrastimulus (A–H jump); this we defined as the slow-pathway modification. Otherwise, we considered the slow pathway to have been eliminated.

All intracardiac electrograms and surface electrocardiograms (ECGs) were displayed on a screen and simultaneously recorded on a digital computer-based system with hard-drive storage (LabSystem™ PRO EP Recording System, Bard Electrophysiology), then were periodically backed up on external disks. Intracardiac electrograms were filtered at frequencies of 30 to 500 Hz and measured with computer-assisted tools at a sweep speed of 100 or 200 mm/s. Pacing was performed

by means of an EPS320 Cardiac Stimulator (Micropace EP Inc.; Santa Ana, Calif), with stimuli of 2-ms duration at twice the diastolic threshold.

Follow-Up Protocol

All studied patients underwent 12-lead electrocardiography (ECG) on the day after the procedure, before their discharge from the hospital. All patients were discharged without antiarrhythmic drugs and underwent follow-up 12-lead ECG in the outpatient clinic, at 1 and 6 postprocedural months. In the event of symptoms that suggested short-duration supraventricular tachycardia (SVT), patients who could not undergo standard 12-lead ECG during their symptomatic periods underwent 24- to 48-hr Holter monitoring. Late follow-up (from 12–28 mo) for the recurrence of tachycardia symptoms was done by telephone. If the patient developed symptoms identical to those before ablation, or if there arose any documented evidence of tachycardia recurrence, such as an ECG or a Holter recording showing an SVT, we repeated EP studies and, if necessary, repeated catheter ablation.

Statistical Analysis

Continuous variables are presented as mean ± SD. Categorical variables are expressed as absolute frequencies and percentages. For continuous variables, comparisons between the groups were performed by means of the independent Student *t* test or the Mann-Whitney U test, wherever appropriate. Nominal variables were compared by means of the χ^2 test. All statistical tests were 2-tailed, and *P* values <0.05 were considered to be statistically significant. Kaplan-Meyer analysis and log-rank testing were used for event analysis. Hazard ratio (HR) was measured by means of Cox regression analysis. The Statistical Package for the Social Sciences 16.0 (IBM Corporation; Armonk, NY) was used for statistical analysis.

Results

Table I shows the patients' procedures and follow-up data. In 172 of the 175 patients (98.3%), a junctional rhythm was observed during RF application. At the end of the procedure, successful ablation of AVNRT was achieved in 86 patients (49.1%) with slow-pathway modification, whereas it was achieved in 89 patients (50.9%) with slow-pathway elimination. There were no significant differences between the 2 groups in terms of whether the slow pathway was modified or eliminated. The mean procedural time, measured as the time from insertion to removal of venous sheaths, was longer in Group 1 patients than in Group 2 patients (80.5 ± 26.3 vs 71.2 ± 25.1 min; *P*=0.005), and the mean number of applied RF lesions was higher (6.6 ± 4 vs 5.9 ± 4.9 lesions, *P*=0.037). In Group 1, 5 patients were noninducible at baseline post ablation but were inducible during isoproterenol infusion, which resulted in further ablation.

Follow-Up Periods

The mean follow-up time in the group with isoproterenol (Group 1) was 19.8 ± 4.9 months, and in the group without isoproterenol (Group 2) it was 17.5 ± 3.7 months. Because this difference in follow-up time was significant (*P*=0.001), we monitored the patients in Group 2 for an additional 2 months as well. No recurrence was detected during this additional follow-up period (Fig. 1).

In total, 4 patients in Group 1 and 3 patients in Group 2 experienced recurrence of AVNRT (HR=0.781; 95% confidence interval [CI], 0.175–3.488; *P*=0.746).

TABLE I. Patients' Baseline and Follow-Up Characteristics

Variable	Group 1 With Isoproterenol (n=90)	Group 2 Without Isoproterenol (n=85)	*P* Value
Female sex (n)	64 (71.1)	63 (74.1)	0.656
Age (yr)	48.8 ± 14.1	52.4 ± 15.3	0.105
Procedural time (min)	80.5 ± 26.3	71.2 ± 25.1	0.005
Fluoroscopy time (min)	13.1 ± 6.4	12.9 ± 8	0.547
Radiofrequency applications	6.6 ± 4	5.9 ± 4.9	0.037
Junctional beats	89 (98.9)	83 (97.6)	0.527
Slow-pathway elimination	48 (53.3)	41 (48.2)	0.5
Follow-up time (mo)	19.8 ± 4.9	17.5 ± 3.7	0.001*
Recurrence	4 (4.4)	3 (3.4)	0.723

*No recurrences were detected in the additional 2 months' follow-up in Group 2.

Values are stated as mean ± SD or as number and percentage. *P* <0.05 was considered statistically significant.

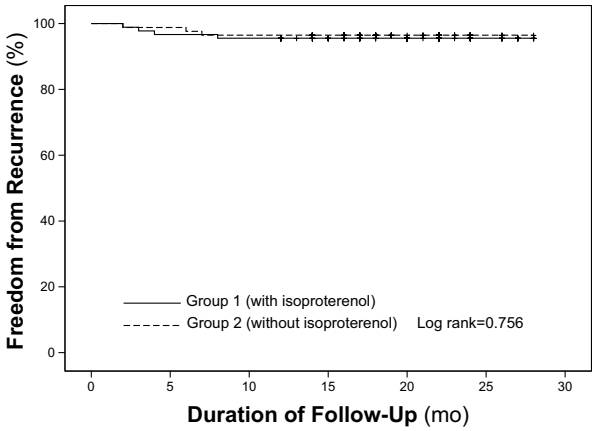

Fig. 1 *Kaplan-Meier curve for recurrence-free survival. Group 1 had isoproterenol after slow-pathway ablation, and Group 2 did not. No significant difference between the groups was found.*

These patients had a mean age of 52.8 ± 13.6 years, and 5 of the 7 were women. All patients with AVNRT recurrence had shown accelerated junctional rhythm during their initial successful ablations.

In both groups, persistence of the slow-pathway conduction at the end of the procedure did not predict recurrence. In Group 1, recurrence was detected in 2 patients whose slow pathways had been eliminated by catheter ablation and in 2 patients whose slow pathways had been modified (HR=1.096; 95% CI, 0.154–7.779; P=0.927). In Group 2, 2 patients with slow-pathway elimination and 1 patient with slow-pathway modification experienced recurrence (HR=0.462; 95% CI, 0.042–5.093; P=0.528). The mean time between ablation and AVNRT recurrence during follow-up was 4.5 months (range, 2–8 mo). In all cases of recurrence, a successful 2nd ablation procedure was performed, with no complication. There were no further recurrences in these patients during a mean follow-up time of 20.1 ± 3.5 months (after the first ablation procedure).

One patient in Group 2 (a 51-year-old man with an eliminated slow pathway) reported paroxysmal palpitation 17 months after his first ablation procedure. Holter ECG displayed nonsustained SVT. Repeated EP study showed neither slow-pathway conduction nor inducible AVNRT; however, focal right atrial tachycardia was induced and successfully ablated.

Discussion

The present investigation shows that routine use of isoproterenol for the evaluation of post-ablation inducibility does not change long-term outcomes in patients who have reproducibly inducible AVNRT in the basal state before ablation. Consequently, the administration of isoproterenol after ablation can be avoided for this group, which constitutes a substantial portion (53%–86%)[12,15,17] of AVNRT patients.

Previous studies have shown that the application of RF current in the slow-pathway area can make AVNRT noninducible, despite persistent slow-pathway conduction.[7,19] The present results are in accordance with those earlier findings, for they show that there is no difference between long-term outcomes of patients with slow-pathway modification and patients with slow-pathway elimination, regardless of how the inducibility of the arrhythmia is checked after ablation.

The proportion of women (72.5%) in the current study was consistent with those of other series (62%–79%),[3-11,13] and there was no statistically significant difference between our 2 study groups in that regard (Table I). Female sex has been associated with higher recurrence rates.[20] However, the recurrence rate of 3.9% in the current study was comparable with those of other reports,[2,4,8,16] and there was no statistically significant difference (P=0.945) in the proportions of women among patients who experienced recurrence (5 women among 7 patients, or 71%) and those who did not (122 women among 168 patients, or 72%).

The Mutual Role of the Reproducibility of Induction and Isoproterenol Infusion in the Ablation of AVNRT

As shown by Hatzinikolaou and colleagues,[21] isoproterenol might or might not facilitate AVNRT induction, depending upon its exact effects on the refractoriness and conduction velocity of AV nodal pathways. Indeed, isoproterenol might actually prevent the induction of AVNRT by decreasing to a high degree the effective anterograde refractory period of the fast AV nodal pathway, thereby abolishing the difference between the anterograde refractory periods of fast and slow pathways.[21]

Accordingly, the use of isoproterenol for reinduction of AVNRT in patients who did not require it for the original induction is a matter of controversy. Some physicians consider the slow pathway to be successfully ablated only if AVNRT is not inducible both in the basal state and during isoproterenol infusion, regardless of how it was induced before ablation (strategy 1).[3-7] Others assume that the infusion of catecholamines is unnecessary after ablation, in cases wherein AVNRT is inducible in the basal state before ablation (strategy 2).[8-12] Neither group, however, has sufficiently examined the reproducibility of the arrhythmia, and both ignore the potential effect of reproducibility on the acute or long-term results of AVNRT ablation.[3-12]

Weismuller and colleagues[13] studied reinduction of AVNRT after ablation in the basal state in comparison with reinduction during the infusion of orciprenaline as a β-adrenergic stimulator. Of 121 patients with AVNRT who underwent successful ablation without sequela, 95 had inducible arrhythmia in the basal state. After ablation, the arrhythmia was not inducible in the basal state in any of these 95 patients, nor did the

addition of catecholamine to the stimulation protocol change that. They concluded that, in patients who have inducible AVNRT in the basal state before ablation, catecholamine infusion is not necessary for reinduction of the arrhythmia after ablation.

The findings of the present study are consistent with the study by Weismuller and colleagues.[13] However, they did not limit their patients to those with reproducibly inducible AVNRT—which might account for the spontaneous recurrence of AVNRT in one of their patients immediately after completion of the procedure. Tachycardia had not been inducible in this patient after ablation, with or without the infusion of orciprenaline.

On the other hand, Stern and associates,[14] in a meta-analysis, showed that the "nonuniform use of isoproterenol" (strategy 2, in our present study) would lead to a higher long-term recurrence rate of AVNRT in cases of slow-pathway modification than in cases of slow-pathway elimination. Conversely, "uniform use of isoproterenol" (strategy 1 in our present study) yielded the same recurrence rates, whether the slow pathway was modified or eliminated. Stern and colleagues concluded that residual slow-pathway conduction after ablation should prompt routine isoproterenol infusion for the evaluation of inducibility, to avoid high recurrence rates.

However, the studies retrieved in Stern's meta-analysis included all cases with inducible AVNRT, and, in common with the study by Weismuller and colleagues,[13] did not delimit them by reproducibility. That might explain the apparent conflict between our study and the meta-analysis of Stern and colleagues. As shown by Stellbrink and co-authors,[15] arrhythmia induction in patients with AVNRT was not reproducible in the basal state in 36% of cases and remained nonreproducible in 7.5% of patients, even with the administration of isoproterenol. Therefore, noninducibility of AVNRT after ablation can sometimes result simply from lack of reproducibility, not from the effect of ablation.

To avoid the confounding effect of nonreproducibility, we included in the current study only patients in whom sustained AVNRT was reproducibly inducible before ablation. Our study findings indicated that, in this group of patients, the use of isoproterenol for checking inducibility after ablation could be overlooked without endangering long-term outcomes.

Study Limitations

This study has a few potential limitations. First, as mentioned in Patients and Methods, our Group 2 patients underwent catheter ablation after our Group 1 patients; the learning-curve effect might for that reason favor the 2nd strategy. We suggest a randomized study to resolve this issue. Second, the arrhythmia recurrence rate was relatively low. Despite our including 175 patients, the study size might still have been too limited to reveal small differences between treatment groups.

Conclusion

The results of the present study signify that withholding isoproterenol from post-ablation evaluation of inducibility of AVNRT might not lead to higher long-term recurrence rates if this approach is confined to properly selected patients, those who have reproducibly inducible AVNRT without the use of provocative agents before ablation. However, the relatively shorter procedure durations and the relatively fewer RF applications observed through this approach might be clinically insignificant.

Acknowledgment

The authors thank Hosein Abrishami, BSc, for his valuable assistance in the data collection.

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