ORIGINAL ARTICLE

Atrioventricular Nodal Reentrant Tachycardia Treatment using Novel Potential

Andrey V Ardashev, MD, PhD, Alexandr S Makarenko, MD, PhD, Eugeny G Zhelyakov, MD, PhD, Andrey A Shavarov, MD, PhD

83 Clinical Hospital of Federal Biomedical Agency of Russia, Moscow, Russia

ABSTRACT

Radiofrequency ablation of atrioventricular nodal reentrant tachycardia is commonly guided by slow and sharp bipolar potentials of the atrioventricular slow nodal pathway. We optimized the morphology of the guiding potential by unipolar mapping of the slow nodal pathway. We identified a novel unipolar dual-component atrial electrogram at the anterior limb of the coronary sinus ostium. The first component was a positive delta-wave type that corresponded to the isoelectric phase on a bipolar electrogram. The second component had fast biphasic morphology and corresponded to the R wave on a bipolar atrial electrogram. Of 104 consecutive patients with typical atrioventricular nodal reentrant tachycardia, 51 were treated with ablation guided by the novel potential, and 53 underwent ablation using the conventional technique. There was no recurrence of tachycardia in any of these patients. In those treated by the novel potential, there was significantly less radiofrequency power applied and a shorter duration of application than in patients treated by the traditional approach. The novel approach to mapping and ablation of the slow nodal pathway in atrioventricular nodal reentrant tachycardia guided by unipolar recording was safe and effective, and comparable to the traditional technique.

(Asian Cardiovasc Thorac Ann 2010;18:529–35)

KEYWORDS: Atrioventricular Node, Bundle of His, Catheter Ablation, Electrocardiography, Tachycardia, Atrioventricular Nodal Reentry

INTRODUCTION

Radiofrequency (RF) ablation of the slow atrioventricular (AV) nodal pathway has evolved as the therapy of choice for AV nodal reentrant tachycardia (AVNRT).[1] Slow-pathway modification by RF is commonly guided by bipolar (slow and sharp) potentials.[2–4] Guiding bipolar potentials from the ablation electrode have a well-known morphology (Figures 1 and 2). However, in 10% of cases, the slow-pathway potential cannot be recorded in the area of interest.[5] Experimental morphological and electrophysiological data obtained in the last 2 decades have significantly improved our knowledge of the nature of AVNRT.[6–11] The inferior extension of the AV node has been identified and investigated in detail.[8] Medkour and colleagues[10] used unipolar recording to study electrophysiological features of the inferior extension in rabbits. However, there is no clear understanding of the anatomical and electrophysiological mechanisms of AVNRT in humans, and there are no data regarding the clinical usefulness of endocardial unipolar mapping in the Koch triangle for slow-pathway targeting. We aimed to optimize the conventional approach to AVNRT catheter ablation using unipolar electrogram (EG) recordings. We also used bipolar mapping for comparison. Empirically, we found a novel unipolar potential morphology that corresponds to the sites of effective RF ablation on the anterior edge of the coronary sinus ostium. The purpose of this study was to evaluate the efficiency and safety of this novel unipolar potential approach to AVNRT ablation and compare it with the traditional method.

Andrey V Ardashev, MD, PhD Tel: +7 495 395 0678 Fax: +7 495 395 6323 Email: ardashev@yahoo.com
28 Orekhovy Bulvar, Moscow, 115682, Russian Federation.
doi: 10.1177/0218492310387852

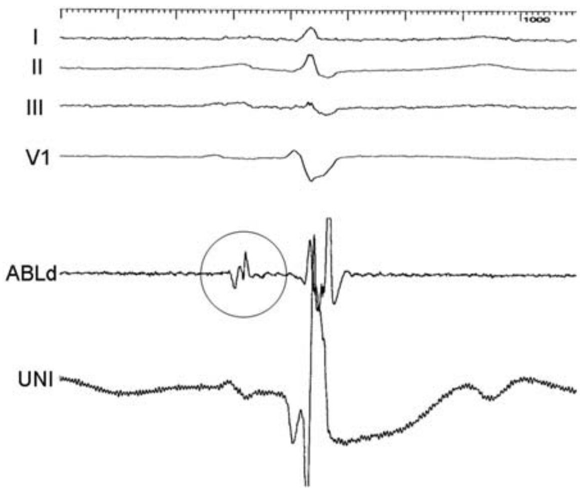

Figure 1. Classic sharp bipolar potential of the slow nodal pathway recorded from the distal pair of bipolar mapping electrodes. The sharp potential is following the 1st component of electrogram reflected atrial activation. Top to bottom: electrocardiogram leads I–V1, bipolar (ABLd), and unipolar (UNI) mapping channels.

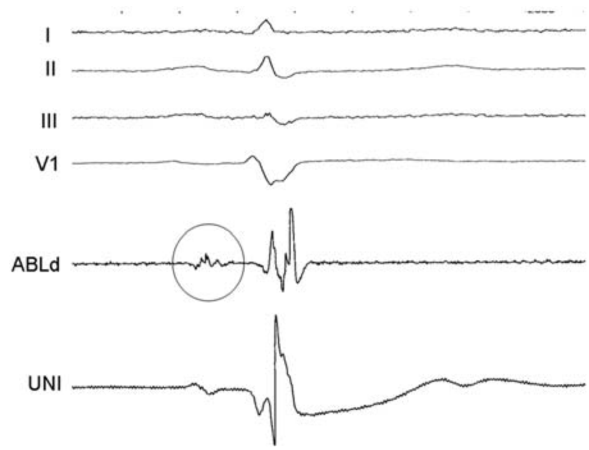

Figure 2. Classic slow bipolar potential of the slow nodal pathway recorded from the distal pair of bipolar mapping electrodes. The atrial component on the bipolar channel represents the slow potential after which the potential of a slow pathway follows. The atrial/ventricular ratio is 1/3.5. Top to bottom: leads I–V1, bipolar (ABLd), and unipolar (UNI) mapping channels.

Patients and Methods

The study was conducted on 104 consecutive patients (44 women; mean age, 44.3 ± 13.6 years) with the typical (slow-fast) form of AVNRT undergoing RF ablation. The study group consisted of 51 patients (21 women; mean age, 40.6 ± 12.3 years) treated with RF ablation guided by the novel approach. The other 53 patients (23 women; mean age, 47.7 ± 13.8 years) underwent AVNRT ablation using the conventional potentials (control group).

Filtered unipolar (1–100 Hz) and bipolar (30–500 Hz) EG recordings were carried out in both groups, using a 7F quadripolar deflectable electrode catheter with a 4-mm distal tip Daig electrode (St. Jude Medical, USA). The unipolar EG was recorded with the tip of the exploring electrode connected to the positive pole. Combined leads located on the inferior edge of the right 6^{th} rib and the left thigh served as indifferent electrodes. A diagnostic catheter with 2–5–2-mm interelectrode distances was positioned in the region of the His bundle. A bipolar EG was recorded from the proximal pair of electrodes of a decapolar catheter positioned in the coronary sinus (CS). The electrophysiological study was performed with both groups in a fasting and mildly sedated state. Antiarrhythmic drugs were discontinued at least one week before the electrophysiological studies. Three 5F–6F quadripolar Cardiorhythm catheters (Medtronic, Minneapolis, MN, USA) with 2–5–2-mm interelectrode distances were introduced into the femoral vein and positioned in the high right atrium, His bundle region, and right ventricular apex for recording and stimulation. Multiple bipolar EG and surface ECG were recorded with a Sensis system (Siemens-Elema AB, Sweden). Bipolar intracardiac EG were filtered at a bandpass of 30–500 Hz. A programmable UHS-20 stimulator (Biotronik, Germany) with a 2-ms pulse duration at twice the diastolic threshold delivered electrical stimulation. The anterograde effective refractory period of the AV nodal fast pathway, Wenckebach point values, and the presence of dual AV nodal physiology (jump) before and after ablation were evaluated. The jump was determined as a sudden increase in the A2–H2 interval of more than 40 ms in programmed atrial stimulation.[4] Tachycardia was diagnosed as AVNRT by conventional criteria and by excluding intraatrial reentrant tachycardia and atrioventricular reciprocating tachycardia.

RF energy was applied in an identical manner in both groups. The upper limits of RF power and temperature were estimated as 45 W and 50°C, respectively. The duration of each RF application was 60 sec in both groups. RF application was delivered between the distal large-tip electrode and an electrosurgical adhesive plate on the patient's back, using an Atakr II RF pulse generator (Medtronic, Minneapolis, MN, USA). The mean RF power and temperature parameters, duration of RF delivery, and number of RF pulses were recorded in both groups. Accelerated AV junctional rhythm or appearance of low atrial rhythm was evaluated during RF pulses. If AVNRT was induced after RF application, we optimized catheter positioning for effective RF ablation, guided by the novel criteria. RF ablation was interrupted if dislocation of an electrode was observed, or if impedance arose. Tachycardia-induction pacing was performed after every RF pulse. The non-inducibility of

AVNRT served as the ablation endpoint. The acute control protocol consisted of intravenous atropine $0.02\,mg{\cdot}kg^{-1}$ and repeated programmed electrical stimulation in attempts to re-induce AVNRT within 45 min after the final RF application. At the anterior limb of the CS ostium, we identified and tested a novel unipolar dual-component potential. The first component of the potential was a positive delta-like wave, and the second (rS/RS-like) component was fast and biphasic during sinus rhythm (Figure 3). Using the traditional approach, we recorded bipolar and unipolar EG with multi-component morphology corresponding to the slow and sharp potentials. Slow potentials were defined as low-amplitude activity with a slow rate of rise. Sharp potentials were the latest atrial EG following a low-amplitude atrial EG during sinus rhythm (Figures 1 and 2). All patients were free of antiarrhythmic drugs during the follow-up period. Late follow-up consisted of hospital visits at 2, 6, and 12 months after RF ablation.

All data are expressed as mean ± standard deviation. Comparison of the 2 groups was performed with a paired t test. A probability value <0.05 was considered significant.

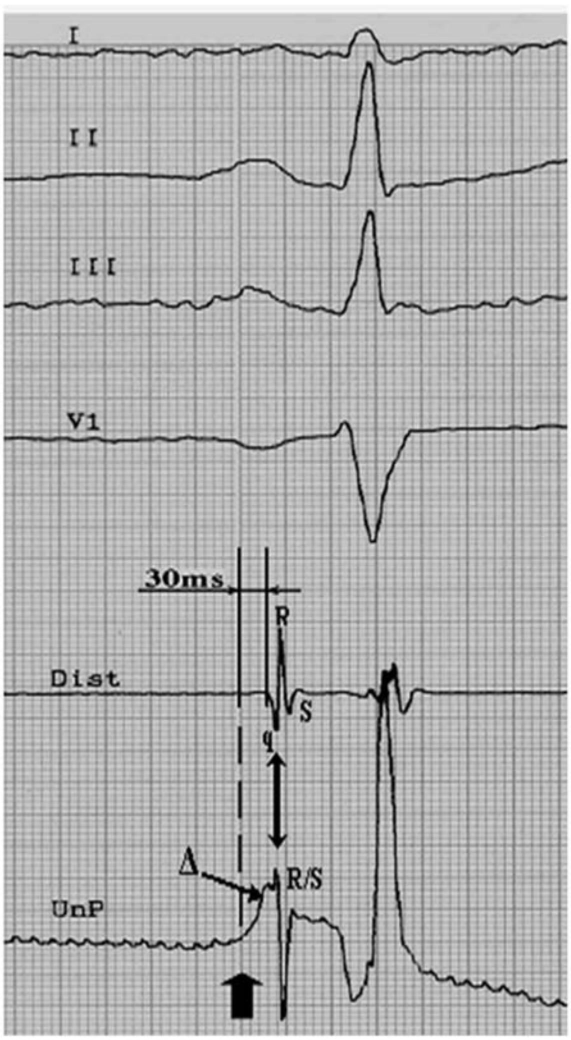

Figure 3. Novel potential of atrioventricular slow nodal pathway mapping. Top to bottom: leads I–V1, distal bipolar (Dist), and unipolar (UnP) mapping channels. The first delta wave-like unipolar component is positive and 30 ms long (corresponding to the isoelectric phase preceding qRsr'-like configuration 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.

Results

The targeting potential was characterized by a dual-component atrial unipolar electrogram. The first component was a positive delta-like wave of 30.0 ± 2.7 ms in length, which corresponded to isoelectric phase preceding qR/qRs/qRsr'-like configuration on a bipolar EG. The second rS/RS component had a sharp and biphasic morphology and corresponded to so-called R wave on a bipolar atrial EG. The atrial component of the bipolar phenomenon of the potential of interest had qR/qRs/qRsr'-like morphology, and the AV ratio was variable, ranging from 0.75 to 1.6. Unipolar mapping of the Koch triangle showed that the earliest atrial activation during sinus rhythm was recorded in the fast nodal pathway area and the novel potential region. After that atrial activation was verified in the sharp potential area, and the latest atrial activation was recorded at the slow potential region (Figure 4). The novel potential area was generally far from the His bundle area (Figure 5). Typical AVNRT was inducted by programmed atrial stimulation in 82 (79%) patients and during incremental atrial pacing in 22 (21%) in both groups during the electrophysiological study. The characteristics both groups are summarized in Tables 1 and 2. Values of the Wenckebach point were not determined before RF ablation in 15 (29%) patients in group 1 and 22 (42%) in group 2, due to AVNRT induction. All patients were successfully treated by RF ablation, and there were no RF-related complications. The jump phenomenon was eliminated in 44 (90%) patients in group 1 and 46 (92%) in group 2. Single atrial echo beats were registered during programmed atrial stimulation in 20 (39%) patients in group 1 and 13 (25%) in group 2 after effective RF ablation; there were no other significant differences in electrophysiological phenomena between the 2 groups after effective AVNRT elimination. There were no early or late complications, and no recurrences of arrhythmia were observed in either group during the follow-up period (32.8 ± 11.0 months).

Discussion

Treatment of AVNRT with RF ablation has become highly effective due to the concept of AV nodal

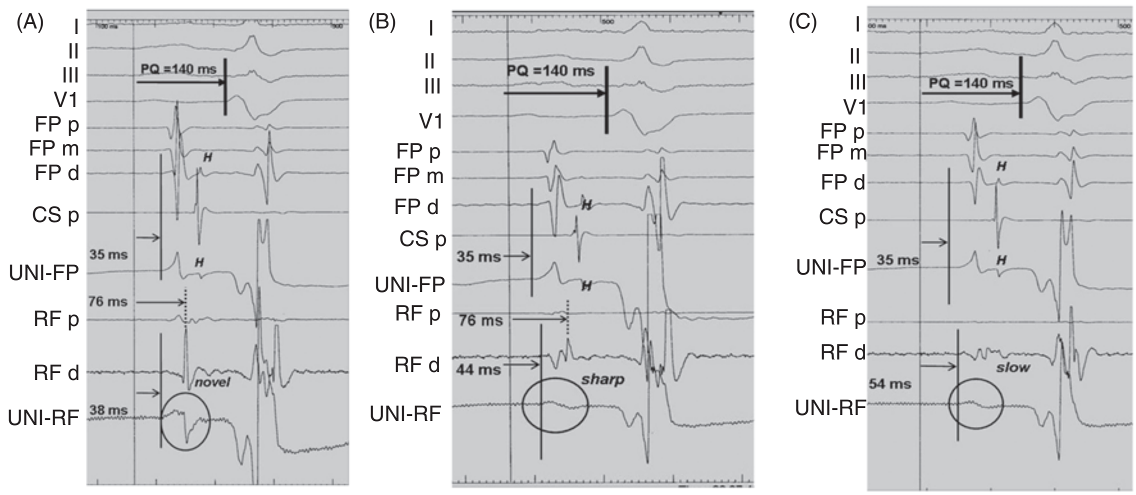

Figure 4. Activation of the Koch triangle during sinus rhythm. Top to bottom: leads I–V1, electrogram fast pathways (FP p, m, d), coronary sinus ostium (CS p), unipolar channel from distal tip of the fast pathway electrode (UNI-FP), proximal and distal bipolar electrogram from an ablation catheter, unipolar electrogram recorded from the distal tip of an ablation catheter (UNI-RF). (**A**) On unipolar recording, the earliest atrial activation was verified in the fast-pathway area (P–A interval, 35 ms) or the novel potential region (P–A interval, 38 ms) during sinus rhythm; the bipolar and unipolar morphological features of the fast pathway (FP and UNI-FP) and the novel potential (RF d and UNI-RF) were different. (**B**) On unipolar registration in the sharp-potential area (UNI-RF), the P–A interval was 44 ms. Note that the interval from P-wave onset to the sharp bipolar potential (RF d) and the interval from P-wave onset to an R wave of the novel bipolar electrogram (RF d, see panel A) were the same (76 ms). (**C**) Unipolar mapping of the Koch triangle, showing the latest activation at the slow-potential region (UNI-RF); the P–A interval was 54 ms.

pathways as a complex of anatomically and functionally related components of the Koch triangle. The electro-anatomical approach to mapping and ablation of slow AV pathways is based on the fact that therapeutic targets are determined by the phenomenological characteristics of conventional bipolar potentials.[2–4] In clinical practice, RF ablation of the slow pathway is based on bipolar recording of so-called slow and sharp potentials. Both potentials are characterized by a multi-component or fractionated nature, mainly due to anisotropic conduction at the site of recording.[1–4] The slow potential is recorded at mid septum, superior and anterior to the CS ostium, but not inferior to it. Its most specific pattern is the progressive response to increasing atrial rates. Its electrophysiological behavior demonstrates a rate-dependent decline in amplitude and slope, a corresponding increase in duration, and separation from preceding atrial potentials until disappearance. The current explanation of the origin and significance of this potential is either activation of the transitional cells approaching the AV node or activation of the rightward inferior extension of the AV node.[10–12]

The sharp potential is recorded at the inferior part of the septum, usually below the CS ostium. It shows multi-component behavior with a prominent sharp phase, which is the latest atrial EG following a low-amplitude atrial EG during sinus rhythm. It is considered to express electrical activation of atrial fibers that connect to transitional cells approaching the inferior AV node extension.[2–4] These 2 potentials permit identification of 2 areas very close to each other, with an overlap zone that belongs to the AVNRT reentrant circuit.[3,4] Autopsic examination as well as experimental work corroborates the hypothesis that the efficiency of RF ablation of AVNRT is associated with RF injury of the AV nodal inferior extension.[13,14] Our data suggest that the morphology of the unipolar signals in the areas where conventional high- and low-frequency bipolar potentials are detected appear to have low diagnostic value and show no pattern geometry during sinus rhythm. However, we have not found any data concerning an endocardial unipolar approach to mapping the slow AV nodal pathway to treat AVNRT.

It is well known that the anterior limb of the CS ostium might be a critical site for AVNRT ablation, even using the traditional approach.[2,3,11,15] We used unipolar recording for slow-pathway mapping, and identified a novel unipolar potential for targeting AVNRT ablation in this area. Unipolar recordings offer some advantages over bipolar recordings. Configurations of the unipolar EG are influenced by electrophysiological and structural characteristics of the underlying myocardial tissue, and indicate the direction of wavefront propagation.[16] When the exploring electrode is located at the site of initial activation, depolarization produces a wavefront that spreads away from the electrode, generating

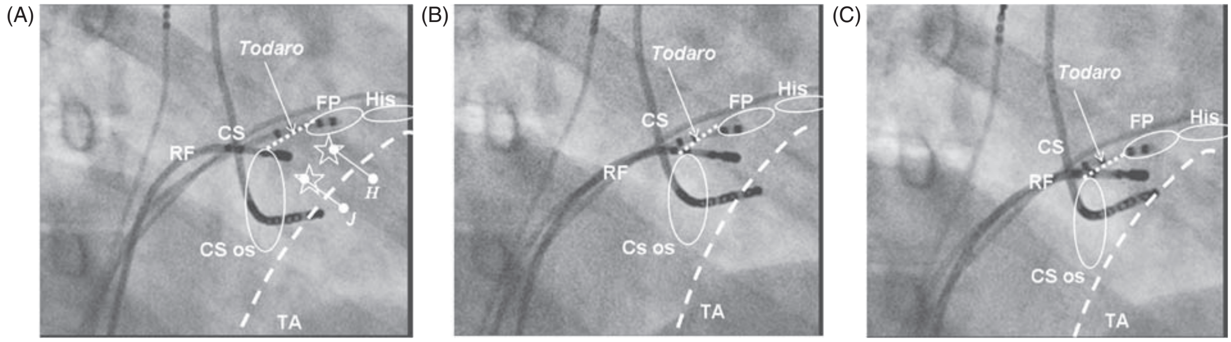

Figure 5. Radiographic images of the positioning of the electrodes (right anterior oblique 40° view) relative to the Koch triangle. (**A**) Mapping of the novel potential area was generally performed far from the His bundle area (stars show mapping areas of conventional slow-pathway potentials (J = Jackman, H = Haissaguerre). Positions of ablation electrodes recording the (**B**) sharp and (**C**) slow potential. CS os = coronary sinus ostium electrode, His = His bundle, FP = fast pathway, RF = mapping electrode, TA = tricuspid annulus, Todaro = ligamentum of Todaro.

Table 1. **Characteristics of study and control groups undergoing radiofrequency ablation**

Variable	Study Group ($n = 51$)	Control Group ($n = 53$)	p Value
Mean age (years)	40.6 ± 12.3	47.7 ± 13.8	NS
Sex (M/F)	30/21	30/23	NS
Electrophysiological data before ablation			
Anterograde ERP of fast pathway (ms)	317 ± 40	326 ± 42	NS
Presence of a jump	49	50	NS
Wenckebach point value (ms)	335 ± 41	350 ± 33	NS
Electrophysiological data after ablation			
Anterograde ERP of fast pathway (ms)	282 ± 14	301 ± 11	NS
Jump preserved	7	7	NS
Single atrial echo beats	20	13	<0.05
Wenckebach point value (ms)	345 ± 37	365 ± 38	NS

ERP = effective refractory period, NS = not significant.

a monophasic QS complex. Recording the dead-end tissue activation produces a monophasic R wave. As the wavefront propagates past the recording electrode, an S wave is inscribed, and thus an RS complex occurs.[16,17] This may provide essential information about the direction of impulse propagation, which may be helpful in determining the precise localization of accessory pathways and the origin of focal arrhythmias. The major disadvantage of unipolar recordings is that they contain a substantial far-field signal generated by depolarization of tissue remote from the recording electrode. Bipolar EG recordings are affected by electrode configuration and the direction of wavefront propagation with respect to electrode positioning.[16,17] Thus unipolar and bipolar EG recordings may provide complementary information.

Based on our experience, the variable bipolar atrial potential differs from the classic morphology of the slow-pathway potential (A/V ratio was 0.75–1.6). The positive delta wave-like atrial unipolar component corresponds to the bipolar isoelectric phase. Hence, its existence may be explained by a far-field atrial musculature depolarization, whereas the higher-frequency component is reflected in local atrial activity. This second unipolar sharp component is biphasic (rS/RS-like) and corresponds to the R wave on a bipolar atrial EG. The absence of fractionation of the atrial component of the bipolar EG suggests insignificant anisotropy at this particular site, which may illustrate the activation of relatively unidirectional and isotropic fibers forming a muscular bundle or fascicle oriented in a circumferential direction all along the anterior/superior limb of the CS orifice, before its connection with the transitional cells approaching the inferior AV nodal extension. This suggestion agrees with the experimental data of McGuire and colleagues[18] who found similar potentials posterior and inferior to the CS orifice and in the proximal CS, using a unipolar technique. Regardless of the similarity between our potential and McGuire's potential, there are some discrepancies. McGuire's unipolar filter settings of 0.01–500 Hz and amplification gain of 500 to 1,000 differed from those used in our study (1–100 Hz). The McGuire potential was found in a

Table 2. **Parameters and electrophysiological phenomena of radiofrequency ablation**

Variable	Study Group ($n = 51$)	Control Group ($n = 53$)	p Value
Parameters of RF ablation			
Duration (sec)	50.9 ± 13.9	89.2 ± 12.7	< 0.05
No. of RF applications	2.1 ± 1.5	3.0 ± 2.9	NS
Power (W)	20.4 ± 4.9	24.5 ± 4.9	< 0.05
Temperature	43.0°C ± 1.9°C	49.3°C ± 7.7°C	NS
Electrophysiological phenomena			
Accelerated AVJR	18	24	NS
Low atrial rhythm	17	9	NS
AVJR + low atrial rhythm	5	7	NS

AVJR = accelerated atrioventricular junctional rhythm, NS = not significant, RF = radiofrequency.

relatively large area from the CS to the tricuspid annulus, posterior and inferior to the CS orifice, and in the proximal CS, in experimental animals. Whereas our potential was mapped in humans in a relatively restricted area, approximately 5 mm in diameter, at the superior edge of the CS ostium, while continuously trying not to slip into the CS by catching the edge of the ostium. It is obvious that our area of interest is different in the proximal CS aspects and timings of channels (CSp, RFd, UNI-RF on Figure 4A). McGuire and colleagues[18] used referential stimulation in 75% of hearts (8 porcine and 4 canine), whereas we mapped during sinus rhythm in all cases.

Our findings show that the unipolar technique might be useful for analyzing an atrial activation hierarchy during sinus rhythm, at least in the triangle of Koch. We suggest that during the sinus rhythm, the initial atrial activation in the triangle of Koch occurs in 2 different areas: the fast-pathway insertion, and the site where the novel potential is recorded (Figure 4). After penetrating the triangle of Koch in the area of the superior limb of the CS ostium, the depolarization front spreads to the area of the sharp potential and subsequently to the zone of the slow pathway suggested by Haissaguerre and colleagues[2] (Figure 4). Interestingly, the timing of the bipolar novel potential corresponds to that of the sharp EG (Figure 4A, 4B). Have we been dealing with the so-called sharp bipolar potential all long? Had we worked with the potential of Jackman and colleagues,[3] it would have been characterized by 2 different unipolar configurations. This means that there are at least 2 different sharp potentials within the Koch triangle. This is either a paradox or confirmation of the novelty of our potential. Bear in mind that this study arose when we found a novel unipolar potential that corresponded to the sites of effective RF ablation at the superior edge of the CS ostium. We supposed that RF ablation guided by our potential eliminated conduction via fascicles that continued from the AV node inferior extension, while conduction via nearby fibers was preserved.[19] This may be clarification of preservation of jump parameters and echo beats in some cases after successful ablation of AVNRT. These phenomena may be explained by a discrepancy in the anatomical substrate for the sustained AVNRT circuit and AV nodal echo beats.[19,20]

We concluded that our novel approach for mapping and ablation of the slow nodal pathway in AVNRT patients, guided by unipolar recording, was safe and effective. We hope that this technique might be of help in RF ablation of typical AVNRT.

References

1. Katritsis DG, Camm AJ. Classification and differential diagnosis of atrioventricular nodal re-entrant tachycardia. Europace 2006;8:29–36.
2. Haissaguerre M, Gaita F, Fischer B, Commenges D, Montserrat P, d'Ivernois C, et al. Elimination of atrioventricular nodal reentrant tachycardia using discrete slow potentials to guide application of radiofrequency energy. Circulation 1992;85:2162–75.
3. Jackman WM, Beckman KJ, McClelland JH, Wang X, Friday KJ, Roman CA, et al. Treatment of supraventricular reentry by radiofrequency catheter ablation of slow-pathway conduction. N Engl J Med 1992;327:313–8.
4. Gaita F. Ablation of typical AVNRT. In: Zipes DP, Haissaguerre M, editors. Catheter ablation of arrhythmias, 2nd edn. Futura Publishing Co, Armonk, NY, 2002:226–75.
5. Nakagawa H, Jackman W. Catheter ablation of paroxysmal supraventricular tachycardia [Review]. Circulation 2007;116:2465–78.
6. McGuire MA, Bourke JP, Robotin MC, Johnson DC, Meldrum-Hanna W, Nunn GR, et al. High resolution mapping of Koch's triangle using sixty electrodes in humans with atrioventricular junctional (AV nodal) reentrant tachycardia. Circulation 1993;88:2315–18.
7. Nikolski V, Efimov I. Fluorescent imaging of a dual-pathway atrioventricular-nodal conduction system. Circ Res 2001;88:E23–30.
8. Nikolski VP, Jones SA, Lancaster MK, Boyett MR, Efimov IR. Cx43 and dual-pathway electrophysiology of the atrioventricular node and atrioventricular nodal reentry. Circ Res 2003;92:469–75.
9. Inoue S, Becker AE. Posterior extension of the human compact atrioventricular node: a neglected anatomic feature of potential clinical significance. Circulation 1998;97:188–93.

10. Medkour D, Becker AE, Khalife K, Billette J. Anatomic and functional characteristics of a slow posterior AV nodal pathway. Role in dual-pathway physiology and reentry. Circulation 1998;98:164–74.

11. Matsuyama T, Ishibashi-Ueda H, Ikeda Y, Yamada Y, Okamura H, Noda T, et al. The positional relationship between the coronary sinus musculature and the atrioventricular septal junction. Europace 2010;12:719–25.

12. Katritsis DG, Becker AE, Ellenbogen KA, Giazitzoglou E, Korovesis S, Camm AJ. 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;1:582–6.

13. Wu D, Yeh SJ, Wang CC, Wen MS, Chang HJ, Lin FC. Nature of dual atrioventricular node pathways and the tachycardia circuit as defined by radiofrequency ablation technique. J Am Coll Cardiol 1992;20:884–95.

14. Inoue S, Becker AE, Riccardi R, Gaita F. Interruption of the inferior extension of the compact atrioventricular node underlies successful radio frequency ablation of atrioventricular nodal reentrant tachycardia. J Intrervent Card Electrophysiol 1999;3:273–7.

15. Waki K, Kim JS, Becker AE. Morphology of the human atrioventricular node is age dependent: a feature of potential clinical significance. J Cardiovasc Electrophysiol 2000;11: 1144–51.

16. Stevenson WG, Soejima K. Recording techniques for clinical electrophysiology [Review]. J Cardiovasc Electrophysiol 2005;16:1017–22.

17. de Bakker JMT, Hauer RNW, Simmers TA. Activation mapping: unipolar versus bipolar recording. In: Zipes DP, Jalife J, editors. Cardiac electrophysiology. From cell to bedside, 3rd edn. Saunders, Philadelphia, 2002:1068–78.

18. McGuire MA, de Bakker JM, Vermeulen JT, Opthof T, Becker AE, Janse MJ. Origin and significance of double potentials near the atrioventricular node. Correlation of extracellular potentials, intracellular potentials, and histology. Circulation 1994;89:2351–60.

19. 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;97:860–5.

20. Loh P, Ho SY, Kawara T, Hauer RN, Janse MJ, Breithardt G, et al. Reentrant circuits in the canine atrioventricular node during atrial and ventricular echoes. Circulation 2003;108: 231–8.