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Case Report

# Implication of the Unipolar Recording for the mapping of Slow Pathway of AV Node in Patient with Atrioventricular Nodal Reentrant Tachycardia and Prolonged PR

**Ardashev Andrey and Zhelyakov Evgeny***

*Moscow State University, Moscow, Russia*

***Corresponding Author**: Zhelyakov Evgeny, Department of Electrophysiology, Moscow State University, Moscow, Russia.

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## Abstract

We described the case of a 71-year-old female with atrioventricular nodal reentrant tachycardia (AVNRT) who had prolonged PR interval during sinus rhythm. Slow pathway ablation was performed, guided by bipolar and unipolar slow pathway potential. The slow pathway radiofrequency (RF)-modification improved conduction of the proximal atrioventricular (AV) nodal structures and resulted in decreasing of the PR and atrio-His intervals. Moreover, the improvement of AV conduction after the slow pathway ablation lasted for at least 18 months.

***Keywords:*** *Atrioventricular Nodal Reentrant Tachycardia; Unipolar Mapping; Slow Pathway; Ablation*

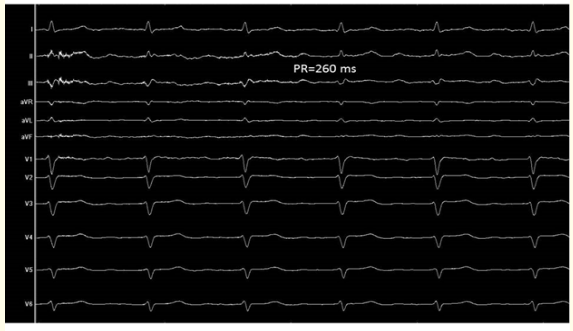
## Introduction

Radiofrequency (RF) catheter ablation of the slow AV nodal pathway has evolved as the therapy of choice for (AVNRT) [1]. Although injury of AV-node conduction was observed in 1.5% of patients who underwent slow pathway ablation [2,3] commonly guided by bipolar (slow and sharp) potentials [4,5], it was reported that it occurred more frequently in patients with prolonged PR interval before intervention [6]. Also it has been reported that slow pathway modification led to improvement of the AV conduction in such patients [7]. Using unipolar recording allows optimizing mapping and ablation of the slow AV nodal pathway [8]. We present the patient with AVNRT and prolonged PR interval during sinus rhythm who underwent successful ablation of AV slow pathway, guided unipolar and recording approach [8] without AV node conduction impairment.

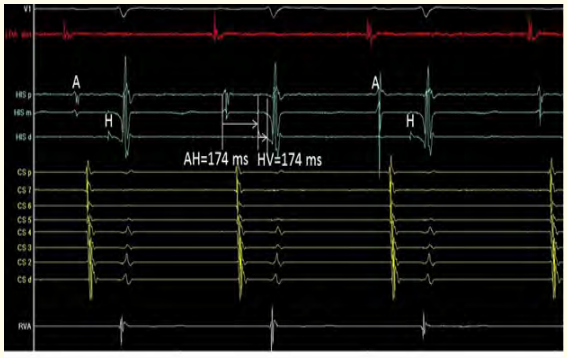
## Case Report

71-year-old female with 6 years history of palpitation was referred for electrophysiological (EP) study and catheter ablation of arrhythmia. The 12-lead ECG in sinus rhythm showed remarkable PR-interval prolongation (260 ms) and no ventricular pre-excitation (Figure 1). Baseline evaluation did not revealed structural heart diseases (ejection fraction was 60%, no cardiomegaly) and severe comorbidities.

After informed consent which was obtained from the patient EP study and catheter ablation were performed. Three 5F quadripolar catheters with 2–5–2-mm interelectrode distances were introduced into the left femoral vein and positioned in the high right atrium, His bundle region, and right ventricular apex for recording and stimulation. A 5F decapolar catheter was introduced and positioned at the coronary sinus vial left subclavian approach. Unipolar (Hz) and bipolar electrograms (EG) were filtered at the 1 - 100 and 30 - 500 Hz, respectively. A programmable UHS-20 stimulator (Biotronik, Germany) with a 2-ms pulse duration at twice the diastolic threshold delivered electrical stimulation. At the beginning of the procedure sinus rhythm cycle length was 980 ms with AH interval of 174 ms and VH interval of 48 ms (Figure 2).




***Panel A***




***Panel B***

***Figure 1:*** *The surface 12 lead ECG (panel A) and intracardiac electrograms (panel B) in sinus rhythm with heart rate of 76 per min before ablation. Top to bottom: ECG leads I–V1, electrogram of high right atrium (HRA), His (His p, His m, His d), coronary sinus (CS p – CS d), right ventricle apex (RVA). A remarkable prolongation of PR interval (260 ms) and AH interval (174 ms) were demonstrated.*

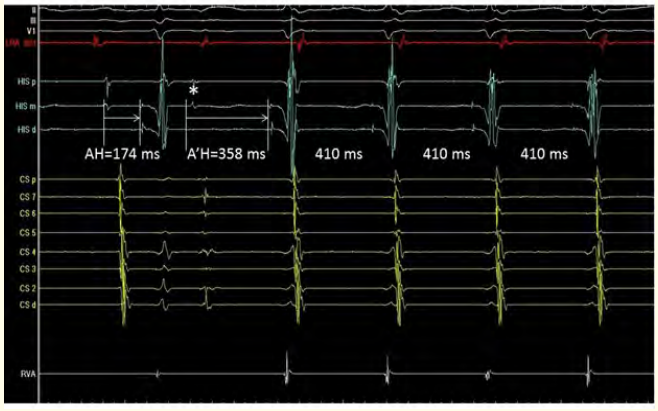



***Figure 2:*** *AV node re-entry tachycardia (slow-fast) induction after premature atrial contraction originating from fast pathway area of AV node (asterisk) and jump AH interval (from 174 ms to 358 ms). Top to bottom: ECG leads I–V1, electrogram of high right atrium (HRA), His (His p, His m, His d), coronary sinus (CS p – CS d), right ventricle apex (RVA). Tachycardia cycle length was 410 ms.*

Right ventricle apex pacing showed an earliest retrograde atrial activation site in the His region.

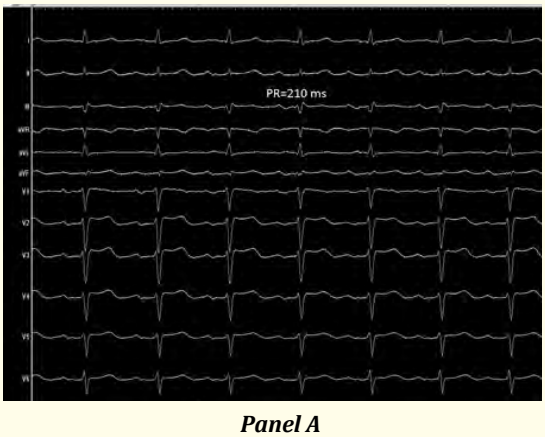
After premature atrial conduction which resulted in the jump AH-interval clinical narrow QRS tachycardia with CL of 410 ms, VA-interval of 90 ms was inducted (Figure 3). Tachycardia was diagnosed as AVNRT by conventional criteria and by excluding intraatrial reentrant tachycardia and atrioventricular reciprocating tachycardia [9].


***Figure 3:*** *Targeting potential of AV slow nodal pathway. Top to bottom: ECG leads I–V1, electrogram of proximal coronary sinus (CS-p), bipolar proximal (Map-p) and distal (Map-d), and unipolar (UnP) mapping channels. During pacing from CS-p the first delta wave-like unipolar component is positive and corresponding to the isoelectric phase preceding qRsr'-like configuration on the bipolar electrogram). The second (rS/RS-like) unipolar component is sharp and biphasic and corresponds to the so-called R wave on the bipolar atrial electrogram. RF application in this site eliminated slow pathway of AV node conduction.*

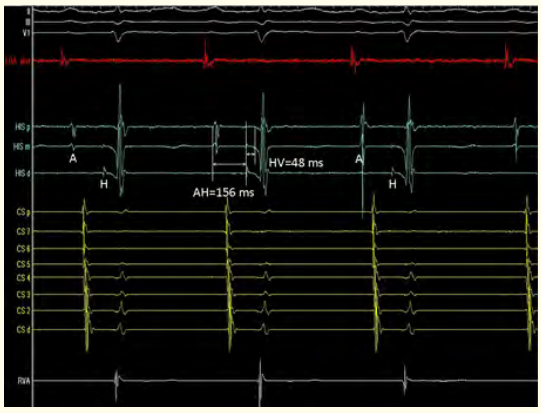
A 4-mm 7F catheter was used for mapping and ablation. The targeting site was determined at the superior edge of the CS ostium guided by bipolar and unipolar slow pathway EG as previously described [8]. Slow pathway potential characterized by atrial component of the bipolar EG had qRs-like morphology, and the AV ratio was 1.0. The unipolar targeting potential showed dual-component atrial electrogram, where the first component was a positive delta-like wave, which corresponded to isoelectric phase preceding qRsr'-like configuration on a bipolar EG. The second rS/RS component of unipolar EG had a sharp and biphasic morphology and corresponded to so-called R wave on a bipolar atrial EG.

RF-application at this site with target temperature of 55 C and power output 45 W resulted in appearance of accelerated junctional rhythm without VA conduction block.

Following the elimination of the slow pathway, the PR and atrio-His intervals became shortened from 260 and 174 to 200 and 100 ms, respectively (Figure 4). Moreover, the improvement of atrioventricular conduction after the slow pathway ablation lasted for at least 18 months.




***Panel A***




***Panel B***

***Figure 4:*** *The surface 12 lead ECG (panel A) and intracardiac electrograms (panel B) in sinus rhythm with heart rate of 86 per min after ablation. Top to bottom: ECG leads I–V1, electrogram of high right atrium (HRA), His (His p, His m, His d), coronary sinus (CS p – CS d), right ventricle apex (RVA). A remarkable prolongation of PR interval (200 ms) and AH interval (120 ms) were demonstrated.*

## Discussion

The possibility of effective ablation of AVNRT in patients with impaired AV conduct previously reported. RF ablation in these patients is associated with increased risk of impairment of AV-conduction [6]. There are few reports of incidental improvement of AV nodal conduction after catheter ablation of slow-pathway region in patients with typical AVNRT [7,8]. S Yamashita., *et al*. demonstrated that RF-modification of the slow pathway can improve AV conduction in patient with prolonged PR prior ablation [8].

Recently, Fukunaga M., *et al*. showed that ablation at the inferior left ganglionic plexi was critical for restoration of normal AV conduction in patient with neurally mediated syncope [9].

The patient under our discussion underwent successful RF ablation of AVNRT guided unipolar and bipolar slow pathway targeting approach [10]. Moreover slow pathway modification accompanied with shortening PR interval.

We can propose that targeting slow pathway potential which was verified at the superior edge of the CS ostium using by bipolar and unipolar recordings may reflect activity of structure autonomic nervous system. It may have important clinical implication in such patients.

## Conclusion

Main clinical implication of unipolar and bipolar recording for the AV slow pathway mapping and ablation in patients with prolonged PR concludes that it approach can allow to treat AVNRT avoiding AV conduction impairment.

## Bibliography

1. Katritsis DG and Camm AJ. "Classification and differential diagnosis of atrioventricular nodal re-entrant tachycardia". *Europace* 8.1 (2006): 29-36.

2. Reithmann C., *et al*. "Ablation for atrioventricular nodal reentrant tachycardia with a prolonged PR interval during sinus rhythm: the risk of delayed higher-degree atrioventricular block". *Journal of Cardiovascular Electrophysiology* 17.9 (2006): 973-979.

3. Martino Crosato., *et al*. "Catheter ablation of atrioventricular nodal reentrant tachycardia in patients with a prolonged PR interval at sinus rhythm". *Journal of Cardiovascular Medicine* 13.5 (2012): 325-329.

4. Haissaguerre M., *et al*. "Elimination of atrioventricular nodal reentrant tachycardia using discrete slow potentials to guide application of radiofrequency energy". *Circulation* 85.6 (1992): 2162-2175.

5. Jackman WM., *et al*. "Treatment of supraventricular reentry by radiofrequency catheter ablation of slow-pathway conduction". *New England Journal of Medicine* 327.5 (1992): 313-318.

6. Meiltz A and Zimmermann M. "Atrioventricular nodal reentrant tachycardia in the elderly: efficacy and safety of radiofrequency catheter ablation". *Pacing and Clinical Electrophysiology* 30.1 (2007): S103-S107.

7. Klank-Szafra M., *et al*. "Radiofrequency ablation and cardioneuroablation for AVNRT and atrioventricular block". *Kardiologia Polska* 68.6 (2010): 720-724.

8. Yamashita S., *et al*. "Improvement of atrioventricular conduction following catheter ablation of atrioventricular nodal reentry tachycardia in a patient with a prolonged PR interval". *Heart and Vessels* 28.1 (2013): 120-125.

9. Fukunaga M., *et al*. "Differential effect of ganglionic plexi ablation in a patient with neurally mediated syncope and intermittent atrioventricular block". *Europace* 19.1 (2017): 119-126.

10. Ardashev A., *et al*. "Atrioventricular Nodal Reentrant Tachycardia Treatment using Novel Potential". *Asian Cardiovascular and Thoracic Annals* 18.6 (2010): 529-535.

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