Original Research

Slow-Pathway Ablation for Atrioventricular Nodal Re-Entrant Tachycardia with No Risk of Atrioventricular Block

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Background: Slow-pathway ablation or modification eliminates typical atrioventricular nodal re-entrant tachycardia (AVNRT) but with a 1% risk of AV block. We report our experience from a series of consecutive patients with typical AVNRT who were ablated in our unit.

Methods: Consecutive patients (n=227), aged 22 to 56 years, 172 women, with slow-fast AVNRT underwent slow-pathway ablation. Mapping was restricted to the inferior part of the triangle of Koch, at the right or left septum, below the ostium of the coronary sinus, and was aimed at recording multicomponent, low-amplitude potentials. The endpoints of ablation were induction of a retrogradely conducted junctional rhythm, and non-inducibility of AVNRT on isoprenaline.

Results: All procedures were successful, with no change in the AH interval. Right-sided ablation was successful in 223 (98.2%) of cases. In four patients (1.8%) left-sided ablation was necessary. Procedure and fluoroscopy times were 70.1 ± 21.4 and 11.2 ± 5.8 min, respectively. In total, 4.5 ± 1.2 radiofrequency lesions per patient were given. During a follow-up period of one to three years, three patients (1.3%) had AVNRT recurrence. All of them had residual dual pathway physiology following ablation, while only 19.6% of patients without AVNRT had residual dual AV nodal conduction ($p<0.001$). No patient developed AV conduction disturbances.

Conclusions: Ablation at the inferior part of the triangle of Koch with the protocol described offers a high success rate, with no risk of AV block, in patients with typical AVNRT. Residual dual AV nodal conduction carries an increased risk of AVNRT recurrence.

Slow-pathway ablation for the treatment of typical slow-fast atrioventricular nodal re-entrant tachycardia (AVNRT) is usually implemented by means of a combined anatomical and mapping approach.[1,2] Ablation lesions are delivered in the inferior or mid part of the triangle of Koch,[1-4] and multi-component atrial electrograms or low amplitude potentials, although not specific for the identification of slow pathway conduction,[5] are successfully used to guide ablation in these areas. Although this approach is effective, it is associated with a 1% risk of atrioventricular (AV) block.[6,7] In an effort to minimise this risk, modification, as opposed to complete ablation, of the slow pathway has been proposed. However, the impact of this approach on the efficacy of the procedure is not established.[8-12]

We have analysed a consecutive series of patients with typical slow-fast AVNRT who underwent slow-pathway ablation in our laboratory. Success rates and complications were considered in the context of residual or not dual pathway conduction following ablation.

Methods

Patients

Consecutive patients with typical slow-fast AVNRT who underwent slow-pathway ablation in our laboratory were studied. All ablation procedures were performed by the same team and all patients provided written, informed consent.

Electrophysiology study

Patients were studied in the post-absorptive state, under sedation with diazepam and diamorphine, and after all antiarrhythmic agents had been discontinued for more than 3 days. No patient had received amiodarone for the preceding three months.

Conventional quadripolar or hexapolar electrodes were introduced into the right atrium across the tricuspid valve to record a right-sided His bundle electrogram, the coronary sinus, high right atrium and right ventricle. Bipolar electrograms were filtered at 30-500 Hz, amplified at gains of 20-80 mm/mV, and displayed and acquired on a physiological recorder (Bard LabSystem Duo), together with surface electrocardiograms. Typical slow-fast AVNRT was diagnosed according to standard criteria.[13] AV nodal conduction jumps were diagnosed using the criterion of an increase of at least 50 ms in the AH interval for a 10 ms decrease in the atrial coupling interval. Demonstration of a conduction jump indicated persistent conduction over the slow pathway.

Mapping and ablation

A deflectable catheter with a 4 mm tip and 2.5 mm interelectrode spacing (Cordis-Webster) was used for mapping and ablation purposes. Radiofrequency (RF) current was supplied by a 500 kHz generator (Cordis, Johnson and Johnson) at a preset electrical power of 35 W, and a target temperature of 60°C. The catheter was positioned at the inferior (posterior) part of the tricuspid annulus until an A/V ratio of <1 was recorded, and the atrial electrogram was delayed relatively to the atrial electrogram recorded at the His bundle.[14] Multi-component atrial electrograms and low-amplitude potentials were sought at the area below the coronary sinus ostium, as visualised in the postero-anterior and, particularly, right anterior oblique projections (Figures 1 & 2). Care was taken to keep the ablating catheter below the ostium of the coronary sinus; mapping was not performed at the mid or anterior septum. When multi-component signals (Figure 3) or separate, low-amplitude potentials were obtained, RF current was delivered for up to 10 s until a junctional rhythm with 1:1 retrograde ventriculo-atrial (VA) conduction was elicited. RF current was delivered for up to 10 s at each site until a junctional rhythm with 1:1 VA conduction was elicited. If VA conduction was not seen, RF delivery was immediately stopped. Once junctional rhythm with VA conduction was recorded, energy delivery was continued for 10 s and arrhythmia induction was attempted with the use of isoprenaline. The endpoints for ablation termination were demonstration of ener-

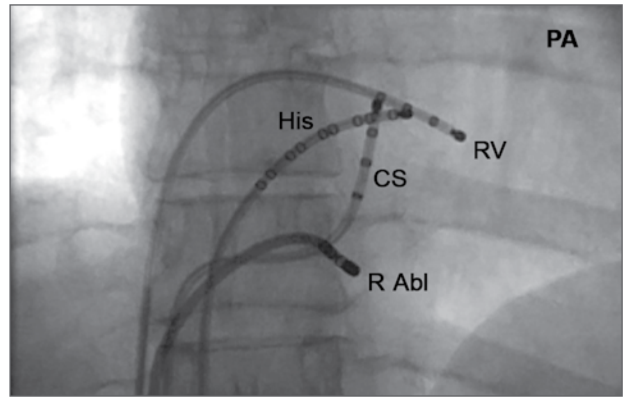

Figure 1. Position of the ablation electrodes in the postero-anterior (PA) projection. Note that the ablating electrode is below the ostium of the coronary sinus. CS – coronary sinus; His – His bundle; Abl – ablation electrode at the right inferior septum.

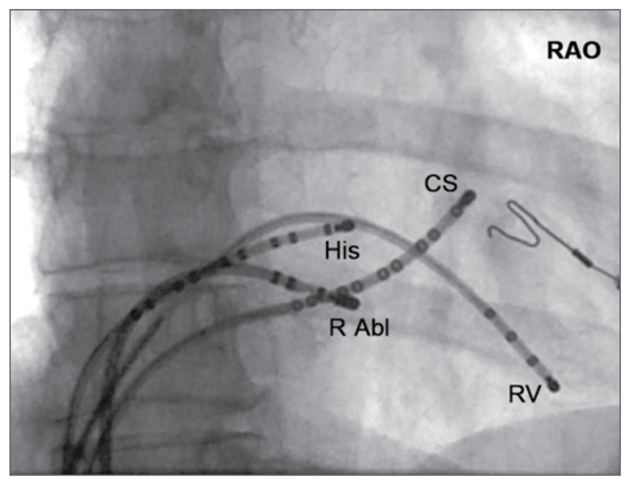

Figure 2. Position of the ablation electrodes in the right anterior oblique (RAO) projection. This is the ideal projection for mapping the inferior part of the triangle of Koch. Abbreviations as in Figure 1.

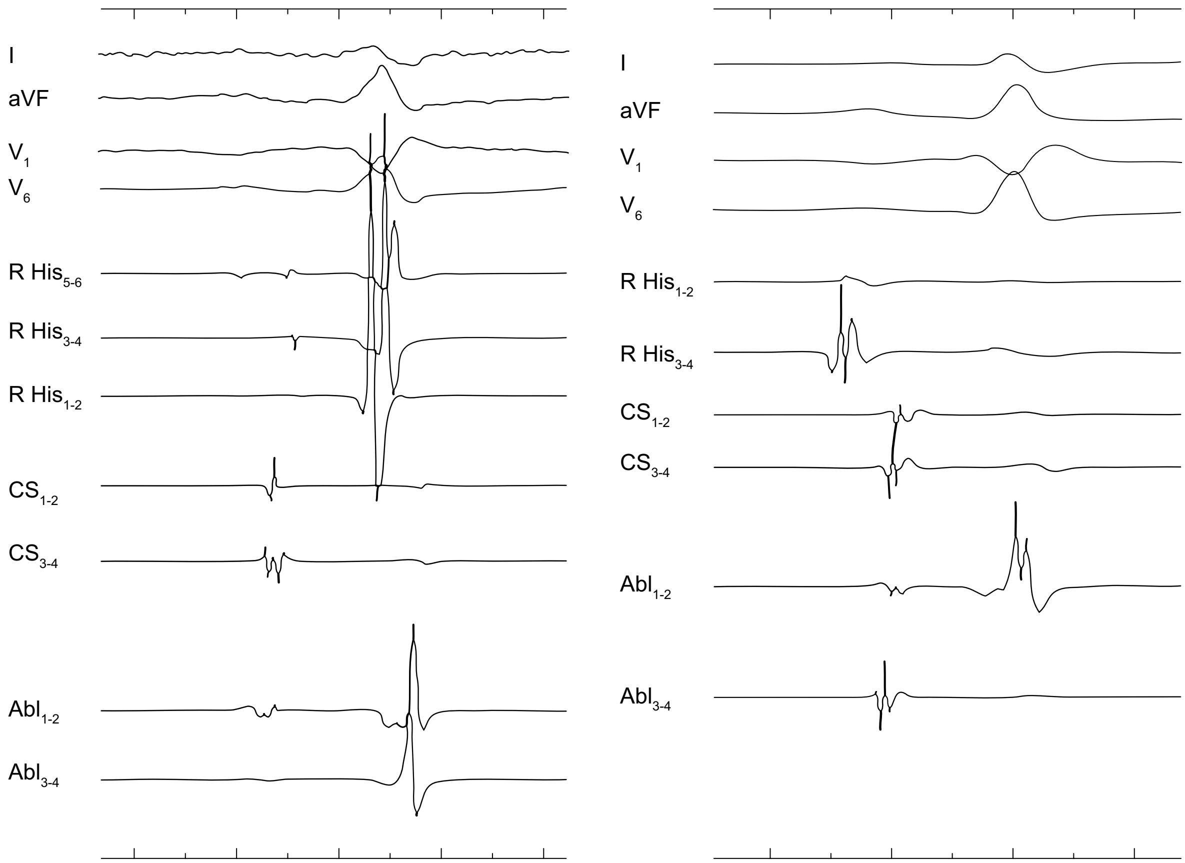

Figure 3. Multi-component atrial electrograms at a successful ablation site during sinus rhythm. I, aVF, V_1, V_6 – standard surface ECG leads; other abbreviations as in Figure 1.

gy-induced junctional rhythm conducted to the atria, and non-inducibility of tachycardia with isoprenaline challenge. No "bonus" pulse was given. If RF induction of junctional rhythm or non-inducibility of tachycardia could not be accomplished from the right side of the septum, mapping at the corresponding part of the left septum was undertaken. A mapping electrode was retrogradely introduced through the non-coronary cusp of the aortic valve to record a left-sided His bundle electrogram, as described elsewhere.[15] Since positioning of the left septal catheter retrogradely through the non-coronary cusp inevitably results in the mapping of the anterior part of the septum, a trans-septal approach, which should allow additional mapping of the left side of the septum, was used for introduction of the left-sided ablation catheter.[16] Mapping of the inferior (posterior) part of the mitral annulus, below the left-sided His was performed with the same principles as in the right septum. Following successful ablation, patients were discharged from hospital within 24 hours on aspirin and no antiarrhythmic drugs.

Statistical analysis

Categorical variables are presented as relative frequencies (%), continuous variables are presented as mean ± standard deviation, while discrete variables (i.e. number of lesions) are presented as median (minimum value, maximum value). Associations between categorical variables (i.e. recurrence of AVNRT and slow pathway residual conduction) were evaluated using the chi-square test or Fisher's exact test, as appropriate. The association between the number of lesions, procedure time, fluoroscopy time and the recurrence of AVNRT was evaluated using the Mann-Whitney test. All reported p-values were based on two-sided tests and were compared to a significance level of 5%. The Statistical Package for Social Sciences (SPSS Inc. version 14.0 for Windows) was used in order to perform all analyses.

Results

Patients

Two hundred and twenty-seven consecutive patients with slow-fast AVNRT (172 women, age 22 to 56 years), were ablated according to the described protocol. All patients had normal left ventricular function, without evidence of underlying structural heart disease.

Slow pathway ablation

All procedures were uneventful, with no change in the AH interval (68 ± 23 ms pre-ablation vs. 59 ± 26 ms post-ablation) and no immediate conduction disturbances. RF-induction of a junctional rhythm with subsequent non-inducibility of AVNRT was accomplished in 223 patients (98.2%) by right-sided ablation. In 4 cases (1.8%) a left-sided approach was necessary. Procedure and fluoroscopy times were 70.1 ± 21.4 and 11.2 ± 5.8 min, respectively. In total, 4.5 ± 1.2 RF lesions per patient were given. Slow pathway ablation with absence of discontinuous curves or conduction jumps was documented in 180 patients (79.9%), whereas modification of the slow pathway was accomplished in the remaining 47 patients, (20.1%) with residual conduction jumps but no arrhythmia inducibility.

Follow up

During a follow-up period of one to three years, three patients (1.3%) had AVNRT recurrence. All three patients had residual conduction jumps following ablation (100%), although no tachycardia was inducible at that time, while only 19.6% of patients without AVNRT had residual conduction jumps ($p<0.001$). No statistically significant difference in left septal ablation rate was detected between patients with recurrence (100%) and those without (98.2%, $p=0.815$). Nor was any significant association detected in the number of lesions, the procedure time, the fluoroscopy time or the recurrence of AVNRT ($p=0.159$, $p=0.146$ and $p=0.100$, respectively). No patient presented with AV block of any degree.

Discussion

Our results indicate that successful ablation of AVNRT can be accomplished without any risk of AV conduction impairment. We believe that the main reason for the avoidance of AV block was the fact that we did not attempt any delivery of lesions to the mid or superior septum, as has been traditionally deemed necessary in difficult cases.[3-5]

Successful slow pathway ablation with no risk of AV block has also been recently described by Steven et al.[17] Modification, as opposed to complete ablation of the slow pathway has been advocated for the elimination of AV block risk in AVNRT ablation procedures. However, even this approach does not avoid a small but considerable AV block risk (0.8%), while it carries a rather high rate of recurrence (6.9%).[12] In addition, in several,[8,9,11] although not all,[10,12] previous studies, maintenance of dual AV conduction physiology has been identified as a predictor of recurrence. Our experience is in keeping with the former: all three cases with AVNRT recurrence had residual slow-pathway conduction after ablation, although tachycardia was not inducible at that time.

RF-induction of a junctional rhythm is considered a sensitive marker of successful ablation in AVNRT,[18,19] although its absence does not necessarily preclude a successful outcome.[17,20] Its origin is not known; there has been some evidence that it might be due to transmission of the effects of RF energy to the atrial exit site of the retrograde fast pathway.[21] In our series, however, no AV conduction impairment was documented, despite induction of a junctional rhythm in all patients. The demonstration of stable VA conduction during junctional rhythm appears to be a reliable marker of safe ablation.

The exact origin of multi-component electrograms and low-amplitude potentials recorded at the triangle of Koch is not known. Mazagalev and Tchou[22] have demonstrated that distinct wave fronts, which are likely to represent the slow component of dual AV nodal electrophysiology, can be recorded at the endocardial surface of the triangle of Koch in animals, and most probably originate in the deeper nodal layers that include the inferior nodal extensions. Specific ablation at the anatomic area of the right or left posterior nodal extensions is feasible and may offer a favourable outcome without the need for mid-septal lesions in difficult cases. There has been considerable experimental and clinical evidence that the inferior nodal extensions, and the atrial inputs they facilitate,[23] may be involved in slow pathway conduction.[24-27] We have previously provided evidence that elimination of AVNRT may be due to partial or complete interruption of the inferior nodal extensions by the ablation procedure.[16] Such extensions should be approachable from the right or left inferior part of the septum. Left-sided ablation of the slow or fast pathway in AVNRT has been described previously in single cases.[28-31] In our series, four cases needed a left-sided approach.

Study limitations

Our method has been put on trial exclusively in patients with slow-fast AVNRT. However, eccentric retrograde atrial activation is not uncommon in fast-slow AVNRT,[13] thus indicating diverse anatomic positions of the slow pathway, including the mid or even anterior septum, at least in patients prone to this form of arrhythmia. The efficacy of our method in this clinical setting, therefore, is not proven. However, according to unpublished observations from our laboratory, this approach is apparently effective in either fast-slow or slow-slow forms of the arrhythmia. Secondly, we do not know whether additional ablation in the presence of residual slow pathway conduction might have averted recurrences without an increased risk of AV block.

In conclusion, restriction of ablation to only the inferior part of the triangle of Koch eliminates slow-fast AVNRT in the majority of patients. If arrhythmia cannot be rendered non-inducible with right-sided ablation, the corresponding part of the left septum should be considered for ablation. This method allows successful elimination of typical slow-fast AVNRT with high success rates and no risk of AV block.

References

1. Jackman WM, Beckman KJ, McClelland JH, et al. Treatment of supraventricular tachycardia due to atrioventricular nodal reentry, by radiofrequency catheter ablation of slow-pathway conduction. N Engl J Med. 1992; 327: 313-318.
2. Haissaguerre M, Gaita F, Fischer B, et al. Elimination of atrioventricular nodal reentrant tachycardia using discrete slow potentials to guide application of radiofrequency energy. Circulation. 1992; 85: 2162-2175.
3. Kalbfleisch SJ, Strickberger SA, Williamson B, et al. Randomized comparison of anatomic and electrogram mapping approaches to ablation of the slow pathway of atrioventricular node reentrant tachycardia. J Am Coll Cardiol. 1994; 23: 716-723.
4. Lee P-C, Chen S-A, Hwang B. Atrioventricular node anatomy and physiology: implications for ablation of atrioventricular nodal reentrant tachycardia. Curr Opin Cardiol. 2009; 24: 105-112.
5. Haïssaguerre M, Shah DC, Jaos P. Slow potentials and catheter ablation for AVNRT. Heart. 1997; 78: 1-2.
6. Spector P, Reynolds MR, Calkins H, et al. Meta-analysis of ablation of atrial flutter and supraventricular tachycardia. Am J Cardiol. 2009; 104: 671-677.
7. Ricard P, Latcu DG, Yaoci K, Zarqane N, Saoudi N. Slow pathway radiofrequency ablation in patients with AVNRT: junctional rhythm is less frequent during magnetic navigation ablation than with the conventional technique. Pacing Clin Electrophysiol. 2010; 33:11-15.
8. Li HG, Klein GJ, Stites HW, et al. Elimination of slow pathway conduction: an accurate indicator of clinical success after radiofrequency atrioventricular node modification. J Am Coll Cardiol. 1993; 22: 1849-1853.
9. Baker JH 2nd, Plumb VJ, Epstein AE, Kay GN. Predictors of recurrent atrioventricular nodal reentry after selective slow pathway ablation. Am J Cardiol. 1994; 73: 765-769.
10. Manolis AS, Wang PJ, Estes NA 3rd. Radiofrequency ablation of slow pathway in patients with atrioventricular nodal reentrant tachycardia. Do arrhythmia recurrences correlate with persistent slow pathway conduction or site of successful ablation? Circulation. 1994; 90: 2815-2819.
11. Tebbenjohanns J, Pfeiffer D, Schumacher B, Manz M, Lüderitz B. Impact of the local atrial electrogram in AV nodal reentrant tachycardia: ablation versus modification of the slow pathway. J Cardiovasc Electrophysiol. 1995; 6: 245-251.
12. Clague JR, Dagres N, Kottkamp H, Breithardt G, Borggrefe M. Targeting the slow pathway for atrioventricular nodal reentrant tachycardia: initial results and long-term follow-up in 379 consecutive patients. Eur Heart J. 2001; 22: 82-88.
13. Katritsis DG, Camm AJ. Atrioventricular nodal reentrant tachycardia. Circulation. 2010; 122: 831-840.
14. Hintringer F, Hartikainen J, Davies DW, et al. Prediction of atrioventricular block during radiofrequency ablation of the slow pathway of the atrioventricular node. Circulation. 1995; 92: 3490-3496.
15. Katritsis DG, Ellenbogen KA, Becker AE, Camm AJ. Retrograde slow pathway conduction in patients with atrioventricular nodal re-entrant tachycardia. Europace. 2007; 9: 458-465.
16. Katritsis DG, Becker AE, Ellenbogen KA, Giazitzoglou E, Korovesis S, Camm AJ. Effect of slow pathway ablation in atrioventricular nodal reentrant tachycardia on the electrophysiologic characteristics of the inferior atrial inputs to the human atrioventricular node. Am J Cardiol. 2006; 97: 860-865.
17. Steven D, Rostock T, Hoffmann BA, et al. Favorable outcome using an abbreviated procedure for catheter ablation of AVNRT: results from a prospective randomized trial. J Cardiovasc Electrophysiol. 2009; 20: 522-525.
18. Thakur RK, Klein GJ, Yee R, Stites HW. Junctional tachycardia: a useful marker during radiofrequency ablation for atrioventricular node reentrant tachycardia. J Am Coll Cardiol. 1993; 22: 1706-1710.
19. Jentzer JH, Goyal R, Williamson BD, et al. Analysis of junctional ectopy during radiofrequency ablation of the slow pathway in patients with atrioventricular nodal reentrant tachycardia. Circulation. 1994; 90: 2820-2826.
20. Hsieh MH, Chen SA, Tai CT, Yu WC, Chen YJ, Chang MS. Absence of junctional rhythm during successful slow-pathway ablation in patients with atrioventricular nodal reentrant tachycardia. Circulation. 1998; 98: 2296-2300.
21. Yu JC, Lauer MR, Young C, Liem LB, Hou C, Sung RJ. Localization of the origin of the atrioventricular junctional rhythm induced during selective ablation of slow-pathway conduction in patients with atrioventricular node reentrant tachycardia. Am Heart J. 1996; 131: 937-946.
22. Mazgalev TN, Tchou PJ. Surface potentials from the region of the atrioventricular node and their relation to dual pathway electrophysiology. Circulation. 2000; 101: 2110-2117.

23. Inoue S, Becker AE. Posterior extensions of the human compact atrioventricular node: a neglected anatomic feature of potential clinical significance. Circulation. 1998; 97: 188-193.
24. Reid MC, Billette J, Khalife K, Tadros R. Role of compact node and posterior extension in direction-dependent changes in atrioventricular nodal function in rabbit. J Cardiovasc Electrophysiol. 2003; 14: 1342-1350.
25. Dobrzynski H, Nikolski VP, Sambelashvili AT, et al. Site of origin and molecular substrate of atrioventricular junctional rhythm in the rabbit heart. Circ Res. 2003; 93: 1102-1110.
26. Katritsis DG, Becker AE, Ellenbogen KA, et al. The right and left inferior extensions of the atrioventricular node may represent the anatomic substrate of the slow pathway in the human. Heart Rhythm 2004; 1: 582-586.
27. Katritsis DG, Becker A. The atrioventricular nodal reentrant tachycardia circuit: a proposal. Heart Rhythm. 2007; 4: 1354-1360.
28. Jaïs P, Haïssaguerre M, Shah DC, et al. Successful radiofrequency ablation of a slow atrioventricular nodal pathway on the left posterior atrial septum. Pacing Clin Electrophysiol. 1999; 22: 525-527.
29. Sorbera C, Cohen M, Woolf P, Kalapatapu SR. Atrioventricular nodal reentry tachycardia: slow pathway ablation using the transseptal approach. Pacing Clin Electrophysiol. 2000; 23: 1343-1349.
30. Khairy P, Seslar SP, Triedman JK, Cecchin F. Ablation of atrioventricular nodal reentrant tachycardia in tricuspid atresia. J Cardiovasc Electrophysiol. 2004; 15: 719-722.
31. Kobza R, Hindricks G, Tanner H, Kottkamp H. Left-septal ablation of the fast pathway in AV nodal reentrant tachycardia refractory to right septal ablation. Europace. 2005; 7: 149-153.